KAVAKA 49: 50- 58(2017)

Fusarium wilt of Melon: Resistance breeding and gene deployment

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(Submitted in August, 2017; Accepted on December 15, 2017)

ABSTRACT

Fusarium wilt incited by *Fusarium oxysporum* f. sp. *melonis* (*Fom*) cause severe losses up to 100% in melon worldwide. *Fusarium* is epidemic due to continuous cultivation of susceptible varieties. Different edaphic factors influence wilt development in melon. Among preventive measure, resistant varieties are the most reliable control measure in hand. Four *Fom* physiological races were identified: 0, 1, 2 and 1, 2. In majority cases, resistance was governed by *Fom*-1 (race 0 and 2) and *Fom*-2 (race 0 and 1). *Fom*-2 encodes a protein which is characterized by the presence of NBS-LRR (Nucleotide Binding Site-Leucine Rich Repeat) domains. *Fom*-3 and *fom*-4 resistant gene have been identified which can built resistance along with *Fom*-1. While most of the present cultivars are susceptible to races 1, 2. Accessions of sub sp. *agrestis* have high level of resistance to races 1, 2 while, certain measurable resistance have also been found within sub sp. *melo viz.*, few Far-East melon. Indian melon germplasm have great potential to combate against existing and newly evolving *Fom* races. Fluorescent study may open new window for identifying resistant cultivars. Biotechnological tools for identifying resistance genes can serve for selection of genotypes. QTL and omic's approach were reported for developing multiple resistant cultivar for different races. An integrated approach may prevent disease until durable and multigenic resistant variety/hybrid will not develop.

Keywords: Melon, *Fusarium oxysporum* f. sp. *melonis*, *Fom*-1, *Fom*-2, Snapmelon

INTRODUCTION

Muskmelon is cultivated on 1.2 million ha area with 29.5 million MT production and 24.9 tonnes ha^{-1} productivity on the globe (Anonymous, 2014). China is the largest producer with 50.04% share followed by Turkey (5.76%), Iran (4.98 %), Egypt (3.54 %) and India (3.49 %). In India, it is cultivated on 47 thousand ha with 878 thousand MT production and 20 tons ha^{-1} productivity (Anonymous, 2016). It is extensively cultivated in hot and dry areas of Uttar Pradesh, Punjab, Rajasthan, Madhya Pradesh, Bihar and Karnataka. Riverbeds of Yamuna, Ganga, Narmada, Kavery, Krishna and Godavari are known for muskmelon cultivation in India.

Breeding for disease resistance has been one of the most important objectives in muskmelon from the last few decades. Melon is susceptible to a number of fungal diseases which affect its yield and quality. Among these, the most devastating disease in melon is fusarium wilt which is instigated by a soil-born pathogen, *Fusarium oxysporum* Schlechtend: Fr. f. sp. *melonis* W C Snyder & H N Hansen (*Fom*). In the infected field, yield losses were reported to tune as high as 100 per cent (Shanthi and Vittal, 2013).

According to Vidhi report in 2017, seed sale of hybrid muskmelon was 5 tons with a value of 30 crores INR while open pollinated varieties seed sale was 400 tons with 20 crores INR. The share of muskmelon seeds was 1.07 % and 5.45% for seed sale and economy generated over all India sales of seeds of major vegetables. Multinational companies are engaged in cultivar development of muskmelon. Cultivars like Bobby (Known-You Seeds Ltd), Farmers' Glory No. 1 (Known-You Seeds Ltd), Golden glory (Green Field Seeds Ltd), Caribbean Gold RZ (Siegers Seeds Ltd), etc had been released for commercial cultivation along with some public sector cultivars and hybrids i.e. Pusa Madhuras, Durgapura Madhu, Pusa Rasraj, Punjab Sunehri, Punjab Anmol, Punjab Hybrid, MH-27, MH-10, MH-51, etc. many of the above mentioned cultivars and hybrids scrummed to Fusarium pathogen. Dhillon *et al.* (2011) documented that all the present day melon cultivars grown by Indian farmers (NS-7455, Punjab Sunehri, Punjab Hybrid) were susceptible to fusarium wilt. This mainly happened due to mono culture of particular variety in a specific geographical area, change in the genome structure of a pathogen i.e. development of new pathotype or mechanical and environmental factor. Recently few cultivars have been released *viz.*, Nirmal 24 muskmelon (Nirmal Seeds Ltd), Trisha 2 (Known-You Seeds Ltd), Patasha (Kalash Seeds Ltd) which are reported to be tolerant against it.

Fusarium oxysporum is the most important pathogen causing wilt in muskmelon and some other important vegetable crops in India (Joshi *et al*., 2013). *Fusarium oxysporum* f. sp. *melonis* was first reported as fundamental agent of muskmelon wilt in Rajasthan (Mathur and Shekhawat, 1992) and later on from other parts of North India (Chattopadhyay and Sen, 1996; Sen *et al.,* 1999). The control of fusarium wilt diseases with fungicides is an ineffective (Mahdikhani, 2016) and has several disadvantages, particularly the cost of fungicides and their residual effects. Therefore, it is imperative to develop cultivars that are resistant to diseases.

India being one of the secondary center of origin (Zeven and Zhukovsky, 1975), sizable melon diversity is present in India (Ganesan, 1991), which can be exploited for genetic improvement in melon. An Indian melon possesses a unique gene sequence which makes them stand discretely against rest of the world germplasm. North Indian wild accessions (34.5%) and landraces from south (24.2%) and eastern part of India (30.4%) have potential gene sources for resistance breeding in melon (Dhillon *et al*., 2009). The purpose of the review is to summarize the knowledge related to *Fom* resistant gene and its current status in the field of research.

PATHOGEN AND ITS BIOLOGY

Fusarium oxysporum Schlechtendahl emend. Synder & Hansen is a cosmopolitan species (Booth, 1971) comprising both pathogenic and non-pathogenic isolates (Gordon and Martyn, 1997). The pathogenic isolates of *F. oxysporum* causes wilt on several horticultural crops and are accordingly subdivided into *Formae speciales* (Baayen, 2000). One of the

most economically important and devastating *Formae speciales* causing melon wilt is *F. oxysporum* f. sp. *melonis* Snyder & Hansen. The first report of melon fusarium wilt in New York was in 1930 (Chupp, 1930), and it later has been found in many melon-growing areas worldwide.

Four physiological races 0, 1, 2 and 1,2 of the pathogen have been reported for its virulence on different melon cultivars (Risser *et al*., 1976). Resistance to race 1 and 2 is conferred by two single dominant genes; *Fom-2* and *Fom-1*, respectively. Both genes also confer resistance to race 0 (Zink and Thomas, 1990). Zink (1991) reported *Fom-3*, a dominant gene, responsible for resistance to race 2. Oumouloud *et al.* (2012) reported a complementary gene *fom-4* with recessive inheritance. However, the race 1,2 is capable of overcoming these resistance genes. Resistance to race 1,2 has been controlled by polygenic recessive genes (Perchepied *et al*., 2005).

Variation in virulence helps in assigning pathotypes to pathogenic races within a formae speciales. Races are defined by their differential interaction with host genotypes (Armstrong and Armstrong, 1978), while in some cases cultivars are known to carry few major resistance genes. Based on pathogenicity on three melon differential cultivars *viz*., Charentais-T, Doublon and CM-17187, Risser *et al.* (1976) classified *Fom* isolates into four common races and designated as 0, 1, 2 and 1, 2.

Fusarium oxysporum f. sp. *melonis* (*Fom*) has no known sexual reproductive stage. Asexual reproductive structures include both micro- and macroconidia. Single celled microconidia are capable of infecting roots but due to their ephemeral nature, they play minor role to cause initial infection under field condition (Egel and Martyn, 2007). Sporodochia are the clumps of macroconidia which are of fusiform structure (spindle-shaped). Macroconidia also infect roots just like microconodia. This fungus produces chlamydospores *i.e.* resting structure under suboptimal conditions. Kannaiyan and Prasad (1975) also stated that *Fom* pathogen survive even under non-host condition under favourable condition which has been discussed under disease development and epidemiology section of this review.

Fusarium oxysporum is an important soil-borne fungus with pathogenic and non-pathogenic strains. *Formae speciales* with concept of pathogenic race are mostly widely used classification in *Fusarium oxysporum* to identify most susceptible crop. All the four physiological races of *Fom* cause disease on susceptible cultivars. Among different *Fom* races, race 1,2 was categorized into two pathotypes: 1,2Y causing yellowing symptoms while 1,2W cause wilting and death of infected plant without yellowing (Chikh-Rouhou *et al*., 2011). Pathotypes were categorized into pathogenic races on the basis of virulence difference. Host pathogen interaction defines races, while handful cultivars were also found to have one or few major resistance gene(s). Four common races were identified on the basis of differential cultivars (**Table 1**). *Formae speciales* were grouped based upon vegetative compatibility which provides a means of characterizing subspecies groups based on the genetics of fungus more willingly than host-pathogen interaction.

Table 1: Modified classification of *Fusarium oxysporum* f. sp. *melonis* race (Risser *et al*., 1976)

Fusarium oxysporum f. sp. *melonis*	Differential cultivars and their gene for resistance					
	Charentais-T	Doublon (*Fom*-1)	CM-17187 (*Fom*-2)	Perlita FR (*Fom*-3)	Tortuga (*fom*-4)	Isabelle
Race 0	S	R	R	R	R	R
Race 1	S	S	R	S	S	R
Race 2	S	R	S	R	R	R
Race 1,2	S	S	S	S	S	IR

For *Fom* detection, different method includes visualizing symptoms for disease identification, spores or mycelia and isolating fungus on selective medium. Since, such medium were not identified for *Fom*, as fungal growth takes time, similarly identification on selective medium also takes time and identification is tentative (Oumouloud *et al*., 2013). DNA based technique with the help of PCR, viz., RT-PCR, offers additional gains over conventional detection for the reason that they are rapid, precise and consistent in nature. Zhao *et al.* (2014) tested pathogenicity using molecular tools on seedlings of cucumber, muskmelon and watermelon with 13 strains of *F. oxysporum* after cutting the root. The results demonstrated that all studied *Fusarium* strains were pathogenic on watermelon seedlings in comparison to other melons.

Vast variations in pathotypes have been observed by number of researchers (Megnegneau and Branchard, 1988; Zuniga *et al.*, 1997; Moretti *et al.*, 2002; Chikh-Rouhou *et al.*, 2010). This includes both pathogenic and non-pathogenic isolates. Among asexually reproduced *Fusarium* species, gene flow is low thus peril is low to breakdown major genes governing resistance. Comparative genomic study also revealed that pathogenicity can be transferred within genus and so cause disease on wide range of host. Different vegetative groups and stains were classified into four races which were identified until now and against these few differentials were reported for its confirmation. There is more probability of having more races from the region of Mediterranean since least attention was given even though much crop failure was observed.

DISEASE DEVELOPMENT AND EPIDEMIOLOGY

The *Fusarium* grows profusely with compatible host crops. An epidemic could be due to continuous cultivation of susceptible variety. Under non host condition, pathogen survives in the form of chlamydospores (resting structure) and these become saprophytic under favourable condition (60% soil moisture, pH 5.0 - 6.0 and temperature 25 - 35° C) (Kannaiyan and Prasad, 1975). Pathogen can get introduced to new area via seed. Seed infection in bottle gourd, muskmelon, cucumber, watermelon and pumpkin have also been reported (Palodhi and Sen, 1983). The fungi enter the root tips and older roots through passive way i.e. natural wounds, nematode feeding and other wounds. After penetration, the fungi grow into the xylem tissue, march into other parts of the plant, clog the vessels and produce typical wilt symptoms in the foliage (Elsadig, 2015).

High level of nitrogen, along with low dose of potassium and

calcium helps in disease development. Sekhon and Singh (2007) studied the influence of edaphic factors on the fusarium wilt incidence on muskmelon and found that wilt incidence is more at 25°C than other temperature in sandy soil. The population density of *Fom* was highest at 25 ± 2°C. Singh *et al*. (2015) screened 20 germplasm lines of melon against fusarium wilt and root knot nematode. They found all the genotypes susceptible to both the pathogens. Wilt disease incidence was increased in the presence of *Meloidogyne incognita*. It states that wilting symptoms appeared earlier and were higher in combined inoculation indicating synergistic effect for reducing plant growth.

Fungal chlamydospores were produced for survival by colonizing itself around residues of muskmelon and other crops in rotation. In muskmelon for plant to plant infection irrigation water and contaminated farm equipments are reported to be the main cause. Since muskmelon is transplanted to main field, fungus can infect after transplanting. Seed contamination is also the way but found to be sporadic.

DISEASE MANAGEMENT

Fusarium wilt is difficult to control even if long crop rotations are followed as *Fom* colonizes the roots of a broad range of plants. Additionally, in the form of chlamydospores, *Fom* is able to survive in the soil owing to the existence of roots of alternate hosts. Therefore, crop rotation provides limited protection against melon wilt since pathogen survives even for more than 6 years without host plant. Soil disinfection using chloropicrin or methyl bromide, grafting of melon onto resistance rootstocks for instance *Cucumis pustulatus* (Liu *et al*., 2015), soil solarization with transparent polyethylene sheet, selection of raw material for agro-industrial composts (Blaya *et al*., 2015) and use of non- pathogenic strain of *Fusarium* to compete with pathogenic strain are quite promising preventive measures.

Kanaan *et al*. (2017) observed soil solarization along with compost (85% cattle manure + 15% wheat straw on dry weight basis) have suppressed effects on artificially inoculated melon plant. Shanthi and Vittal (2013) reported *Pseudomonas aeruginosa* (MIC2 and MTCC2581) inhibit radial growth of *F. oxysporum* 1 and 2, respectively. Some researchers also found use of *Gliocladium* spp. and *Trichoderma* spp., as an effective bio agent. *Streptomyces olivaceus* (strain 115) is also reported to have anti-fusarium activity by inhibiting pathogen mycelia which will reduce crop losses. *Bacillus subtilis* Y-IVI have antifungal lipopeptides and also have antagonistic effects by colonizing plant rhizosphere which prevent *Fom* invasion (Zhao *et al*., 2013). Haliscosamine (metabolite from Moroccan marine sponge, *Haliclona viscosa*) was reported to have similar effect as DESOGERME SP VEGETAUX@ and constitutes a potential candidate against *Fom* (Amraoul *et al*., 2015).

Kawaide (1975) in his review of rootstock utilization stated that in Japan for greenhouse culture, resistant melon varieties (Barnett Hill Favourite, Emerald Gem, Ooi) are used while *Cucurbita* sp. is effective for plastic house culture. Use of *C. moschata* has less vigorous growth and high graft compatibility. *C. africanus*, *C. anguria*, *C. metuliferus*, *C. prophetarum*, *C. subsericeus* and *C. zeyheri* could be used as a rootstock. Nisini *et al.* (2002) stated that there is influence of rootstock on disease resistance as well as quality and productivity of scion fruit. Rootstock PGM 96 - 05 and P360 were resistant to *Fom* race 1,2 and were best genotypes in improving productivity without harnessing quality of scion fruit. Similarly Crinò *et al.* (2007) documented four *Cucurbita* rootstocks resistant to race 1,2. On evaluation, RS 841 rootstock (*Cucurbita* sp.) performed better than *C. melo* rootstocks. Hirai *et al.* (2002) tested and released two melon rootstock cultivars (Dodai No. 1 and Dodai No. 2) resistant against *Fom* race 1,2y. Park *et al.* (2013) found better yield and quality fruit from Earl's elite grafter onto resistant melon rootstock as compared to non-grafted.

Chemical control of this disease is not feasible because of the residue problem in the soil and non target effects on other beneficial organism in the soil. The best alternative to control fusarium wilt in melon is developing durable resistant cultivars along with above discussed management practices. Host resistance may not provide complete resistance against all the races due to breakdown of resistance under high pathogenic population. The rate of developing new pathogenic race against new R gene is also fast which ultimately leads to susceptibility of cultivar even though they have R gene. Still host resistance is most efficient method of controlling wilt in melon. None of the above mentioned methods gives sufficient control under field condition. Though combining several methods, a sufficient level of control could be achieved.

CULTIVAR RESISTANCE

Screening of germplasm is a very crucial step in identifying a resistant genotype. Screening against local pathotypes may lead to the discovery of new resistance gene(s) which may overcome the new developing pathotypes. Different screening techniques have been reported by researchers. Root dip is a standard method while continuous-dip inoculation technique is rapid method to assess pathogenicity others are tray method in which whole tray is dipped and kept in conidial suspension. Even though the reliability of this method is limited, however, the exposure of intact seedling at a concentration of 1×10^6 conidia per millilitre, the method is not accurate at this range for excised seedling. Initially resistance against *Fom* race 1,2 in melon were reported in two Far East melon accessions Ogon-9 and Piboule. Further, race 1,2 resistance was mostly reported in *C. melo* sub sp. *agrestis*. While different geographical origin melon, studies depict that resistance was found only up to 3% against race 1,2 and most of them have Far East origin. These resistant accessions allow development of moderately resistant lines, for instance Isabelle, Nad-1 and Nad-2 and F_1 hybrids 'Adir'. Besides, Dodai No. 1' and 'Dodai No. 2' are two rootstock of melon which also show incomplete resistance to the race 1,2y.

A breeding line of Israel 'BIZ' was found to exhibit near-complete resistance against race 1,2 with root wounding even at 10^6 spores ml^{-1}concentration. This denotes that BIZ has stronger resistance than that of 'Isabelle' (Dhillon *et al*., 2011).

Japanese accessions, are morphologically similar with commercial Spanish types which were also reported to have high level of resistance to race 1,2 and at significant level of resistance in Spanish and Russian accessions. Ramos *et al.* (2015) aided melon breeding program with green fluorescence protein (GFP) technology by transforming *Fom* isolate and using for assay.

Chikh-Rouhou *et al.* (2010) found incomplete resistance against race 1,2 in BG-5384 (Portuguese accession) belonging to var. *cantalupensis*. So far, these resistant genes are being extensively used by breeder to produce resistant cultivars in melon. Since pathogen adapt relatively fast and follow boon and burst theory, so additional resistance sources are needed in near future to combat against them. Thus, researchers are eager to dwell upon novel gene of resistance to *Fom*. Patel *et al.* (2016) also identified eight accessions which were highly resistant and three accessions have moderate level of resistance against local *Fom* isolate. All the three differentials i.e. Ein Dor (No gene), Hemed (*Fom*-1 gene) and FM-65 (*Fom*-2 gene) were found to be susceptible against local *Fom* isolate. They concluded that new race or pathotype was present over there and resistant genotypes could have novel gene against local isolate that helps in building disease resistance. The above mentioned studies states that, resistance to *Fom* races 0, 1 and 2 is more common than that of race 1,2. Resistance genes were reported in different botanical varieties from various geographical origins and diverse germplasm *viz*. Far East, Iberian Peninsula and Middle East.

According to Flor hypothesis, for every virulent gene in pathogen there is a resistant gene in the host. But the evolution of pathogen is much faster than the host. Still a wide variability is present in the melon accession which is a treasure for many resistant gene(s). There is a need for regular screening of the germplasm for resistance gene (s) which can further be used in breeding programs. Asian germplasm needs to be investigated more for such disease resistance gene(s) as some centres around the world are working on lines viz. MR-1, PI 124112, PI 164723, etc which was reported from India.

RESISTANCE GENES AND THEIR INHERITANCE

Messiaen *et al.* (1962) were the pioneer in genetic studies of *Fom* resistance gene and depicted resistance in genotypes from French 'Cantaloupe Charentais' and later non-segregating cultivars were selected as 'Doublon' and 'Ve´drantais'. It was reported to have a dominant nature and named as *Fom*-1. In early seventies, *Fom-2*, an autonomous dominant gene was discovered on screening of Far East accessions. The *Fom-2* gene was characterized by the presence of NBS-LRR domains which encodes a protein for resistance gene. Resistance to race 0 was governed by *Fom*-1 and *Fom*-2 genes. Earlier, *Fom*-3, a dominant gene, responsible for resistance to races 0 and 2 in melon line 'Perlita-FR' was discovered. While Risser (1987) admitted that Perlita-FR have *Fom*-1 gene that controls resistance and to test allelism, susceptible plants were detected in the F_2 population of a cross "Perlita-FR × Doublon". A residual segregation was observed which resulted due to parent **"**Perlita-FR". *Fom*-1 and *Fom*-3 are reported to be tightly linked with each other and are supposed to be allelic. While there are some differences in the phenotypes of resistant genes, *Fom*-3 is reported to confer resistance to the same set of races as like *Fom*-1 (Oumouloud *et al*., 2013).

Other than *Fom*-1 and *Fom*-3, Oumouloud *et al.* (2010) symbolize *fom*-4, a recessive gene in Tortuga melon line that confers resistance to races 0 and 2. The extensive cultivation of cultivars having resistance due to *Fom*-1 may be variable. Thus resistance for race 0 and 2 can be used to strengthen using *Fom*-3 and *fom*-4. Hence Tortuga can be a new alternative source against race 0 and 2. Moreover, durability can be increased, if resistance is governed by few genes. Thus, *Fom*-1 and *Fom*-3/*fom*-4 could give better protection against race 2. *Fom*-3 might have different alleles at the same locus where *Fom*-1 gene is present. During inheritance study of *Fom* resistance gene, Oumouloud *et al.* (2010) reported resistance in Tortuga being controlled by one dominant and one recessive gene independently. Later with CAPS marker (**Table 2**) analysis, they found that 618-CAPS (0.9cM) was associated to *Fom*-1 gene and thus corroborated that Tortuga carries both dominant as well as recessive gene which is symbolized as *fom*-4.

Resistance with *Fom*-1 and *Fom*-2, was overcame by a new strain of *Fom* i.e. race 1,2. A complex nature of resistance was observed for *Fom* race 1,2 which was moreover due to recessive polygene and not race specific. In some Far East accessions, resistance was controlled by polygenic recessive gene against race 1,2. Herman and Perl-Treves (2007) used F_2 and backcross generations developed from BIZ × PI 414723 for studying the nature of *Fom* race 1,2 resistances. Result depicted the presence of two recessive complementary genes, labelled as *fom*-1, 2a and *fom*-1, 2b, which are required for attaining complete resistance. Later, they located resistance gene at a distal end of the LG II (opposite to the gene *a*, andromonoecious).

Vashisht and Singh (2013) studied the inheritance of fusarium wilt resistance in six basic generations of crosses Punjab Sunehri × KP_4HM-15 and Punjab Sunehri × IC-267379. Among parents, KP_4HM-15 and IC-267379 high level of resistance against unidentified local *Fom* isolate was noted. Recently, Schmidt *et al.* (2015) revealed undisclosed protein by using comparative genomics of *Fom* and recognised *Fom-2* gene in muskmelon. Along with other advanced techniques, genome sequencing was used for a set of *Fom*-2 strains to identify *AVRFOM2* which encodes the virulence protein which is further recognised by *Fom*-2 resistance gene. A counterpart of *AVRFOM2* results in protecting melon cultivars due to the presence of *Fom*-2 gene. They found that *AVRFOM2* have two residues of cysteine, which was small, but secreted protein and have fewer similarities with other fungi secreting such proteins. Identifying such secreted protein will not only help breeder to select fusarium wilt resistant cultivars but also monitor fungal population for deploying cultivars containing *Fom*-2 resistance gene in muskmelon susceptible field.

To understand the flow of resistance gene(s) in population, a rigid genetic study along with phenotyping must be done. A

durable resistance can be developed by combing major and minor gene into one background. Identification of such resistant gene(s) in short period may help breeder to speed-up breeding programme with molecular approach.

R GENE LINKED MARKERS

Identification of molecular markers linked with resistant genes served a valuable tool during selection of resistant genotypes. It helps in screening during period of disease escape or uncertain symptom expression under artificial inoculation tests. A plant which subsists *Fom* race 1 inoculation further more did not show disease symptoms after successive inoculation with *Fom* race 2, so marker set concurrent selection of resistance genes for more than one *Fom* races can be done. Developed polymorphic markers chop down the way to detect resistant genes from different species. Before its introgression into commercial variety, a complete characterization of these resistance genes is necessary. Further these gene(s) can be transferred through backcross or other conventional and biotechnological approaches.

Artificial inoculation followed by wilt symptoms was affected by genetics of plant itself, pathogenic *Fom* isolates and environment in which host and pathogen were interacting. However, host may produce abstruse symptom or escape disease symptoms after artificial inoculation, which may bring about false selection, thus reduce genetic gain in breeding cycle. Oumouloud *et al.* (2013) stated that to eliminate problem of false selection, resistance genes linked markers will be the way-out, even if, the inoculation test was not much consistent. Plant may get cross protection by inoculating with non-pathogenic race. Therefore, inoculated pathogenic race may hamper successive selection for an additional race or pathotype on same plant. That's why selection of resistant genes can be done simultaneously for more than two races with the help of molecular markers.

Wang *et al.* (2000) found some amplified fragment length polymorphism (AFLP) markers linked to the *Fom-2* locus by combining BSA technique and changed these markers into SCAR markers, labelled as 'AM' and 'FM' (**Table 2**). Further as indirect selection, these SCAR markers were confirmed on diverse melon accessions resistant to race 1 along with susceptible origins and melon breeders are using such markers in map based cloning of Fom-2 gene. Joobeur *et al.* (2004) identified the *Fom-2* resistance gene with positional cloning technique with 16 SSR and STS markers using a population of 159

RILs descended from a cross Védrantais and PI 161375. As reported in previous studies, two observations strongly signposted mapping of *Fom*-2 gene between STS178 and SSR154 at an analogous position and specified the DNA sequence formerly affirmed by Wang *et al.* (2000). Wang *et al.* (2011) revealed characteristics features of NBS-LRR R protein through protein family (Pfam) analysis, wherein two significant Pfam-A match structures were put forwarded. In addition, one Sfi1 C (spindle body associated protein C-terminus) domain and EAF (ELL-associated factor) family was also reported (Oumouloud *et al*., 2013). A low density

Table 2. *Fom* gene linked molecular markers in melon (modified from Oumouloud *et al*., 2013)

Marker Name	Type	Sequence (5'-3')	R. Enzyme	Ref.
***Fom*-2 gene**				
FM	SCAR	GAAGATGCAAAGAAAAAGAGAAG	-	Wang *et al.* (2000)
AM	SCAR	TCAATTATTAAACATTCTGATGCC CTTCATCACTATTCGAGGATGAC CTTTCTGCACACCAACCAAAAGG	-	
SSR138	SSR	GACACGACCTGATCCATGTG CCACATGTTGAATGATGAGGA	-	
SSR154	SSR	CCCTTCTGTCATTTGGCTTG CGTCAATTATTAAACATTCTGATGC	-	
STS178	SSR	TTCGTTCATTACTGCCGTAGG TCTGTGTTCCCTACCCCAAC	-	
SSR180	SSR	GGATTTGTTTGCGTCATTTTG GGGGCATTTTTGGTATTTTC	-	
SSR181	SSR	AAATCGAAGCCCAGTGAAAG TCTGGCTGGGAATATGATTG	-	
SSR184	SSR	AGCTTATGTCAACGAGGTTTGG CCTCCAACAAAGATGACACTG	-	
STS259	STS	CATTGATCCGAAACAATCCAG TTCGCCTCCCTTCAAGTATG	-	
SSR281	STS	AGTTTGAAGGTGCTGCTTGAC AGACAAGCCCACAACGAATC	-	Joobeur *et al*. (2004)
STS296	STS	CCACAAAAGGAGCTTGACC GCCAATTGCCCAAATCAG	-	
STS303	STS	CAAATTTTGGGGCGTTACAC ACTGGTCATGCTGGTGATTC	-	
STS308	STS	TGCAGCTATTCCATGGTCAG TAAAATAGGGCCCGAAACTG	-	
STS312	STS	GGAGGATTTGGGAAGTGAG TGTCCATACCTCCTCCAAGC	-	
STS411	STS	TTTCTAAAATTTACCATCATTGGAG AATGGCAAATTCAACCTTCAC	-	
SSR430	SSR	CCATCATGATTTGGAATGAATTAG CGTTGCAATTTGATCTTTTAATG	-	
SSR451	SSR	CGTGAGAAAGTACAATGATTGGTG GCCAAGCTAAGCAATTAGGC	-	
P458		TGAAAACTAAAAAGATGGCATGG TGCAATGGCAATTTCAAGG	-	
F R		GAGTCTATTGTTTCGCTTC TGCTCGTCTCTGGGTCACCTTC	-	
CAPS1	CAPS	TGCTCCTTTGGCTTCCTGT GAGTCTATTGTTTCGCTTCA	-	Wang *et al*. (2011)
CAPS2	CAPS	GGAAGTGAGGTGTTGAATT TACACATTGGTCCGTTAGAC	*Eco*R1	
CAPS3	CAPS	AGACGTAGCATTGCTTCTCTAG TGGCATCCTTCAGCACCTTC	*Xba*1	
Fom2-LRR1639		AGGGAAACGAGTTGAGAGAGCTAGA CGAGGATTCTTAACTAGCATGGA	-	
Fom2-R_{408}	SCAR	GAGAAATTGCAATGGGTGG TTACACTATTATTGCTCAACTTGC	-	Oumouloud *et al*. (2013)
Fom2-S_{342}	SCAR	ATGAAAAGAAAAGATAACGACGA ATTGCTCTAAGTTGATCATATTCTG	-	
***Fom*-1 gene**				
$SB17_{645}$	SCAR	AGGGAACGAGTTGAGAGAGCTAGA CGAGGATTCTTAACTAGCATGGA	-	
$SV01_{574}$	SCAR	TGACGCATGGAATGAAATAAA GCATGGCCAAGGTCGAATA	-	Oumouloud *et al*. (2008)
$SV06_{1092}$	SCAR	ACGCCCAGGTTACGAAGTCA ACGCCCAGGTTATCATATACACC	-	
S-TAG/GCC-470	STS	GAATTCTAGACTGAGCTTATAAACC TTAAGCCTAAAAGGAATGGCCCCC	-	
S-TCG/GGT-400	STS	GAATTCTCGTAAGAACCAAAATATT TTAAGGTTATATGAACTTGTCTTGAT	-	
C-TCG/GGT-400	CAPS	TTCAAAATCAAAGGAAATGCAA GGACCCAAACTTACCTACACTT	*Eco*R1	
CAPS2	CAPS	CAATTTTGGTTTCTTTGGATGG TTTCGAGGTTAGAGGTTGTCA	*Taq*1	Tezuka *et al*. (2009)
TAIL-F2		GCATATTATTGTCAATGGAACAACAAT TCTTTTGC	-	
TAIL-F3		GAACTTGTCTTGATTACGCCAATCGTAT AATTTC	-	
618-CAPS	CAPS	CCACCCACTTGAGATCCATCGTGAAA TGGATCTCAAGTGGGTGGCAAAAGAAT TGT	*Tfi*1	Oumouloud *et al*. (2010)
S-MRGH9	STS	CTGGAGCCCAAATGAACAAAC GCTGGAGCATTCTAGTAATGAAA	-	
CAPS3	CAPS	GGTTGGCGATCTCACTGGAG TTTACCAATTCCGCCCATCC	*Hae*III	Tezuka *et al*. (2011)
C-MRGH12	CAPS	GTTGGAGATGTTCCCTTGGA ACCTGGCAACTTTGGTTTTG	*Xmn*1	
C-MRGH13	CAPS	CGTCGGGTATGTCTCCATCT TGATGCTGCTGATGGACTTC	*Taq*1	
FA-15	SCAR	CCACCCATTCCCCATATTCC TGAGGAAGCAGGAGGGGAAC	-	Luongo *et al*. (2012)
160P4-T7	CAPS	TAGGGATGATAGCGGTCTGG GCTAGTTCGAGGCAATTGGA	*Tai*1	
RG10-1	CAPS	GGTTAGATGTTGAATTAATGG TCTCGAGTGTTTAGTGAGTTGG	*Dde*I	
RG-A	CAPS	CCTGTACTCTTGAAATCGAACAA TTGTGGAAGACTAAAAGAGGTTCA	*Swa*I	
RG9-2	CAPS	GCCATGCTTCTTCTCTGAGC CCAATTCTCTCAAACACCACTTT	*Dde*I	
RG-G	CAPS	TCTGTTGGAAGCGTTTGATG TTGGCTCCAAATCATTTAGCTT	*Bsu*R1	Bortman *et al*. (2012)
160P4-SP6	CAPS	AGCGTGTGGTATGCAGACAG CAAGAATCCAACACGGGAGT GCATGTTAGGGATTGTACATTC	*Taq*1	
NBS1-CAPS	CAPS	TTGTTGGCTTTTAGTTTTTCTGTCCATTT CGTTC TATTGCTAAAGCTGTTTTCAAAAGCG AACAAAAACTTTTCGATTTCCTAAGTT	*Alw261*	
62-CAPS	CAPS	GGAGAAGATGCTAGAGCCATTC AATCGGGCATCCTGTTTTGG	*Nco*I	
RG9-1		ATGAGTTTTGATAGTTTCATAAG TTACCAATTCCGCCCATCC	-	
RG9-3		TTGGTTTGGTTCAGGGTCC CTATTTACCATAATGAAGTTAC		Oumouloud *et al*. (2015)
Fom-1_{568}		ATGAGTTTTGATAGTTTCATAAG GAACACTCCCTTAGATACTT		

map with RAPDs and AFLPs was generated by Shojaeivan *et al*. (2012) from the F_2 population developed by crossing Khatooni (Iranian melon accession) and Charentais-Fom2 against fusarium wilt resistant gene race 1. They reported E-CA/M-CTG (AFLP marker) at a distance of 21 cM from *Fom*-2 gene in LG1. High density map is reported to make better understanding about *Fom*-2 resistant gene.

Fom-1 was mapped on proximal end of linkage group 9 (formely 5), at 2 cM from the PRSV resistance gene, Prv^2 (Pitrat, 1991). Brotman *et al*. (2012; **Table 2**) also developed two CAPS markers CMTC47, a SSR marker drag with *Fom*-1 locus in F_2 segregating population (originated from a cross between Charentais-*Fom*-1 and TRG-1551 and combined

BSA utilizing RAPD markers to develop SCAR markers), which allowed Oumouloud *et al*. (2008) to develop markers in the region around this marker. Three decamers were identified after screening 400 RAPD markers. These primers were sequenced and converted to SCAR markers $SB17_{645}$ and $SV01_{574}$, which were found to be present in Charentais-*Fom*1 (resistant parent) at a distance of 645 and 574 bp, respectively (**Table 2**). Whilst Tezuka *et al*. (2009) used population developed from a cross between P11 and MR-1 for construction of linkage map and they allocated *Fom*-1 gene to linkage group 7. AFLP markers were identified, which were converted to 2 STS and 2 CAPS markers (**Table 2**). Further, four new DNA markers were developed, which were interconnected with *Fom*-1 locus. Tezuka *et al*. (2011) recognized *Fom*-1 gene linkage analysis in 125 individuals derived from the cross between P11 and MR-1 indicated the presence of resistance gene between marker C-MRGH12 (0.4 cM) and 62-CAPS (1.2 cM). They reported newly developed marker on the same linkage group with different set of SSR markers.

Brotman *et al*. (2012) located *Fom*-1 resistance genes between RG-G and RG9-2 markers and *Prv* between RG-A and RG10-1. Later Oumouloud *et al*. (2015) used three markers (**Table 2**) to clone and sequence DNA and cDNA of *Fom*-1 from several melon accessions. Eight nucleotide substitutions were observed within *Fom*-1 regions, out of which four were dissimilar. RT-PCR results followed Flor hypothesis, which shows that the *Fom*-1 expression was tempted by inoculation with *Fom* race 2. On this basis, further they generated two CAPS markers, which demonstrated the use of these functional CAPS markers in marker assisted breeding programs. Sebastiani *et al*. (2017) studied transcriptome analysis against *Fom*-1.2 pathosystem in susceptible genotype Charentias-T and resistant genotype NAD at 24 and 48 hrs post inoculation. The expression profile identified early defense response in NAD as compared to Charentias-T. They added FRP1/SKP1 interaction which was lacking which prevent activation of cell wall degrading enzyme and thus prevent entry of pathogen into host xylem vessel. In the 21st century, biotechnology has demonstrated its irrevocable contribution in the development of resistant genotypes. Different methods like bulk segregants analysis (BSA), marker assisted selection (MAS), background and foreground selection along with conventional breeding technique reduce the gap of crop failure across the world.

FUTURE AREA OF RESEARCH

Since preventive measure for MFW is not effective all the time, disease resistance breeding is one of the most important breeding objectives from last few decades. In India, the melon growing area like Punjab, Rajasthan, Uttar Pradesh, Maharashtra and Karnataka were infected with *Fusarium* pathogen. Till date no pathogenic race was identified or characterized from India. Being a center of diversity, Indian germplasm have resistance and could contribute to strengthen further as like MR-1. A public-private partnership should be considered for exchange of germplasm and cultivar development. In the world, a complementary gene for *Fom*-1 were identified which may strengthen the genetic resistance against race 0 and 2 if brought under one background**.** According to Flor hypothesis, host plant have complementary resistance gene against virulent gene of the pathogen, so there is probability of having resistance gene against race 1,2. As melon fusarium wilt is a complex disease, criteria for determining the virulence needs to be worked out; role of spore in virulence, survival of pathogen and relationship with other soil borne pathogen and role of seed and root exudates in resistant and susceptible accessions. Different NBS-LRR class of R gene should be searched throughout melon accessions to overcome this problem. A role of various biological agents needs to be studied further. There are many unknown aspects to the study of fusarium wilt in melon. We hope that additional advances will be made, building on knowledge of the characteristics of the pathogen and the physiology and genetics of plant resistance. Still resistance to race 1,2 was not confirmed on molecular basis. In future, race *Fom*-1,2 (Y and W) resistance gene linked marker may break the production plateau and overcome all the existing pathotypes.

REFERENCE

Amraoul, B. E., Blard, J. F., Ikbal, F. E., Wahidi, M. E., Kandil, M., Amraoul, M. E. and Fassouane, A. 2015. Activity of Haliscosamine against *Fusarium oxysporum* f. sp. *melonis*: *in vitro* and *in vivo* analysis. *SpringerPlus* pp. 1-5. http://doi.org/ 10.1186/s40064-015-0797-x

Anonymous 2014. *Vegetables and Melons Outlook*. http://faostat.fao.org

Anonymous 2016. Indian Horticulture Database. *National Horticulture Board* pp 4.

Armstrong, G. M. and Armstrong, J. K. 1978. *Formae speciales* and races of *Fusarium oxysporum* causing wilts of the *Cucurbitaceae*. *Phytopathology* **68**: 19-28. DOI: 10.1094/Phyto-68-19

Baayen, R. P. 2000. Diagnosis and detection of host-specific forms of *Fusarium oxysporum*. *EPPO Bulletin* **30**: 489-491. http://doi.org/10.1111/j.1365-2338.2000.tb00935.x

Blaya, J., Lloret, E., Ros, M. and Pascual, J. A. 2015. Identification of predictor parameters to determine agro-industrial compost suppressiveness against *Fusarium oxysporum* and *Phytophthora capsici* diseases in muskmelon and pepper seedlings. *Journal of the Science of Food and Agriculture* **95**: 1482-1490. http://doi.org/ 10.1002/jsfa.6847

Booth, C. 1971. *The genus Fusarium*. Common wealth Mycological Institute, Kew, pp 237.

Brotman, Y., Normantovich, M., Goldenberg, Z., Zvirin, Z., Kovalski, I., Stovbun, N., Donniger, T., Bolger, A.M., Troadec, C., Bendahmane, A., Cohen, R., Katzir, N., Pitrat, M., Dogimont, C. and Perl-Treves, R. 2012. Dual resistance of melon to *Fusarium oxysporum* races 0 and 2 and to Papaya ring-spot virus is controlled by a pair of head-to-head oriented NB-LRR

genes of unusual architecture. *Molecular Plant* **6**: 235238. https://doi.org/10.1093/mp/sss121

Chattopadhyay, C. and Sen, B. 1996. Integrated management of wilt of muskmelon by organic soil amendments. *Indian Phytopathology* **44**:476-79.

Chikh-Rouhou, H., Gonzalez-Torres, R., Oumouloud, A. and Alvarez, J. M. 2011. Inheritance of race 1,2 Fusarium wilt resistance in four melon cultivars. *Euphytica* **182**: 177-186. http://doi.org/ 10.1007/s10681-011-0411-4

Chikh-Rouhou, H., Rafael, G. T. and Alvarez, J. M., 2010. Screening and morphological characterization of melon for resistance to *Fusarium oxysporum* f. sp. *melonis* Race 1,2. *HortScience* **45**: 1021-1025.

Chupp, C. 1930. Fusarium wilt of muskmelon. *Plant Disease Report* **14**: 160.

Crinò, P., Bianco, C. L., Rouphael, Y., Colla, G., Saccardo, F. and Paratore, A. 2007. Evaluation of rootstock resistance to fusarium wilt and gummy stem blight and effect on yield and quality of a grafted 'Inodorus' melon. *HortScience **42**:* 521-525.

Dhillon, N. P. S., Monforte, A. J., Pitrat, M., Pandey, S., Singh, P. K., Reitsma, K. R., Garcia-Mas, J., Sharma, A. and Mc Creight, J. D. 2011. Melon Landraces of India: Contributions and Importance. In: *Plant Breeding Reviews* (Ed.:Janick, J.). John Wiley and Sons, USA, **35**: 85-150. Http://doi.org/10.1002/ 9781118100509.ch3

Dhillon, N. P. S., Singh, J., Fergany, M., Monforte, A. J. and Sureja, A. K. 2009. Phenotypic and molecular diversity among landraces of snapmelon (*Cucumis melo* var. *momordica*) adapted to the hot and humid tropics of eastern India. *Plant Genetic Resources* 7, 291-300. https://doi.org/10.1017/S1479262109990050

Egel, D. S. and Martyn, R. D., 2007. Fusarium wilt of watermelon and other cucurbits. *Plant Health Instructor.* https://doi.org/10.1094/PHI-I-2007-0122-01

Elsadig, E. A. 2015. *A Study on Fusarium oxysporum f. sp. fabae wilt in Broad bean (Vicia faba L.) In Wad Ramly Area* (Doctoral dissertation, UOFK). http://khartoumspace.uofk.edu/handle/123456789/11060

Ganesan, J. I. 1991. Botanical nomenclature of Indian melons (Cucumis melo L.). *Plant Breeding News Letter* **1**: 2.

Gordon, T. R. and Martyn, R. D., 1997. The evolutionary biology of *Fusarium oxysporum*. *Annual Review of Phytopathology* **35**: 11-28.

Herman, R. and Perl-Treves, R. 2007. Characterization and inheritance of a new source of resistance to *Fusarium oxysporum* f. sp. *melonis* race 1,2 in *Cucumis melo*. *Plant Disease* **91**: 1180-1186. http://dx.doi.org/ 10.1094/PDIS-91-9-1180.

Hirai, G., Nakazumi, H., Yagi, R. and Nakano, M. 2002. Fusarium wilt (race 1, 2y) resistant melon (*Cucumis melo*) rootstock cultivars 'Dodai no. 1 and Dodai no. 2. In II *International Symposium on Cucurbits* **588**: 155-160.

Joobeur, T., King, J. J., Nolin, S. J., Thomas, C. E. and Dean, R. A. 2004. The fusarium wilt resistance locus *Fom*-2 of melon contains a single resistance gene with complex features. *Plant Journal* **39**: 283-297. http://doi.org/10.1111/j.1365-313X.2004.02134.x

Joshi, M., Srivastava, R., Sharma, K. and Prakash, A. 2013. Isolation and characterization of *Fusarium oxysporum*, a wilt causing fungus, for its pathogenic and non-pathogenic nature in tomato (*Solanum lycopersicum*). *Journal of Applied and Natural Science* **5**: 108-117.

Kanaan, H., Medina, S. and Raviv, M. 2017. The effects of soil solarization and compost on Soil suppressiveness against *Fusarium oxysporum* f. sp. *melonis*, *Compost Science & Utilization*. pp 1-5.

Kannaiyan, S. and Prasad, N. N. 1975. Saprophytic activity of muskmelon wilt fungus in soil. *Indian phytopathology* **28**: 167-170.

Kawaide, T. 1975. Breeding for disease resistance of vegetable crops in Japan. I. Cucurbits. *Japan Agricultural Research Quarterly* **9**: 212-216.

Liu, B., Ren, J., Zhang, Y., An, J., Chen, M., Chen, H., Xu, C. and Ren, H. 2015. A new grafted rootstock against root-knot nematode for cucumber, melon and watermelon. *Agronomy Sustain. Dev*. **35**: 251-259. http://doi.org/10.1007/s13593-014-0234-5

Luongo, L., Vitale, S., Haegi, A. and Belisario, A. 2012. Development of scar markers and pcr assay for *Fusarium oxysporum* f. sp. *melonis* race 2 specific detection. *Journal of Plant Pathology* **94**: 193-199. http://dx.doi.org/10.4454/jpp.fa.2012.010.

Mahdikhani, M. 2016. Genetic variability among *Fusarium oxysporum* isolates from melon (*Cucumis melo*) in Qazvin province, Iran. *Horticultural Biotechnology Research* **2**: 1-7. 0.19071/jhcbt.2016.v2.2996.

Mathur, K. and Shekhawat, K. S. 1992. Varietal screening of muskmelon against wilt. *Indian Journal of Mycology and Plant Pathology* **22**:78-79.

Megnegneau, B. and Branchard, M. 1988. Toxicity of culture filtrates of different Fusarium f. sp. melonis pathotypes on muskmelon, In: *Eucarpia* Meeting *Cucurbitaceae 88* (Eds. : Risser, G. and Pitrat, M.). Avignon-Montfavet (France) 85.

Messiaen, C.M., Risser, G. and Pecaut, P. 1962. Etude des plantes résistantes au *Fusarium oxysporum* f. sp. *melonis*. Dans la variété de melon Cantaloup Charentais. *Annales de l'amélioration des plantes* **12**: 157-164.

Moretti, A., Belisario, A., Tafuri, A., Ritieni, A., Corazza, L. and Logrieco, A. 2002. Production of beauvericin by different races of *Fusarium oxysporum* f. sp. *melonis*,

the Fusarium wilt agent of muskmelon. *European Journal of Plant Pathology* **108**: 661-666.

Nisini, P. T., Colla, G., Granati, E., Temperini, O., Crino, P. and Saccardo, F. 2002. Rootstock resistance to fusarium wilt and effect on fruit yield and quality of two muskmelon cultivars. *Scientia Horticulturae* **93**: 281-288.

Oumouloud, A., Arnedo-Andre´s, M. S., González-Torres, R. and A´lvarez, J. M. 2010. Inheritance of resistance to *Fusarium oxysporum* f. sp. *melonis* races 0 and 2 in melon accession Tortuga. *Euphytica* **176**: 183-189. http://doi.org/ 10.1007/s10681-010-0201-4

Oumouloud, A., Arnedo-Andres, M. S., Gonzalez-Torres, R. and A´lvarez, J. M. 2008. Development of molecular markers linked to the *Fom-1* locus for resistance to Fusarium race 2 in melon. *Euphytica* **164**: 347-356. http://doi.org/10.1007/s10681-008-9664-y

Oumouloud, A., El-Otmani, Chikh-Rouhou, H., Claver, A. G., Torres, R. G., Perl-Treves, R. and Alvarez, J. M. 2013. Breeding melon for resistance to fusarium wilt: recent developments. *Euphytica* **192**: 155-169. http://doi.org/10.1007/s10681-013-0904-4

Oumouloud, A., Mokhtari, M., Chikh-Rouhou, H., Arnedo-Andres, M. S., Gonzalez, T. R. and Alvarez, J. M. 2012. Characterization of the fusarium wilt resistance *Fom*-2 gene in melon. *Molecular Breeding* **30**: 325-334.

Oumouloud, A., Otmani, M. E. and Alvarez, J. M. 2015. Molecular characterization of *Fom-1* gene and development of functional markers for molecular breeding of resistance to *Fusarium* race 2 in melon. *Euphytica* pp. 1-11. http://doi.org/10.1007/s10681-015-1420-5

Palodhi, P. R. and Sen, B. 1983. Perpetuation of cucurbit wilt pathogen in riverbed cultivation. *Indian Journal of Mycology and plant Pathology* **13**: 164-168.

Park, D.K., Son, S.H., Kim, S., Lee, W.M., Lee, H.J., Choi, H.S., Yang, E.Y., Chae, W.B., Ko, H.C. and Huh, Y.C., 2013. Selection of melon genotypes with resistance to Fusarium wilt and Monosporascus root rot for rootstocks. *Plant Breeding and Biotechnology* **1**: 277-282.

Patel, S. A., Vashisht, V. K. and Kaur, J. 2016. Identification of sources of resistance against fusarium wilt in Indian melon (*Cucumis melo* L.). *Environment, Ecology and Conservation* **22**: 99-104.

Perchepied, L., Dogimont, C. and Pitrat, M. 2005. Strain specific and QTLs involved in the control of partial resistance to *Fusarium oxysporum* f. sp *melonis* race 1,2 in a recombinant inbred line population of melon. *Theoretical Applied Genetics* **111**: 65-74. http://doi.org/10.1007/s00122-005-1991-y

Pitrat, M. 1991. Linkage groups in *Cucumis melo* L. *Journal of Heredity* **82**: 406-411. https://doi.org/10.1093/oxfordjournals.jhered.a111112

Ramos, B., Lopez, G. and Molina, A. 2015. Development of a *Fusarium oxysporum* f. sp. *melonis* functional GFP Fluorescence tool to assist melon resistance breeding programmes. *Plant Pathology* pp 1-9. http://doi.org/10.1111/ppa.12367

Risser, G. 1987. Controversy on resistance to Fusarium wilt in 'Perlita' (*Cucumis melo* L.). *Cucurbit Genetics Cooperative Report* **10**: 60-61.

Risser, G., Banihashimi, Z. and Davis, D. W. 1976. A proposed nomenclature of *Fusarium oxysporum* f. sp. *melonis* races and resistance genes in *Cucumis melo*. *Phytopathology* **66**: 1105-1106. http://doi.org/10.1094/Phyto-66-1105

Schmidt, S. M., Lukasiewicz, J., Farrer, R., Dam, P. V., Bertoldo, C. and Rep, M. 2015. Comparative genomics of *Fusarium oxysporum* f. sp. *melonis* reveals the secreted protein recognized by the *Fom*-2 resistance gene in melon. *New Phytology* pp. 1-12. http://doi.org/10.1111/nph.13584

Sebastiani, M. S., Bagnaresi, P., Sestili, S., Biselli, C., Zechini, A., Orrù, L., Cattivelli, L. and Ficcadenti, N. 2017. Transcriptome analysis of the melon- *Fusarium oxysporum* f. sp. *melonis* race 1.2 pathosystem in susceptible and resistant plants. *Frontiers Plant Science* **8**: 362. http://doi.org/10.3389/fpls.2017.00362.

Sekhon, R. K. and Singh, P. P. 2007. Influence of edaphic factors and cultural practices on the development of fusarium wilt of muskmelon. *Journal of Research* **44**: 50-54.

Sen, B., Majumdar, S. and Kumar, S. 1999. Fungal and bacterial diseases of cucurbits. In: *Diseases of Horticulture Crops Vegetables, Ornamentals and Mushrooms* (Eds.: Verma, L.R. and Sharma, R.C.) Indus Pub. Co., New York: 237-239.

Shanthi, A. T. and Vittal, R. R. 2013. Biocontrol protentials of plant growth promoting rhizobacteria against fusarium wilt disease of cucurbit. Inter. *Journal of Phytopathology* **2**: 155-161.

Shojaeiyan, A., Ghareyazie, B., Arzani, K., Rahimian, H. and Lelley, T. 2012. Linkage map in Iranian melon (*Cucumis melo* L.) with emphasis on Fusarium wilt resistance gene. In *Cucurbitaceae 2012. Proceedings of the Xth EUCARPIA Meeting on Genetics and Breeding of Cucurbitaceae, Antalya, Turkey, 15-18 October, 2012* pp. 514-519. University of Cukurova, Ziraat Fakultesi.

Singh, H., Dhillon, N. K., Kaur, J. and Vashisht, V. K. 2015. Reaction of muskmelon genotypes to *Meloidogyne incognita* and *Fusarium oxysporum* f. sp. *melonis*. *Plant Disease Research* **30**: 163-168.

Tezuka, T., Waki, K., Kuzuya, M., Ishikawa, T., Takatsu, Y. and Miyagi, M. 2011. Development of new DNA markers linked to the fusarium wilt resistance locus

Fom-1 in melon. *Plant Breeding* **130**: 261-267. http://doi.org/10.1111/j.1439-0523.2010.01800.x

Tezuka, T., Waki, K., Yashiro, K., Kuzuya, M., Ishikawa, T., Takatsu, Y. and Miyagi, M. 2009. Construction of a linkage map and identification of DNA markers linked to *Fom*-1, a gene conferring resistance to *Fusarium oxysporum* f. sp. *melonis* race 2 in melon. *Euphytica* **168**: 177-188. http://doi.org/10.1007/s10681-009-9881-z

Vashisht, V. K. and Singh, B. 2013. Inheritance of fusarium wilt resistance in muskmelon (*Cucumis melo* L.). National symposium on abiotic and biotic stress management in vegetable crops, IIVR, Varanasi, India. (http://conference.isvs.org.in/Index.php/conf_Doc/ncab/paper/view/288).

Vidhi, J. (Assessed on 6/10/2017) Vegetable seed industry in India. http://www.biologydiscussion.com/india/vegetable-seed-industry-in-India/67963.

Wang, S., Yang, J. and Zhang, M. 2011. Developments of functional markers for *Fom-2*-mediated fusarium wilt resistance based on single nucleotide polymorphism in melon (*Cucumis melo* L.). *Molecular Breeding* **27**: 385-393. http://doi.org/10.1007/s11032-010-9439-8

Wang, Y. H., Thomas, C. E. and Dean, R. A. 2000. Genetic mapping of a fusarium wilt resistance gene *Fom-2* in melon (*Cucumis melo* L.). *Molecular Breeding* **6**: 379-389. http://doi.org/10.1023/A:1009671925793

Zeven, A. C. and Zhukovsky, P. M. 1975. *Dictionary of cultivated plants and their centers of diversity*. Center for Agricultural Publishing and Documentation, Wageningen. pp. 30.

Zhao, B., Yan, J., Zhang, S., Liu, X. and Gao, Z. 2014. Phylogeny and pathogenicity of *Fusarium* spp. isolated from greenhouse melon soil in Liaoning Province. *Saudi Journal of Biological Science* **21**: 374-379. http://doi.org/10.1016/j.sjbs.2013.10.004

Zhao, Q., Ran, W., Wang, H., Li, X., Shen, Q., Shen, S. and Xu, Y. 2013. Biocontrol of *Fusarium* wilt disease in muskmelon with *Bacillus subtilis* Y-IVI. *BioControl* **58**: 283-292. http://doi.org/10.1007/s10526-012-9496-5

Zink, F. W. 1991. Origin of Fusarium wilt resistance in Texas AES muskmelon cultivars. *Plant Disease* **75**: 24-26.

Zink, F. W. and Thomas, C. E. 1990. Genetics of resistance to *Fusarium oxysporum* f. sp. *melonis* races 0, 1, and 2 in muskmelon line MR-1. *Phytopathology* **80**: 1230-1232.

Zuniga, T. L., Zitter, T. A., Gordon, T. R., Schroeder, D. T. and Okamoto, D. 1997. Characterization of pathogenic races of *Fusarium oxysporum* f. sp. *melonis* causing fusarium wilt of melon in New York. *Plant Disease* **81**: 592-596.