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Issue Article

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CUCUMIS MELO L. UNTAPPED SOURCES OF FUSARIUM WILT RESISTANCE

Abstract: *Melon crops are severely impacted by fungal diseases like Fusarium wilt, powdery mildew, Monosporascus root rot, and charcoal root, caused by Macrophomina phaseolina. These diseases reduce fruit quality and yield, necessitating harsh chemical treatments. Fusarium wilt is one of the most dangerous diseases in the Mediterranean region, causing 10-30% of global melon crop loss each year. Fusarium oxysporum f. sp. melonis is the most well-known species affecting melon crops, with all four pathogenic races found in the Mediterranean basin, including Tunisia. Melon breeding projects aim to create genotypes resistant to race 1.2 and other races of the virus, which has expanded throughout the Mediterranean region and poses a threat to melon farming. Screening techniques, such as fake inoculations, have been employed to identify resistance sources. Functional markers for Fom-1 and Fom-2 have been created, and functional markers for Fom-1 and Fom-2 have been identified.*

Key words: *mildew, monosporascus root rot, charcoal root, melon farming, fungal diseases*

Language: *English*

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Introduction

Melon crops (Cucumis melo L.) are severely impacted by a number of fungal diseases, including Fusarium wilt, powdery mildew, Monosporascus root rot, and charcoal root, which are brought on by Macrophomina phaseolina [1]. These diseases reduce fruit quality and yield and necessitate the use of harsh chemical treatments. Fusarium wilt is one of the most dangerous melon crop diseases in the Mediterranean region [3]. Considerable damages caused by Fusarium wilt have been reported in many parts of this area, including Tunisia. The loss of output from plant death and the decline in marketable fruits are viewed as the most visible damages. According to estimates, Fusarium wilt causes 10–30%, and occasionally even 100%, of the global melon crop loss each year [2].

Fusarium oxysporum f. sp. melonis W.C. Snyder and H.N. Hansen (Fom) is the most well-known species affecting melon crops, and all four of its pathogenic races [9] have been found in the Mediterranean basin, including Tunisia [6]. Fusarium wilt is caused by a number of soil-borne fungal species and formae speciales that belong to the genus Fusarium [5]. The main reason that Fusarium wilt is one of the most challenging diseases to control is its soil-borne lifestyle, which allows the pathogen to survive in the soil as chlamydospores for decades.

A primary goal of melon breeding projects is to create genotypes that are resistant to both race 1.2 and the other races of the virus, as the detrimental race 1.2 has expanded throughout the Mediterranean region and is now posing a threat to melon farming [1, 6]. Race 1.2 resistance appears to be polygenically inherited and quantitative [1]. Two complimentary recessive alleles, fom 1.2a and fom 1.2b, govern the melon breeding line BIZ's near-complete resistance to Fom race 1.2, according to Herman and Perl-Treves [2]. Due to the detection of epistatic effects and

somewhat significant broad-sense heritabilities, Chikh-Rouhou et al. [3] indicated that resistance to race 1.2 is under complicated genetic regulation.

A number of screening techniques have been employed to find the sources of resistance, with fake inoculations being the most effective [7, 8]. Reliable sources of resistance could be identified by combining the use of molecular markers with artificial inoculations. The two dominant Fom-1 and Fom-2 genes have been associated with a number of molecular markers [5,6]. The Tunisian melon cultivars could not be tested for consistent polymorphism using those markers, which are exclusively useful in the particular melon genetic backgrounds in which they were created [2,1]. Based on a functional nucleotide polymorphism found between the susceptible and resistant Fom-2 alleles and a single nucleotide polymorphism within the coding region of the Fom-1 locus encoding for resistance to Fom, respectively, Oumouloud et al. [4,5] created functional markers for Fom-1 and Fom-2.

Although there are currently no useful molecular markers for the Fom 1.2 race, Perchepied et al. [2,7] found a total of 9 recessive QTLs associated with resistance, located on 5 linkage different groups, and Herman et al. [2,8] detected a major QTL for such resistance. These functional markers [4,2] are highly predictive of the phenotype because they target the functional polymorphism within a desired gene, overcoming the problem of recombination and linkage between marker and trait [2,6].

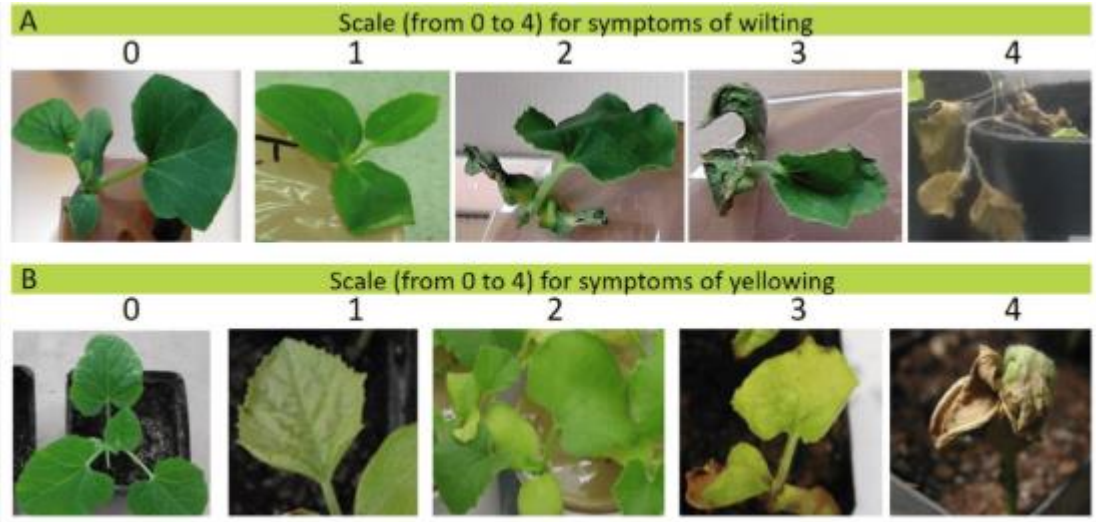

Figure 1. Typical symptoms of Fusarium wilt disease in melon seedlings (A) symptoms of wilting and (B) symptoms of yellowing. Scale used for symptom evaluation: 0 = no symptoms; 1 = beginning of wilting or yellowing on leaves; 2 = leaves heavily affected by wilting or yellowing; 3 = all leaves completely wilted or yellowed; 4 = death of the plant. (Hela Chikh-Rouhou)

The melon accessions' seeds were planted in sanitized sand and cultivated in a growth chamber at 24 °C for 16 hours each day under light and 16 °C for 8 hours each day under dark conditions. When the seedlings were about 15 days old and at the first true leaf stage, they were carefully uprooted from the sand and their roots were cleaned with tap water. Artificial inoculation was performed for each melon accession and Fom race by immersing the roots, which had been previously cut at 0.5 cm, in a conidial suspension (106 conidia/mL) of the relevant race for 15 minutes [8].

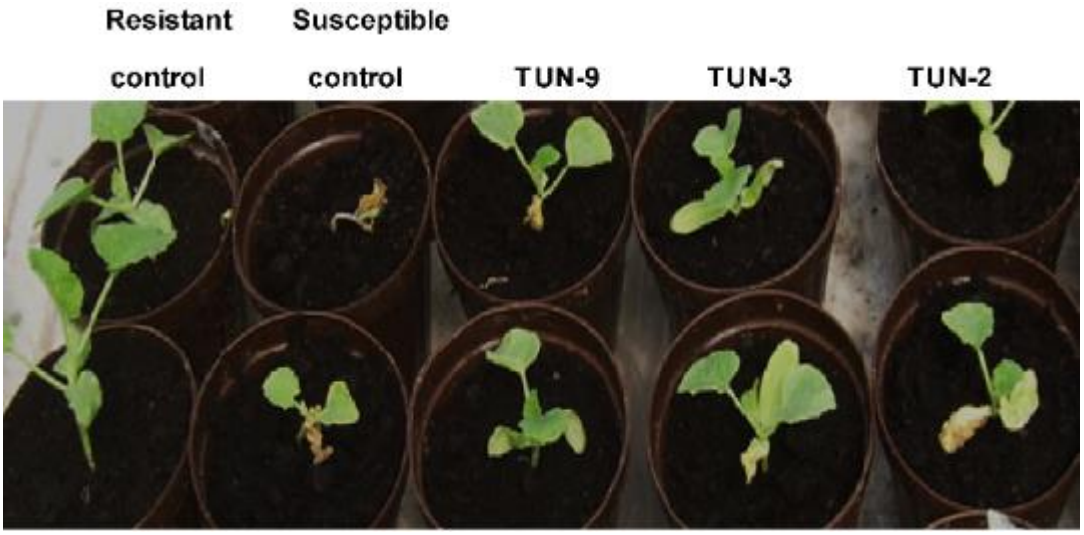

Figure 2. Symptom evaluation of yellowing caused by Fusarium wilt disease in some melon accessions inoculated with *Fom*0124 isolate belonging to race 2 in comparison to the susceptible and resistant controls. (Hela Chikh-Rouhou)

After being transplanted straight into plastic pots filled with sterile substrate (one-part soil, one-part sand, and one-part peat), the inoculated seedlings were kept in a controlled growth environment for a month at 28–20 °C (day/night) with a 16:8 h (light/dark) photoperiod. The plants were distributed using a completely randomized strategy, with each plant serving as a replicate for each isolate inoculation assay.

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