Article

Evaluation of the response of different tomato genotypes to *Fusarium oxysporum* breed 3

Odilón Gayosso-Barragán[1, 5]
Alfonso López-Benítez[2]
José Ángel Marroquín-Morales[3§]
Kelvin Lopéz-Aguilar[4]
Dreyli Maygualida Hidalgo-Ramos[5]
Griselda Chávez-Aguilar[1]

[1]CENID-AF-INIFAP. Highway Ojuelos-Lagos de Moreno km 8.5, Ojuelos, Jalisco, Mexico. ZC. 47540. (gayosso.odilon@inifap.gob.mx; chavez.aguilar@inifap.gob.mx). [2]Department of Plant Breeding-Antonio Narro Autonomous Agrarian University. Saltillo, Coahuila, Mexico. ZC. 25315. (alfopezbe_2000@hotmail.com). [3]Norman E. Borlaung Experimental Field-INIFAP. Street Norman E. Borlaung km 12, Obregon City, Sonora, Mexico. ZC. 85000. [4]Postgraduate in Protected Agriculture-Antonio Narro Autonomous Agrarian University. Saltillo, Coahuila, Mexico. ZC. 25315. (uaaan88@hotmail.com). [5]Postgraduate in Agricultural Sciences-Antonio Narro Autonomous Agrarian University. Peripheral Raúl López Sánchez, Torreón, Coahuila, Mexico. ZC. 27054. (dreyli_maygualida@hotmail.com).

[§]Corresponding author: suker_588@hotmail.com.

Abstract

One of the phytosanitary problems limiting the production of tomato crop (*Solanum licopersicum* L.) worldwide and nationally is vascular wilt caused by *Fusarium oxysporum* f. sp. *lycopersici* (*Fol*), causing large economic losses. The objective of this study was to determine the variability in virulence of *Fusarium* strains isolated from tomato plants with typical symptoms of vascular wilt in tomato-producing plots in Hidalgo state and to assess the incidence and severity of *Fol* breed 3 in seven tomato genotypes. For the identification of breeds pathogenicity tests were performed on differential materials, in addition a disease index was used to calculate the area under the disease progress curve (ABCPE), which allowed to identify genotypes with tolerance to *Fol*. *Fol* breeds 2 and 3 were identified in a ratio of 33 and 67%, respectively, in addition information is provided about behavior of seven tomato genotypes, of which Y53 and D3 had the lowest incidence of *Fol*, highlighting the importance of being used in future work on resistance to this pathogen.

Keywords: genetic resistance, inoculation, pathogenicity.

Reception date: December 2020
Acceptance date: March 2021

Introduction

Tomato (*Solanum lycopersicum* L.) is one of the most cultivated vegetables in the world and the most economically valuable (Srinivas *et al*., 2019). The area harvested globally in 2017 was 4.8 million hectares with a production of 182.3 million tons, with China, India, Turkey, the United States being the main producer countries of this vegetable worldwide (FAOSTAT, 2018). Mexico ranks eighth in production, with a total area planted at 49 415 ha in 2018 and production of 3.8 million tonnes, overall average yield of 76.83 t ha^{-1} and as the main tomato-producing states, Sinaloa is located with 813 095 t, San Luis Potosí 380 627 t, Michoacán 226 762 t, Zacatecas 182 019 t, Jalisco 144 443 t and Baja California Sur with 137 341 t (SADER-SIAP, 2018).

Among the most important factors that affect the normal development of this crop are infectious diseases caused by fungi, which occur in most areas of tomato production in Mexico. One of the diseases that affect tomato production is vascular wilt, caused by *Fusarium oxysporum* f. sp. *lycopersici*, Snyder and Hansen (*Fol*) with reports of decrease in yields of up to 60% (do Amaral *et al*., 2008).

The characteristic symptoms of the disease appear due to the blockage of conduction vessels caused by the accumulation of fungal hypfas and by factors derived from host-pathogen interaction, such as the release of toxins, gels and formation of tilides. After this, the characteristics of the disease appear, such as the epinasty of the leaves, the whitening of the veins, the withering, defoliation and finally the death of the host plant. During this phase, the fungus spreads through the parenchymal tissue and begins to sporulate abundantly on the surface of the plant (Srinivas *et al*., 2019).

The fungus has great genetic capacity to generate variants in the appearance and coloration of the colonies, as well as in the production of microconidia and chlamydospores. To date, three physiological races 1, 2 and 3 have been generated, which have been identified in the loci: *i-1, i-2* and *i-3*, conferring resistance to the pathogen, through dominant genes (Scott *et al*., 2004; Panthee and Chen, 2010; Malafaia *et al*., 2013).

The original descriptions of the breeds of *Fol* 1, 2 and 3 appeared in 1886 in England, in 1939 in the United States of America and in 1978 in Australia, respectively (Cai *et al*., 2003). In Japan, races 1, 2 and 3 were registered in Fukuoka in 1905, 1966 and 1997, respectively (Komada *et al*., 2011). In Mexico the presence of race three has been reported in Culiacán, Sinaloa (Valenzuela-Ureta *et al*., 1996; Carrillo-Fasio *et al*., 2003; Ascencio-Álvarez *et al*., 2008; Sánchez-Peña *et al*., 2010) and San Luis Potosí (Hernández-Martínez *et al*., 2014).

According to Inami *et al*. (2012), *Fol* race 2 emerged from race 1, by the loss of the *avr1* gene or through the loss of gene function, by the insertion of a transposon, while race 3 emerged when a point of mutation occurred in the *avr2* gene. Different breeds of the fungus carry in several combinations three avirulence genes: *avr1*, *avr2* and *avr3*, which activate defense responses against the fungus, being recognized by the corresponding resistance genes in tomato (Rep *et al*., 2004; Houterman *et al*., 2009). Currently there are few commercial cultivars with resistance to breed 3 available for farmers.

This breed can attack cultivars with loci for resistance *i* and *i-2*, the source of resistance to breed3 was found in the wild species *Solanum pennellii* P1414773 that designates the locus that confers control to this breed of *Fol* (*i-3*) (Catanzariti *et al*., 2015). Knowledge of the pathogen and its physiological breeds is an important aspect in the management of the disease to understand the behavior of cultivated varieties, in addition, it allows the producer to choose the most suitable varieties (Grattidge and Brien, 1982).

The area under the disease progress curve (ABCPE) is an important tool for measuring crop damage due to pathogen attack (Ferrandino and Elmer, 1992) and in epidemiological studies of diseases, especially those related to quantitative resistance studies (Jeger and Viljanen-Rollinson, 2001). The conventional ABCPE estimator is the equation developed by Shaner and Finney (1977), which considers information from multiple severity assessments and produces a single value.

The objective of this study was to determine variability in virulence of *Fusarium* strains isolated from tomato plants with typical symptoms of vascular withering in tomato-producing plots in Hidalgo state and to assess the incidence and severity of *Fusarium oxysporum* f. sp. *lycopercisi* breed 3 in seven tomato genotypes (*Solanum lycopersicum* L.).

Materials and methods

Sampling and identification of the pathogen

Samples of stems from tomato plants were collected with symptoms typical of vascular withering disease caused by *Fol* in tomato-producing plots in six locations in the municipality of Metepec, Hidalgo, in commercial varieties: Reserva, Donnatello, El Cid, Palermo, Andino and Mezcal. The collected stems were deposited in polyethylene bags and subsequently transferred to the Agricultural Pathosystems Laboratory of the Department of Plant breeding at the Antonio Narro Autonomous Agrarian University (UAAAN).

Longitudinal cuts were made to the stem for the purpose of observing symptoms of internal brown necrosis in the conductive vessels and then cutting sections of the stem of approximately 3 mm. These stems were disinfected with 1% sodium hypochlorite solution for 3 min, then sown in a culture medium potato dextrose agar (PDA) in Petri plates and incubated at 25 °C for eight days, after which hypha tip transfer was performed to obtain monospiric crops (Amini, 2009). The identification of the pathogen was made considering the symptomatology in diseased plants and by the morphological characteristics of mycelium and conidia in the growing medium at the microscopic level (Gerlanch and Nirenberg, 1982; Leslie and Summerell, 2006).

Fol breed identification

10-day-old PDA cultures were washed with sterile distilled water to obtain inoculum suspension from the pathogen. The crops were then filtered, washed with sterile water and adjusted to a concentration of 1×10^6 conidia per ml. The viability of conidia was verified by dilutions in plates in PDA media. For each insulation the concentration of spores was measured by counting macro conidia using a Neubauer hematocimeter under a 40X magnification microscope.

The pathogenicity of each isolate was tested on cv tomato seedlings. Bony Best, susceptible to breeds 1, 2 and 3 of *Fol*, following the methodology of immersion of root tips for 10 min in a fungal solution at a concentration of 1×10^6 spores ml^{-1} (Williams, 1981; Inami *et al*., 2012). After inoculation the seedlings were transplanted into 10 cm diameter pots, filled with commercial peat moss substrate and kept in a greenhouse.

The identification of races of the isolates collected from *Fol* was carried out using the pathogenicity test in 4 differential tomato crops: Bonny Best, without resistance genes and susceptible to the three races, Manapal resistant only to race 1 by the presence of locus *i*, but susceptible to breeds 2 and 3, Walter resistant to breeds 1 and 2 due to the presence of loci *i* and *i-2*,but susceptible to race3 and I3R3 resistant to races 1, 2 and 3 due to the presence of the *i-3* gene (Reis *et al*., 2004; Scott *et al*., 2004). Differential materials were provided by Dr. John Paul Jones of the Institute of Sciences for Agriculture and Food at the University of Florida.

When the seedlings had three true leaves they were extracted from the substrate; its roots were washed with running water, rinsed with sterile distilled water and submerged for 10 min in a fungal solution at a concentration of 1×10^6 spores ml^{-1} (Inami *et al*., 2012). The control plants were submerged in distilled water. All seedlings were transplanted into 10 cm diameter pots containing a sterile mixture of sand and soil, 15 g of NPK (15:15:15) were added to each pot. The pots were kept in a greenhouse at 23-28 °C, 60-70% relative humidity and 16 h of light, 8 h of darkness.

Evaluation of tomato genotype resistance for *Fol* breed 3

The incidence and severity of *Fusarium oxysporum* f. sp. *lycopercisi* breed 3was evaluated in seven tomato genotypes (K3, R1, F3, Y53, D4, D3, IR13), which are lines selected for their potential yield in previous work carried out in the Department of Phytomejoration of the Antonio Narro Autonomous Agrarian University (UAAAN). For inoculation, tomato seedlings were used 30 days after germination, using the root immersion technique in a suspension of $1x10^6$ conidia per ml. After inoculation, seedlings were transplanted into 3-litre polyethylene bags containing a mixture of soil and peat and they kept in a greenhouse for 48 days at a temperature of 25 ±2 °C.

The response of tomato plants to *Fol* inoculation was made using a severity scale of 1 to 5 according to Marlatt *et al*. (1996) modified to estimate the severity of the disease, where 1 corresponds to a symptom-free plant, 2 plant with mild chlorosis in the lower leaves, 3 moderate chlorosis, 4 severe chlorosis and 5 dead plant. With the severity scale values assigned to plants, a percentage disease index was estimated for each of the genotypes using the following formula: $IE=\left[\left(\frac{\sum_{i=1}^{n} X_i}{n}\right)0.2\right]100$. Where: Xi= severity of the disease in the i-th seedling, n= number of seedlings evaluated. 0.2= correction factor for disease percentages.

The disease rates obtained were used to determine the progression of the disease and the response of these materials to the inoculation of the pathogen by calculating the area under the disease progress curve (ABCPE) according to the following equation: $ABCPE=\sum_{i=1}^{n-1}\left\{\left(\frac{y_{i+1}+y_i}{2}\right)(t_{i+1}-t_i)\right\}$ Where: 't' is the time of each reading 'y' is the percentage of plants affected at each reading and 'n' is the number of readings (Shaner and Finney, 1977).

Statistical analysis

All pathogenicity tests were performed on a randomized complete block design, with three repetitions. The experimental unit consisted of 5 pots per genotype. With the values obtained for disease index and ABCPE a variance analysis was performed using the GLM procedure and comparison of means using the Tukey test ($p \leq 0.05$). For disease index, the variance test was performed with values obtained at 16, 32 and 48 days after inoculation.

Results and discussion

Isolation and identification of the pathogen

Of thirty isolations obtained from plants with symptoms typical of the disease, six produced colonies in the PDA culture medium with aerial, dispersed and abundant mycelium with variation in white to pale violet, with abundant macroconidia of short and medium length, curved, thin-walled and septate. These characteristics coincide with that described by Leslie and Summerell (2006) for the morphological identification of *Fusarium oxysporum*, in addition to the symptomatology observed in artificially inoculated tomato plants allowed identification as *Fusarium oxysporum* f. sp. *lycopersici*.

Fol breed identification

Results on the reaction of differential cultivars for *Fol* were obtained 16 days after inoculation. Susceptible seedlings had typical symptoms of the disease (yellowing, defoliation, withering and death), while resistant seedlings showed no symptomatology to the strains used. Bonny Best and Manapal had complete susceptibility in the six strains evaluated (Table 1), thus ruling out the presence of race 1. Walter showed complete susceptibility to four of the six insulations and resistance in two, indicating that these two isolations can be identified as race 2, since the Walter variety is resistant to breeds 1 and 2 and susceptible to race 3.

Table 1. Breed identification of *Fusarium oxysporum* f. sp. *lycopersici* on tomato-producing plots in Metepec, Hidalgo.

Property	Strain	Differential varieties				Race
		Bonny Best	Manapal	Walter	I3R3	
Metepec	1	S	S	S	R	3
The acocul	2	S	S	S	R	3
Turtles	3	S	S	R	R	2
I. Zaragoza	4	S	S	S	R	3
Palo gordo	5	S	S	R	R	2
The acocul	6	S	S	S	R	3

S= susceptible; R= resistant.

Some plants of the I3R3 variety considered resistant to race 3 showed some signs of susceptibility probably due to a genetic impurity in the crop, due to the loss of minor genes during the introgression process by backcrossing, which are able to modulate the expression of the reaction of resistance to this pathogen (Reis *et al*., 2004).

The presence of two breeds was detected in plots sampled in a ratio of 33% corresponding to race 2 and 67% to race 3. Therefore, it is necessary to use crops adapted to the region with resistance to strains 2 and 3. Ortega (2010); Sánchez *et al*. (2010); Hernández *et al*. (2014) also reported the presence of *Fol* strains 2 and 3 in the state of Morelos, Sinaloa and San Luis Potosí, respectively. Valenzuela-Ureta *et al*. (1996); Carrillo-Facio *et al*. (2003); Ascencio-Álvarez *et al*. (2008) report the presence of breeds 1, 2 and 3 in commercial lots of tomato in Culiacán Sinaloa; Holguín-Peña (2005) in Baja California Sur and Ortega (2010) in Morelos, it is likely that the origin of these breeds in Mexico was by introduction of contaminated seed.

With the recent increase in the use of commercial tomato seeds produced outside Mexico, there is greater potential to introduce and disseminate the pathogen in areas where it has not been previously reported. The appearance of new breeds may also be due to the selection and mutation of pre-established or isolated avirulent races (Shahi *et al*., 2016). *Fusarium oxysporum* lacks sexual reproduction, therefore, genetic exchange is limited to the parasexual cycle and genetic transformation, which requires heterokaryosis, leading to hyphal fusion followed by cell lysis (Strom and Bushley, 2016).

The plants have developed a complex defense system against various pathogens (Agrios, 2004). Once pathogens overcome mechanical barriers to infection, plant receptors initiate signaling pathways that drive the expression of defense response genes that depend on their ability to recognize harmful molecules, perform signal transduction, and respond defensively through ways involving many genes and their products (Collinge *et al*., 1994). Similarly, pathogens actively try to evade and interfere with response ways, selecting a decentralized and multi-component immune system (Andersen *et al*., 2018).

Disease index and ABCPE in tomato genotypes inoculated with *Fol*

One way to measure *Fol* damage in tomato plantations is by quantifying the incidence, which measures the number of affected plants expressed in percentage, similarly used to determine resistance to this pathogen. The area under the disease progress curve is a quantitative summary of disease intensity over time, useful for comparing genotype response to pathogen inoculation. The results obtained in this study show significant differences ($p \geq 0.05$) for disease index and ABCPE in the genotypes evaluated (Table 2), the above highlights the difference in the genetic basis of the lines evaluated and the resistance or susceptibility to *Fol*.

The first symptoms of the disease (chlorosis) occurred 16 days after inoculation, with increased severity over time. Y53 was the genotype with the lowest percentage of incidence of the disease (60.6%) and ABCPE of 1 100, while IR13 and R1 had the highest incidence values for the disease

(76 and 75.3%, respectively), with ABCPE values of 1 700 and 1 450, respectively (Table 3). The lowest values of ABCPE correspond to materials with the lowest incidence of disease, that is. with a higher level of resistance (Bautista *et al*., 2009).

Table 2. Average squares of variance analysis for disease index and area under disease progress curve (ABCPE) in seven tomato genotypes in response to artificial inoculation of *Fusarium oxysporum* f. sp. *lycopersici.*

FV	GL	IE			ABCPE
		16 DDI	32 DDI	48 DDI	
Repetitions	2	0.619	20.33	0.57	17500
Genotypes	6	39.82**	119.04**	103.2**	163015.87**
Error	20	0.396	7.83	1.18	3194.44
Average value		15.9	49.04	68.14	1357.14
CV		3.9	5.706	2.59	4.16

FV= source of variation; IE= disease index; ABCPE= area under the disease development curve; DDI= days after inoculation; **= significance level at 1%.

Table 3. Percentage of disease development over time and area under disease progress curve (ABCPE) in tomato plants after artificial inoculation by immersion of roots of *Fusarium oxysporum* f. sp. *lycopersici.*

Genotypes	Disease index			ABCDE
	16 DDI	32 DDI	48 DDI	
K3	21.0 a	48.33 bc	65.00 c	1183 de
R1	15.3 c	51.66 b	75.33 a	1450 bc
F3	14.3 c	61.66 a	70.33 b	1600 ba
Y53	11.6 d	46.66 bc	60.66 a	1100 e
D4	20.3 a.	46.66 bc	65.33 c	1316 dc
D3	12.3 d	41.66 c	64.33 c	1150 e
IR13	16.6 b	46.66 bc	76.00 a	1700 a

DDI= days after inoculation. Values followed by the same letter in each column do not differ statistically, Tukey Test ($p \geq 0.001$).

In this regard, plants and pathogens have carried out complex attack and defense mechanisms. The plant defense system is the ability to perceive pathogens and activate effective defense responses (Grube *et al*., 2000). Resistance in plants involves resistance proteins (R) that detect specific effecting proteins (*Avr*) produced by the pathogen. So far, some of the disease resistance genes to *Fol* have been used successfully in plant breeding. The use of tomato varieties with genetic resistance has represented a good sustainable production alternative replacing frequent pesticide applications. In general, most commercial varieties of tomato contain one or two of the *i-1* and *i-2* genes against *Fol* disease (El Mohtar *et al*., 2007).

Plant disease management has always been one of the main goals in genetic improvement programs. However, host-pathogen interactions involving resistance are complex, plants develop resistance mechanisms, pathogens develop strategies to overcome plant resistance; plants, at the same time, develop new defensive measures that select additional changes in the pathogen (Stahl and Bishop, 2000; Gururani *et al*., 2012). Plant pathogens have strategies to recognize the right host, penetrate and invade plant tissue, overcome plant defenses, and optimize their growth within plant.

To carry out these processes, generally, the fungus has to perceive the chemical and physical signals of the host and respond with the metabolic and morphogenetic changes required for pathogenic development (Pietro *et al*., 2001). Figure 1 shows the progress of the disease during plant development over time, it is seen that D3 and Y53 had the lowest incidence values of the disease in the first few days of evaluation, delaying the onset of the disease probably as a mechanism of defense of plants to attack the pathogen. This does not prevent the plants from being infected, but rather reduces the rate of disease increase in each of the infection points, thus delaying the spread of the disease and the development of plant disease epidemics in the field (Van der Plank, 1984).

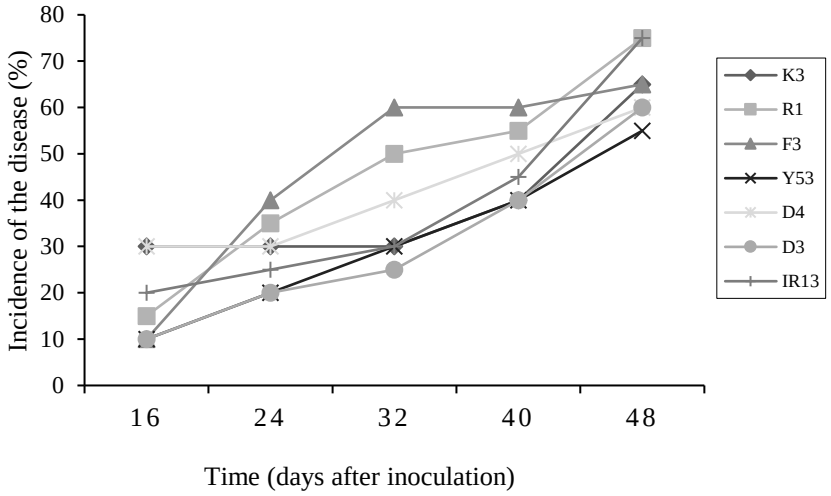

Figure 1. Response to *Fusarium oxisporum* f. sp. *lycopersici* inoculation in seven tomato genotypes.

Pathogens actively try to evade and interfere with response ways, selecting a decentralized, multi-component immune system (Andersen *et al*., 2018), which is finally expressed in a generalized infection.

In this study, disease incidence values of more than 50% were observed, in contrast to other studies, the incidence and severity was variable, because the environmental conditions, variety and virulence of the pathogen are different. In order to evaluate the resistance of tomato genotypes to *Fol* breed 3, Báez-Valdez *et al*. (2010), carry out root immersion inoculations on four tomato rootstocks, finding severity values below 10%. Mitov and Pérez (1973) when determined resistance

to *F. oxysporum* in tomato varieties obtained values of 15% incidence in the evaluations carried out. For his part, Mitidieri *et al*. (2005) determined resistance to *F. oxysporum* in tomato rootstocks, reporting severity percentages of 13% after 30 days of evaluation.

The use of tomato varieties resistant to the breeds of *Fol* is an alternative for its control, therefore, it is essential to have germplasm with resistance to this disease. In this work, the genotypes evaluated showed differences in resistance to this pathogen, showing its broad genetic base. In results obtained by Ascencio-Álvarez *et al*. (2008), based on the incidence of vascular wilt presented in 27 tomato accessions, it was found that four of them presented resistance to *Fusarium oxysporum* f. sp. *lycopersici*, being three of wild origin, two of *S. pimpinellifolium*, one of *S. peruvianum* and one of *S. lycopersicum* var. *Motelle*.

On the other hand, in a study conducted by Dordevic *et al*. (2012) about the reaction of different tomato crops to *Fusarium oxysporum* f. sp. *lycopersici* breed 1, it was found that of 24 tomato genotypes (between these pure and hybrid lines) 15 were not affected by this breed, four were tolerant and five susceptible.

Conclusions

Fusarium oxysporum f. sp *lycopersici*. breeds 2 and 3 was identified, as the main agent cause of vascular wilt in tomato-producing plots in Metepec, Hidalgo, demonstrating that the use of varieties with resistance to these breeds is highly recommended. Important information is provided on the behavior of seven tomato genotypes, of which Y53 and D3 had the lowest incidence of *Fol*, highlighting the importance of being considered in future work on genetic resistance to *Fusarium oxysporum* f. sp. *lycopersici.*

Cited literature

Agrios, G. N. 2004. Plant pathology. Fourth Ed. Academic Press. New York, USA. 635 p.

Amini, J. 2009. Physiological race of *Fusarium oxysporum* f. sp. *lycopersici* in Kurdistan province of Iran and reaction of some tomato cultivars to race 1 of pathogen. Plant Pathol. J. 8(2):68-73.

Andersen, E. J.; Ali, S.; Byamukama, E.; Yen, Y. and Nepal, M. P. 2018. Disease resistance mechanisms in plants. Genes. 9(7):339. Doi: 10.3390/genes9070339.

Ascencio-Álvarez, A.; López-Benítez, A.; Borrego-Escalante, F.; Rodríguez-Herrera, S. A; Flores-Olivas, A; Jiménez-Díaz, F. y Gámez-Vázquez, A. 2008. Marchitez vascular del tomate: I. Presencia de razas de *Fusarium oxysporum* f. sp. *lycopersici* (Sacc.) Snyder y Hansen en Culiacán, Sinaloa, México. Rev. Mex. Fitopatol. 26(2):114-120.

Báez-Valdez, E. P.; Carrillo-Fasio, J. A.; Báez-Sañudo, M. A.; García-Estrada, S.; Valdez-Torres, J. B. y Contreras-Martínez, R. 2010. Uso de portainjertos resistentes para el control de la fusariosis (*Fusarium oxysporum* f. sp. *lycopersici* Snyder & Hansen raza 3) del Tomate (*Lycopersicon esculentum* Mill) en condiciones de malla sombra. Rev. Mex. Fitopatol. 28(2):11-122.

Bautista, M. N.; Soto, R. L. y Pérez, P. R. 2009. Tópicos selectos de estadística aplicados a la fitosanidad. Colegio de Postgraduados en Ciencias Agrícolas. Montecillo, Texcoco Estado de México.

Cai, G.; Gale, L. R.; Schneider, R.; Kistler, H. C.; Davis, R. M.; Elias, K. S. and Miyao, E. M. 2003. Origin of race 3 of *Fusarium oxysporum* f. sp. *lycopersici* at a single site in California. Phytopathology. 93(8):1014-1022. Doi: 10.1094/phyto.2003.93.8.1014.

Carrillo-Facio, J. A.; Montoya-Rodríguez, T. J.; García-Estrada, R. S.; Cruz-Ortega, J. E.; Márquez-Zequera, I. y Sañudo-Barajas, A. J. 2003. Razas de *Fusarium oxysporum* f. sp. *lycopersici* Snyder y Hansen, en tomate (*Lycopersicon esculentum* Mill.) en el Valle de Culiacán, Sinaloa, México. Rev. Mex. Fitopatol. 21(2):123-127.

Catanzariti, A. M.; Lim, G. T. and Jones, D. A. 2015. The tomato I-3 gene: a novel gene for resistance to *Fusarium* wilt disease. New Phytol. 207(1):106-108. Doi:10.1111/nph.13348.

Collinge, D. B.; Gregersen, P. and Thordal-Christensen, H. 1994. The induction of gene expression in response to pathogenic microbes. *In*: mechanisms of plant growth and improved productivity, Modern approaches. Basra, AS. (Ed). New York, Marcel Dekker. 391-433 p.

do Amaral, D. O. J.; de Andrade-Lima, M. M.; Vilela, L. and da Silva, M. V. 2008. Differential gene expression induced by salycilic acid and *Fusarium oxysporum* f. sp. *lycopersici* infection in tomato. Pesq Agrop Bras. 43(8):1017-1023. https://doi.org/10.1590/S0100-204X2008000800010.

Dordevic, M.; Vatchev, T.; Girek, Z.; Šević, M.; Zečević, B.; Zdravković, J. and Ivanović, M. 2012. Reaction of different tomato cultivars toward race 1 of *Fusarium oxysporum* f. sp. *lycopersici*. Genetika. 44(1):109-118. Doi: 10.2298/gensr1201109d.

El Mohtar, C. A.; Atamian, H. S; Dagher, R. B.; Abou-Jawdah, Y.; Salus, M. S. and Maxwell, D. P. 2007. Marker-assisted selection of tomato genotypes with the i-2 gene for resistance to *Fusarium oxysporum* f.sp. *lycopersici* race 2. Plant Dis. 91(6):758-762. https://doi.org/10.1094/PDIS-91-6-0758.

Ferrandino, F. J. and Elmer, W. H. 1992. Reduction in tomato yield due to Septoria leaf spot. Plant Dis. 76(2):208-211.

FAOSTAT. 2018. Food and Agriculture Organization Corporate Statistical Database. Cantidades de producción de tomates, frescos por país. http://www.fao.org/faostat/es/#data/QC.

Gerlach, W. and Nirenberg, H. 1982. The genus *Fusarium*: a pictorial atlas. Kommissionsverlang pall parey, Berlin-Dahlem. 406 p.

Grattidge, R. and Brien, R. 1982. Ocurrence of a third race of *Fusarium* wilt tomatoes in Queensland. Plant Dis. 66(2):165-166.

Grube, R. C.; Radwanski, E. R. and Jahn, M. 2000. Comparative genetics of disease resistance within the solanaceae. Genetics. 155(2):873-887.

Gururani, M. A.; Venkatesh, J.; Upadhyaya, C. P.; Nookaraju, A.; Pandey, S. K. and Park, S. W. 2012. Plant disease resistance genes: Current status and future directions. Physiol. Mol. Plant Pathol. 78(1):51-65. Doi:10.1016/j.pmpp.2012.01.002.

Hernández-Martínez, R.; López-Benítez, A.; Borrego-Escalante, F.; Espinoza-Velázquez, J.; Sánchez-Aspeytia, D.; Maldonado-Mendoza, I. E. y López-Ochoa, L. A. 2014. Razas de *Fusarium oxysporum* f. sp. *lycopersici* en predios tomateros en San Luis Potosí. Rev. Mex. Cienc. Agríc. 5(7):1169-117.

Holguín-Peña, R. J. 2005. Fusarium wilt of tomato caused by *Fusarium oxysporum* f. sp. *lycopersici* race 3 in Baja California Sur, México. Plant Dis. 89(2):1360-1361. Doi: 10.1094/pd-89-1360c.

Houterman, P. M.; Ma, L.; Van Ooijen, G.; De Vroomen, M. J.; Cornelissen, B. J. C.; Takken, F. L. W. and Rep, M. 2009. The effector protein Avr2 of the xylem-colonizing fungus *Fusarium oxysporum* activates the tomato resistance protein I-2 intracellularly. Plant J. 58(6):970-978. https://doi.org/10.1111/j.1365-313X.2009.03838.x.

Inami, K.; Yoshioka-Akiyama, C.; Morita, Y.; Yamasaki, M.; Teraoka, T. y Tsutomu, A. 2012. A genetic mechanism for emergence of races in *Fusarium oxysporum* f. sp. *lycopersici*: inactivation of avirulence gene *Avr1* by transposon insertion. PLoS One. 7(8):44101. https://doi.org/10.1371/journal.pone.0044101.

Jeger, M. and Viljanen-Rollinson, S. 2001. The use of the area under the disease-progress curve (AUDPC) to assess quantitative disease resistance in crop cultivars. Theor. Appl. Genetics. 102(1):32-40.

Komada, H.; Ogawa, K. and Aoki, T. 2011. *Fusarium*. Tokyo: Zenkoku Nouson Kyoiku Kyokai. (en Japones). Malafaia, B. C; Silva, D. T; Jordão, O. D; Alves, C. M; Silva, R. B. M; Santos, C. M. and Vanusa, S. M. 2013. Evaluation of the resistance and differential induction of chitinases in tomato in response to inoculation with *Fusarium oxysporum* f. sp. *lycopersici*. J. Plant Physiol. Pathol. 1(3):1-6.

Leslie, J. F. and Summerell, B. A. 2006. The Fusarium laboratory manual. Oxford: Blackwell Science. 212 p.

Malafaia, C. B.; Silva, T. D.; do Amaral, D. O. J.; de Almeida1, C. M. A.; da Silva, M. L. R. B.; dos Santos, M. T. C. and Silva, M. V. 2013. Evaluation of the resistance and differential induction of chitinases in tomato in response to inoculation with *Fusarium oxysporum* f. sp. *lycopersici*. J Plant Physiol Pathol. 1(3):1-6. Doi:10.4172/2329-955x.1000109.

Marlatt, M.; Correll, J. and Kaufman, P. 1996. Two genetically distinct populations of *Fusarium oxysporum* f. sp. *lycopersici* race 3 in the United States. Plant. Dis. 80(12):1336-1342. Doi:10.1094/PD-80-1336.

Mitidieri, M. S.; Brambilla, M. V.; Piris, M.; Piris, E. y Maldonado, L. 2005. El uso de portainjertos resistentes en cultivo de tomate bajo cubierta: resultados sobre la sanidad y el rendimiento del cultivo. Instituto Nacional de Tecnología Agropecuaria. Buenos Aires, Argentina. 8 p.

Mitov, N. y Pérez, M. 1973. Resistencia de variedades de tomate al *Fusarium oxysporum* f. sp. *lycopersici* (Sacc.) Snyder y Hansen. Agricultura. 6(2):52-61.

Ortega, G. J. G. 2010. Diagnóstico de hongos fitopatógenos de jitomate y efecto de *Trichoderma asperellum* Tc74 sobre *Fusarium* spp. Tesis de Maestría en Ciencias en Manejo Agroecológico de Plagas y Enfermedades. Instituto Politécnico Nacional (IPN).

Panthee, D. R. and Chen, F. 2010. Genomics of fungal disease resistance in tomato. Curr. Genomics. 11(1): 30-39. Doi: 10.2174/138920210790217927.

Pietro, A.; García-Maceira, F. I.; Meglecz, E. and Roncero, M. I. G. 2002. A MAP kinase of the vascular wilt fungus Fusarium oxysporum is essential for root penetration and pathogenesis. Mol Microbiol. 39 (5):1140-1152. Doi: 10.1046/j.1365-2958.2001.02307.x.

Reis, A.; Giordano, L. B.; López, C. A. and Boiteux, L. S. 2004. Novel sources of multiple resistance to three races of *Fusarium oxysporum* f. sp. *lycopersici* in *Lycopersicon* germplasm. Crop Breed. Appl. Biotechnol. 4(4):495-502. Doi:10.12702/1984-7033.v04n04a19.

Rep, M.; van der Does, H. C.; Meijer, M.; van Wijk, R.; Houterman, P. M.; Dekker, H. L.; de Koster, C. G. and Cornelissen, B. J. 2004. A small, cysteine-rich protein secreted by *Fusarium oxysporum* during colonization of xylem vessels is required for I-3-mediated resistance in tomato. Mol Microbiol. 53(5):1373-1383. Doi: 10.1111/j.1365-2958.2004.04177.x.

SADER-SIAP 2018. Cierre de la producción agrícola por cultivo. Secretaría de Agricultura, Ganadería y Desarrollo Rural-Servicio de Información Agroalimentaria y Pesquera. http://www.siap.gob.mx/cierre-de-la-produccion-agricola-por-cultivo/.

Sánchez-Peña, P.; Cauich-Pech, S. O.; Núñez-Farfán, J.; Núñez-Cebreros, R. D.; Hernández-Verdugo, S.; Parra-Terraza, S. and Villarreal-Romero, M. 2010. Incidence of *Fusarium oxysporum* f. sp. *lycopersici* races in tomato in Sinaloa, Mexico. Plant Dis. 94(11):1376-1386. https://doi.org/10.1094/PDIS-04-10-0255.

Scott, J. W.; Agrama, H. A. and Jones, J. P. 2004. RFLP based analysis of recombination among resistance genes to Fusarium wilt races 1, 2, and 3 in tomato. J. Am. Soc. Hortic. Sci. 129(3):394-400. Doi: 10.21273/JASHS.129.3.0394.

Shahi, S.; Beerens, B.; Bosch, M.; Linmans, J. and Rep, M. 2016. Nuclear dynamics and genetic rearrangement in heterokaryotic colonies of *Fusarium oxysporum.* Fungal Gen. Biol. 91(1):20-31. https://doi.org/10.1016/j.fgb.2016.03.003.

Shaner, G. and Finney, R. 1977. The effect of nitrogen fertilization on the expression of slow-mildewing resistance in Knox. Phytopathology. 67(8):1051-1056. https://doi.org/10.21273/JASHS.129.3.0394.

Srinivas, C.; Nirmala, D. D.; Narasimha, M. K.; Chakrabhavi, D. M.; Lakshmeesha, T. R.; Bhim, P. S.; Naveen, K. K.; Niranjana, S. R.; Amber, H.; Abdulaziz, A. A.; Baby, T.; Elsayed, F. A. A.; Chandra, N. S. and Rakesh, K. S. 2019. *Fusarium oxysporum* f. sp. *lycopersici* causal agent of vascular wilt disease of tomato: Biology to diversity. A review. Saudi J. Biol. Sci. 26(7):1315-1324. https://doi.org/10.1016/j.sjbs.2019.06.002.

Strom, N. B. and Bushley, K. E. 2016. Two genomes are better than one: history, genetics, and 483 biotechnological applications of fungal heterokaryons. Fungal Biol. Biotechnol. 3(4):1-14. Doi 10.1186/s40694-016-0022-x.

Stahl, E. A. and Bishop, J. G. 2000. Plant-pathogen arms races at the molecular level. Current Opinion in Plant Biology. 3(4):299-304. https://doi.org/10.1016/S1369-5266(00)00083-2.

Valenzuela-Ureta, J. G; Lawn, D. A; Heisey, R. F. y Zamudio-Guzmán, Z. V. 1996. First report of *Fusarium* wilt race 3, caused by *Fusarium oxysporum* f. sp. *lycopersici* of tomato in México. Plant Dis. 80(1):105. Doi: 10.1094/pd-80-0105a.

Van der Plank, J. E. 1984. Disease resistance in plants. Second Edition. Academic Press, Inc. Orlando, San Diego, San Francisco, N. Y. London. 194 p.

Williams, P. H. 1981. Fusarium Yellows. Screening crucifers for multiple disease resistance. Williams, P. H. (Ed.). University of Wisconsin, Madison. 124-129 pp.