



Plant Pathology Journal

ISSN 1812-5387

science
alert

ANSI*net*
an open access publisher
<http://ansinet.com>

Physiological Race of *Fusarium oxysporum* F. sp. *Lycopersici* in Kurdistan Province of Iran and Reaction of Some Tomato Cultivars to Race 1 of Pathogen

J. Amini

Department of Plant Protection, University of Kurdistan, P.O. Box 416, Sanandaj, Iran

Abstract: In this research, eleven isolates of *Fusarium oxysporum* were collected from tomato plants displaying wilt symptoms in fields in Kurdistan province. Race 1 of pathogen was obtained from Moscow Timiryazev Agricultural Academy in Russia. Pathogenicity of the collected isolates and race 1 of the pathogen were evaluated in glasshouse conditions. Pathogenicity tests and race determination were conducted using root-dip inoculation with different tomato cultivars, Belyi naliv-241 (not resistant), Blagovest (resistant to race 1) and Benito (resistant to both races of 1 and 2). The experimental design was a completely randomized type with six replications (pots) containing two seedlings per pot. Disease severity was measured five weeks following inoculation by using a scale of 0 to 4. The criteria used to assess the response of different cultivars were; leaf disease index, plant height and vascular discoloration index. Results showed that Belyi naliv-241 lacking any resistance gene wilted four weeks after inoculation, but cultivars Blagovest and Benito did not develop symptoms to any of the isolates tested. The reaction of race 1 and the Iranian isolates were similar in pathogenicity suggesting that all of the isolates belong to *Fusarium* f. sp. *lycopersici* race 1. Also, Reaction 23 tomato cultivars against to *F. o. f. sp. lycopersici* indicated that 6 of the them were resistant, 5 were intermediately resistant, 6 were tolerant, 3 were susceptible and the rest 3 were found to be very susceptible.

Key words: Tomato, Fusarium wilt, race, cultivar resistance

INTRODUCTION

Iran is located in the Southern part of the Northern moderate zone (latitude 25°03'-39°47'N, longitude 44°14'-63°20'E), with a mean altitude of more than 1,200 m above sea level. However, Iran has a diverse climate, with three main climatic regions: (1) dry and semidry: covering a large part of the interior and southern border of Iran, where annual rainfall is 25-30 mm; (2) mountainous, which is subdivided into cold and moderate mountainous climates: cold mountainous climate, which covers 40,000 km² and has more than 500 mm annual rainfall and moderate mountainous climate covering 300,000 km² with 250-600 mm annual rainfall and (3) Caspianic climate: a small narrow area between the Caspian Sea and Alborz Mountain Belt with 600-2,000 mm annual rainfall. Tomato cultivation starts in March and harvested in July or August.

Fusarium wilt of tomato (*Lycopersicon esculentum* Mill.), caused by *Fusarium oxysporum* f. sp. *lycopersici* (Sacc.) W.C. Snyder and H.N. Hansen, is an economically important disease and it is a destructive disease of tomato crop worldwide (Jones *et al.*, 1991). Three different host-specific races of pathogen (race 1, 2 and 3) have been identified (Cai *et al.*, 2003). Race 1 was initially observed in 1886 (Booth, 1971) and race 2 was first reported in 1945

in Ohio (Alexander and Tucker, 1945). Race three of *F.o. f. sp. lycopersici* was identified in Australia in 1978 (Grattidge and O'Brien, 1982) and was subsequently reported in several U.S. States and Mexico (Davis *et al.*, 1988). All commercially cultivated tomatoes (*L. esculentum*) lacking I-genes, are susceptible to *F. o. f. sp. lycopersici*. Resistance to race 1 found in line 160 *Lycopersicon pimpinellifolium* PI79532 that has I-gene (Grattidge and O'Brien, 1982). The gene I remained effective to race 1 until the early 1970s (Jones and Crill, 1974). Race 2 of the pathogen was reported in Korea and in Ohio (Valenzuela, 1996), this race of the pathogen overcome the resistance of cultivars which showed resistance to race 1. Then I2 gene was found in tomato cv. *Lycopersicon peruvianum* resistant to both races 1 and 2 (Scott and Jones, 1989). Both gene of I and I2 were mapped on chromosome 11 (Laterrot, 1976; McGrath *et al.*, 1987). The race 3 was observed in Australia and Florida (Grattage and O'Brien, 1982). Since, the attention have shifted to screening for resistance to race 3, which had overcome the resistance to race 1 and 2, respectively. Resistance to race 3 has been observed in genotype of *Lycopersicon pennellii* that consists of I3 gene (McGrath and Toleman, 1983). Gene I3 was mapped on chromosome 7 (Bournival *et al.*, 1989; Sarfatii *et al.*, 1991). Races of *F. o. f. sp. lycopersici* agents could be

distinguished by their differential virulence on tomato cultivars containing different dominant resistance genes (McGrath *et al.*, 1987; Mes *et al.*, 1999). Based on the existence of monogenic dominant resistance traits, a gene-for-gene relationship between *F. o. f. sp. lycopersici* and tomato is generally assumed. Fungal races contain dominant virulence genes that correspond to dominant resistance genes in the cultivars are unable to infect (Flor, 1971). Therefore, the use of resistant varieties is the best strategy for disease control (Akköprü and Demir, 2005; Beckman, 1987).

The aim of this research is to identify the races of *F. o. f. sp. lycopersici* in fields of Kurdistan province and determine the reactions of some tomato cultivars to Fusarium wilt.

MATERIALS AND METHODS

Fungal isolates and Identification of Fusarium species:

Fusarium isolates were collected in 2007 from 10 tomato fields in Kurdistan province of Iran. Sections (3-5 cm long) of tomato plant stem showing vascular discoloration were rinsed thoroughly in tap water. After surface-disinfecting in sodium hypochlorite (5%) for 2 min, the plant pieces were rinsed three times in sterile-distilled water, dried on sterile filter paper and plated onto Potato Dextrose Agar (PDA) medium amended with streptomycin sulphate (300 mg L⁻¹). Fungal cultures were incubated for two weeks at 24°C. The fungal isolates were cleaned up by subculturing successively and were selected by single-spore isolation method on dried agar cultures. For identification of Fusarium species, isolates were cultured on Special Nutrient Agar (SNA) media (Nash and Snyder, 1962) and identified by source of Gerlach and Nirenberg (1982). Identification of pathogen was made under the light microscope and fungal structures were placed on slides, stained with methylene blue. Also, identification of fungi was based on colony morphology, conidial characteristics and phialid type.

Inoculum preparation: The conidia of 10-day-old cultures from (PDA) were washed with sterile distilled water to obtain suspension of inoculums of the pathogen. Cultures were then filtered through one layer of Mira cloth, centrifuged (6000 x g for 15 min), washed with sterile water and adjusted to a concentration of 10⁶ conidia per mL. Viability of conidia was checked by plating dilutions on PDA media. The spore concentration was measured by using a haemocytometer.

Plant material: Seeds of all cultivars of tomato were surface-disinfected with 1% sodium hypochlorite for 5 min

and rinsed three times in sterile-distilled water prior to sowing. Then, the seeds were sown in standardized sand and soil (80:20) and were grown in seedling plug trays (plug size 3.4 by 3.4 by 5 cm, 64 plugs). Trays were maintained in a glasshouse at 23-28°C, 60-70% relative humidity and 16 h light, 8 h darkness.

Pathogenicity tests: The Pathogenicity of each isolate was tested on tomato seedling cv. Belyi naliv-241 (universally susceptible) at the three-true-leaf stage. Their roots were dipped into a conidial suspension (10⁶ microconidia per mL⁻¹) of the test isolate for 10 min, after which seedlings were transplanted into sterilized soils in pots (10 cm in diameter) and kept in glasshouse.

Race identification: Collected isolates of *F. o. f. sp. lycopersici* were identified to race using pathogenicity tests with 3 differential tomato cultivars; Belyi naliv-241 (lacking a resistant gene), Blagovest (resistant to race 1) and Benito (resistant to race 1 and 2). Seedling (at the three leaf stage) were uprooted from the substrate; their roots were washed out in tap water, rinsed by sterile distilled water and dipped for 10 min in fungal suspension solution (10⁶ spores mL⁻¹) (Wellman *et al.*, 1939). Control plants were dipped in distilled water. All seedlings were transplanted into 10-cm-diameter pots containing a sterile mixture of sand and soil, Added to each pot 15 g NPK (15:15:150). Twelve replications of each cultivar (six pots, each pot containing two seedlings) were tested for each isolate and for the control. Pots maintained in a glasshouse at 23-28°C, 60-70% relative humidity and 16 h light, 8 h darkness.

Resistance survey of twenty three tomato cultivars to race 1 of *F. o. f. sp. lycopersici*:

About 23 cultivars of tomato were evaluated against to race 1 of *F. o. f. sp. lycopersici* under glasshouse conditions. These cultivars consist of: (1) FDT101; (2) FDT 101; (3) MB1-56; (4) Viva 110; (5) Rapsodia (F); (6) Blagovest F1; (7) DF1-7; (8) Early urbana VF; (9) NS 4130; (10) Early urbana F1; (11) Petoearly CH; (12) GEM PACK; (13) Peto1509; (14) Rocky F1; (15) Abpopa (F2); (16) CFo4; (17) Kaissa-1 (F); (18) Dozct (10); (19) CHEE; (20) Dozct (14); (21) Dozctl; (22) Peto prides; (23) Belyi nalive-241.

Disease severity index: Disease Severity Rating (DSR) was evaluated to assess 35 days after inoculation by using the following scale (Bora *et al.*, 2004; Banerjee, 1990): 0 = no symptoms (resistant); 1 = <25% of leaves with symptoms (intermediately resistant); 2 = 26-50% of leaves with symptoms (tolerance); 3 = 51-75% of leaves with symptoms (susceptible); 4 = 76-100% of leaves with symptoms (very susceptible).

Inoculated plants were evaluated as diseased when browning of at least one vasculo bundle was visible. In addition, the plant height of inoculated plants was measured.

Statistical analysis: All Pathogenicity tests were conducted in completely randomized design. Mean were compared by Least Significant Difference (LSD) testing. Duncan's multiple Range test was applied to compare means. All statistical analyses were performed using Statistical Package for the Social Sciences, version 11 (SPSS).

RESULTS

Fungal isolates: Eleven isolates of the pathogen were collected from tomato fields in Kurdistan province in year 2007 (Table 1).

Pathogenicity tests: The pathogenicity test of all isolates was confirmed on tomato cv. Belyi naliv-241 (lacking a resistant gene). Symptoms in plants infected appeared two weeks after inoculation. Seven isolates caused typical symptoms of Fusarium wilt and aggressiveness of the isolates was variable but generally high. They showed strong virulence with a DSR>3 on cv. Belyi naliv-241 (Table 2), but four isolates of pathogen (F1, F4, F6 and F14) were unable to produce disease in tomato plant and no symptoms were observed in seedlings infected with these isolates. The first indication of disease was yellowing and drooping of the lower leaves. This symptom often occurs on one side of the plant or on one shoot. At least, disease plants exhibited stunting, dark brown vascular discoloration and death. The measurements of plant growth of susceptible genotypes were analyzed to differentiate the aggressiveness between the isolates. A significant interaction ($p = 0.05$) between Fusarium isolates and tomato cultivars was noted based on disease severity rating and plant height (Table 2). Among isolates, F5 had more disease severity rating (4.2) than the others. Also, the reduction in plant height caused by isolate F5 was statistically significant. Therefore, pathogenicity of all isolates on tomato cultivars cv. Belyi naliv-241 were clearly distinct (Table 2). The pathogen was re-isolated from the discoloured vascular tissue of diseased plants.

Identification of Fusarium species: Identification of *F. o. f. sp. lycopersici* was based on morphological criteria according to Gerlach and Nirenberg (1982). The mycelia of pathogen were white cottony to pink, often with purple

Table 1: Collected isolates of *Fusarium oxysporum f. sp. lycopersici* in Kurdistan province

Isolates	Geographic origin			
	Ghorvah	Kamyaran	Dehgolan	Sanandaj
F1			+	
F2			+	
F3		+		
F4				+
F5			+	
F6		+		
F8		+		
F12	+			
F14	+			
F15	+			
F19	+			

Isolates of F1, F4, F6 and F14 were unable to produce disease on tomato cv. Belyi naliv-241, +: Present

Table 2: Pathogenicity of isolates of *F. o. f. sp. lycopersici* on cv. Belyi naliv-241

Treatments	Disease severity index	Stem height (cm)	Vascular browning (cm)
CW	0.0 ^a	39.0 ^a	0.0 ^a
Race 1	3.3 ^{bc}	9.5 ^{bc}	8.4 ^{bc}
F2	3.4 ^{cd}	9.4 ^{bc}	9.8 ^d
F3	3.6 ^d	8.8 ^b	9.0 ^{cd}
F5	4.0 ^e	7.8 ^c	7.3 ^b
F8	3.5 ^{cd}	9.6 ^{cd}	9.0 ^{cd}
F12	3.0 ^b	11.0 ^f	11.3 ^e
F15	3.2 ^{bc}	9.5 ^{cd}	10.0 ^d
F19	3.4 ^{cd}	9.5 ^{bc}	8.8 ^{cd}
LSD at 5%	0.3	0.7	1.2

Data are means of 12 replicates; Means in the column followed by different letter(s) indicate significant differences among treatments at 0.05 according to Duncan's multiple range Test; CW: Control water

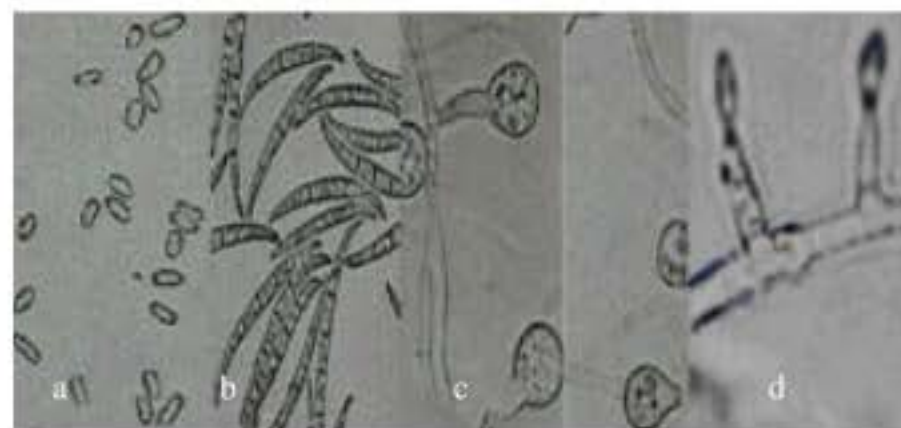


Fig. 1: Type of (a) microconidia, (b) macroconidia, (c) chlamydospore and (d) monophialid of *F. o. f. sp. lycopersici* on PDA media

tinge or reddish coloration of the medium. Microconidia were born on simple phialides arising laterally and were abundant, oval-ellipsoid, straight to curved, 4-12x2.1-3.5 μm. Macroconidia, spores to abundant, were borne on branched conidiophores or on the surface of sporodochia and were thin walled, three to five septate, fusoid-subulate and pointed at both ends, have pedicellate base. Three septate spores were more common. Chlamydospores, both smooth and rough walled, which are abundant and form terminally or on an intercalary basis (Fig. 1).

Race identification: Results of the race determination on hosts Belyi naliv-241 (not resistant), Blagovest (resistant to race 1), Benito (resistant to race 1 and 2) were evaluated and the comparison with race 1 of Moscow Timiryazev Agricultural Academy proved that all isolates belonged to race 1 under greenhouse conditions and both cv. Blagovest and Benito were found to be resistant to *F. o. f. sp. lycopersici* as no disease symptoms were observed. In contrast, tomato cv. Belyi naliv-241 was considered to be susceptible since all plants were severely affected and died (Table 3). Also, the height of tomato cv. Belyi naliv-241 infected with the pathogen were significantly lower than that of control and other cvs. Blagovest and Benito (Table 4). Pathogenicity tests for these isolates were repeated twice.

Resistance survey of twenty three tomato cultivars to race 1 of *F. o. f. sp. lycopersici*: Evaluation of the 23 genotypes of tomato to *F. o. f. sp. lycopersici* (race 1) indicated that 6 were resistant, 5 moderately resistant, 6 tolerant, 3 susceptible and 3 very susceptible (Table 5).

Table 3: Mean disease index of differential tomato cultivars after inoculation with race 1 and Kurdistan isolates of *F. o. f. sp. lycopersici*, after 35 days

T ^a	Race1	F2	F3	F5	F8	F12	F15	F19
Bn ^b	3.1 ^a	3.4 ^a	3.2 ^a	4.1 ^a	3.5 ^a	2.7 ^a	3.4 ^a	3.6 ^a
Bf ^c	0.3 ^b	0.4 ^b	0.4 ^b	0.3 ^b	0.0 ^b	0.0 ^b	0.3 ^b	0.3 ^b
B ^d	0.0 ^c	0.0 ^c	0.0 ^c	0.2 ^b	0.0 ^b	0.0 ^b	0.0 ^c	0.2 ^c
LSD at 5%	0.2	0.2	0.4	0.4	0.2	0.2	0.3	0.2

Data are means of 12 replicates; Means in the column followed by different letters indicate significant differences among treatments at 0.05 according to Duncan's multiple range Test; ^a: treatment; ^b: Belyi naliv-241; ^c: Blagovest; ^d: Benito

Table 4: Effect of isolates *Fusarium o. f. sp. lycopersici* and race 1 on tomato stem height cv. Belyi naliv-241, Blagovest and Benito, after 35 days

Treatments	Belyi naliv-241	Blagovest	Benito
Control water	41.8 ^d	35.0 ^c	31.2 ^c
Race 1	9.5 ^b	35.1 ^c	30.5 ^{abc}
F2	10.2 ^{bc}	33.7 ^a	31.2 ^{bc}
F3	9.6 ^b	35.0 ^{cd}	30.7 ^{bc}
F5	7.9 ^a	33.7 ^a	29.8 ^a
F8	9.6 ^b	34.8 ^{bc}	30.0 ^a
F12	10.0 ^{bc}	34.2 ^{ab}	30.2 ^{ab}
F15	10.8 ^c	35.3 ^{cd}	31.0 ^{ab}
F19	9.5 ^b	35.0 ^c	29.9 ^a
LSD at 5%	0.8	0.6	0.6

Data are means of 12 replicates; Means in the column followed by different letters indicate significant differences among treatments at 0.05 according to Duncan's multiple range test

Table 5: Resistance survey of 23 tomato cultivars to race 1 of *F. o. f. sp. lycopersici*

Degree of infection	Cultivars
Very susceptible	Dozctl, Peto prides, Belyi nalive-241
Susceptible	Dozct (10), CHEE, Dozct (14)
Tolerance	GEM PACK, Peto1509, Rocky F1, Abpopa (F2), CFo4, Kaissa-1 (F)
Intermediately resistant	DF1-7, Early urbana VF, NS 4130, Early urbana F1, Petoearly CH
Resistant	FDT101, FDT 101, MB1-56, Viva 110, Rapsodia (F), Blagovest F1

Disease reaction was evaluated 5 weeks after inoculation

DISCUSSION

The most susceptible plants inoculated by root-dipping developed typical symptoms of wilt, slight vein clearing on outer leaflets, stunting, dark brown vascular discoloration and death. *F. o. f. sp. lycopersici* was recovered from all symptomatic plants, whereas noninoculated tomato seedlings showed no symptoms.

The Pathogenicity tests indicated that some virulent, race 1, isolates could infect the resistant cultivar Blagovest and Benito under certain conditions (Table 3). This effect appears to be related to the inoculum concentration used in the artificial challenge tests, as there is no evidence that cv. Blagovest and Benito are affected by the inoculum dose that operates in the field.

The pathogen invades the vascular tissues and grows in the vascular bundles and inhibits water flow causing wilting, ultimately leading to death of plant (Davis, 1982; Beckman, 1987; Duniway, 1971). Vessel walls of tomato plant often are coated in an amorphous electron-opaque material (Bishop and Cooper, 1983) and this material includes xylem parenchyma, pit cavities and encrusts intertracheary pit membranes (Chamber and Corden, 1963). At least, infection is accompanied by the gradual death of xylem parenchyma cells throughout infected plants (Bishop and Cooper, 1983). Produced materials by pathogen consist of enzymes (Cooper and Wood, 1975; Jones *et al.*, 1972), growth-regulating compounds (Dimond, 1955), toxin (Scheffer and Walker, 1953; Sutherland and Pegg, 1992) and gummosis (Dimond and Waggoner, 1953).

Dark brown vascular discoloration appeared in stem of cv. Belyi naliv-241, but in cv. Blagovest and Benito dark brown vascular discoloration only was seen only in the root of plant.

This study indicated that the wild species of *Lycopersicon* were the major source of resistance. The presence of race 1 in Kurdistan province has important epidemiological implications and the potential spread of the pathogen via transplants represents a risk to tomato crops on others areas in Iran. In conclusion, this is the first report of *F. oxysporum f. sp. lycopersici* race 1 in the Kurdistan province in Iran.

ACKNOWLEDGMENT

The author is thankful to the vice chancellor for research of University of Kurdistan for the financial support (Grant No. 4/25747).

REFERENCES

- Akköprü, A. and S. Demir, 2005. Biological control of Fusarium wilt in tomato caused by *Fusarium oxysporum* f. sp. *lycopersici* by AMF *Glomus intraradices* and some rhizobacteria. *J. Phytopathol.*, 153: 544-550.
- Alexander, L.J. and C.M. Tucker, 1945. Physiological specialization in the tomato wilt fungus *Fusarium oxysporum* f. sp. *Lycopersici*. *J. Agric. Res.*, 70: 303-313.
- Banerjee, M.K., 1990. Studies on Fusarium wilt resistance in Lycopersicon: Screening and method of assessment. *Veget. Sci.*, 17: 167-174.
- Beckman, C.H., 1987. The Nature of Wilt Diseases of Plants. The American Phytopathological Society Press, USA., ISBN-13: 978-0890540749, pp: 175.
- Bishop, C.D. and R.M. Cooper, 1983. An ultrastructural study of vascular colonization in three vascular wilt diseases. I. Colonization of susceptible cultivars. *Physiol. Plant Pathol.*, 23: 323-343.
- Booth, C., 1971. The Genus *Fusarium*. 1st Edn., Commonwealth Mycological Institute, Kew, England, ISBN: 85198 046 5.
- Bora, T., H. Ozaktan, E. Gore and E. Aslan, 2004. Biological control of *Fusarium oxysporum* f. sp. *Melonis* by wettable powder formulation of the two strain of *Pseudomonas putida*. *J. Phytopathol.*, 152: 471-475.
- Bournival, B.L., J.W. Scott and C.E. Vallejos, 1989. An isozyme marker for resistance to race 3 of *Fusarium oxysporum* f. sp. *Lycopersici* in tomato. *Theor. Applied Genet.*, 78: 489-494.
- Cai, G., L.R. Gale, R.W. Schneider, H.C. Kistler, R.M. Davis, K.S. Elias and E.M. Miyao, 2003. Origin of race 3 of *Fusarium oxysporum* f. sp. *Lycopersici* at a single site in California. *Phytopathology*, 93: 1014-1022.
- Chamber, H.L. and M.E. Corden, 1963. Semeiography of Fusarium wilt of tomato. *Phytopathology*, 53: 1006-1010.
- Cooper, R.M. and R.K.S. Wood, 1975. Regulation of synthesis of cell wall degrading enzymes by *Verticillium albo-atrum* and *Fusarium oxysporum* f. sp. *Lycopersici*. *Physiol. Plant Pathol.*, 5: 135-156.
- Davies, J.M.L., 1982. *Verticillium* and Fusarium Wilt of Tomato. Ministry of Agriculture, Fishery and Food (Publications), Northumberland NE66 2PF, pp: 6.
- Davis, R.M., K.A. Kimble and J.J. Farrar, 1988. A third race of *Fusarium oxysporum* f. sp. *Lycopersici* identified in California. *Plant Dis.*, 72: 453-453.
- Dimond, A.E. and P. E. Waggoner, 1953. The water economy of Fusarium wilted tomato plants. *Phytopathology*, 43: 619-623.
- Dimond, A.E., 1955. Pathogenesis in the wilt diseases. *Annu. Rev. Plant Physiol.*, 6: 329-350.
- Duniway, J.M., 1971. Water relation of Fusarium wilt in tomato. *Physiol. Plant Pathol.*, 1: 537-546.
- Flor, H.H., 1971. Current status of the gene-for-gene concept. *Annu. Rev. Phytopathology*, 9: 275-296.
- Gerlach, W. and H. Nirenberg, 1982. The Genus *Fusarium*: A Pictorial Atlas. Kommissionsverlag Pall Parey, Berlin-Dahlem, ISBN: 3-489-20900-1, pp : 406.
- Grattidge, R. and R.G. O'Brien, 1982. Occurrence of a third race of Fusarium wilt of tomatoes in Queensland. *Plant Dis.*, 66: 165-166.
- Jones, J.B., J.P. Jones, R.E. Stall and T.A. Zitter, 1991. Compendium of Tomato Diseases. The American Phytopathological Society. 1st Edn. APS Press, New York, ISBN: 0890541205, pp: 100.
- Jones, J.P. and P. Crill, 1974. Susceptibility of resistance tomato cultivars to Fusarium wilt. *Phytopathology*, 64: 1507-1510.
- Jones, T.M., A.J. Anderson and P. Albersheim, 1972. Host-pathogen interactions. IV. Studies on the polysaccharide-degrading enzymes secreted by *Fusarium oxysporum* f. sp. *Lycopersici*. *Physiol. Plant Pathol.*, 2: 153-166.
- Laterrot, H., 1976. Mapping of I2 allele in tomato, controlling the genetic resistance to pathotype 2 of *Fusarium oxysporum* f. sp. *Lycopersici* wilt. *Ann. Amelior. Plant*, 26: 485-491.
- McGrath, D.J. and M.A. Toleman, 1983. Breeding and selection for resistance to Fusarium wilt race 3. *Proceedings of 4th Tomato Quality Workshop*, pp: 104.
- McGrath, D.J., G. Gillespie and L. Vawdrey, 1987. Inheritance of resistance to *Fusarium oxysporum* f. sp. *Lycopersici* races 2 and 3 of *Lycopersicon pennellii*. *Aust. J. Agric. Res.*, 38: 729-733.
- Mes, J.J., E.A. Weststeijn, F. Herlaar, J.J.M. Lambalk, J. Wijbrandi, M.A. Haring and B.J.C. Cornelissen, 1999. Biological and molecular characterization of *Fusarium oxysporum* f. sp. *Lycopersici* divides race 1 isolates into separate virulence groups. *Phytopathology*, 89: 156-160.
- Nash, S.M. and W.C. Snyder, 1962. Quantitative estimations by plate counts of propagules of the bean root rot Fusarium in field soils. *Phytopathology*, 52: 567-572.
- Sarfatii, M., M. Abu-Abied, J. Katan and D. Zamir, 1991. RFLP mapping of 11, a new locus in tomato conferring resistance against *Fusarium oxysporum* f. sp. *Lycopersici* race 1. *Theor. Applied Genet.*, 82: 22-26.

- Scheffer, R.P. and J.C. Walker, 1953. The Physiology of Fusarium wilt of tomato. *Phytopathology*, 43: 116-125.
- Scott, J.W. and J.P. Jones, 1989. Monogenic resistance in tomato to *Fusarium oxysporum* f. sp. lycopersici race 3. *Euphytica*, 40: 49-53.
- Sutherland, M.L. and G.F. Pegg, 1992. The basis of host recognition in *Fusarium oxysporum* f. sp. Lycopersici. *Physiol. Mol. Plant Pathol.*, 40: 423-436.
- Valenzuela, U.J.G., 1996. First report of Fusarium wilt race 3, caused by *Fusarium oxysporum* f. sp. Lycopersici, of tomato in Mexico. *Plant Dis.*, 80: 105-105.
- Wellman, F.L., 1939. A technique for studying host resistance and Pathogenicity in tomato Fusarium wilt. *Phytopathology*, 29: 945-956.