Fusarium Wilt of Watermelon: Impact of Race 2 of *Fusarium oxysporum* f. sp. *niveum* on Watermelon Production in Texas and Oklahoma

B. D. Bruton[1] and J. P. Damicone[2]

[1]United States Department of Agriculture, Agricultural Research Service, Lane, OK 74555
[2]Department of Entomology and Plant Pathology, Oklahoma State University, Stillwater, OK 74078

ABSTRACT

Fusarium wilt remains a constant threat to watermelon production in Texas and Oklahoma. Highly resistant cultivars, in conjunction with rotation, have provided good control to race 1 of *F. oxysporum* f. sp. *niveum* which is widespread in Texas and Oklahoma watermelon production areas. Race 2, for which there is no acceptable level of resistance in commercial cultivars, does not appear to be widespread within the major watermelon production areas. Additional research is needed to establish the distribution of race 2 and frequency of the three existing races (races 0, 1, and 2). A "Universal Testing System" for evaluating germplasm and monitoring virulence shifts in the pathogen population is essential. An integrated strategy for effective management of the Fusarium wilt fungus must be devised and implemented using crop rotation, cultural management, and highly resistant cultivars.

RESUMEN

La marchitez por Fusarium continúa siendo una amenaza constante para la producción de sandía en Texas y Oklahoma. El uso de cultivares altamente resistentes, conjuntamente con la rotación, ha proporcionado un buen control para la raza 1 de *Fusarium oxysporum* f. sp. *niveum* la cual se encuentra ampliamente distribuida en las áreas de producción de sandía de Texas y Oklahoma. La raza 2, para la cual no existe un nivel aceptable de resistencia en los cultivares comerciales, no parece estar extensamente distribuida dentro de las principales áreas de producción de sandía. Se necesita investigación adicional para establecer la distribución de la raza 2 y la frecuencia de las tres razas existentes (razas 0, 1, y 2). La existencia de un "sistema de análisis universal" para la evaluación del germoplasma y de los cambios en la virulencia en las poblaciones del patógeno es esencial. Una estrategia integrada para el manejo eficaz de la marchitez por Fusarium debe ser diseñada e implementada usando rotación de cultivos, manejo cultural y cultivares altamente resistentes.

Key words. Citron, race designation, resistance, seedless, vine decline, virulence

Fusarium wilt of watermelon [*Citrullus lanatus* (Thunb.) Matsum. & Nakai], caused by *Fusarium oxysporum* f. sp. *niveum* (E.F. Sm.) Snyd. & Hans., was first reported in the United States in 1894 (Smith, 1894). Since then, Fusarium wilt has been the greatest yield-limiting disease of watermelon worldwide. However, it has been one of the real success stories for the control of a devastating disease. Although Fusarium wilt remains an important disease, it is no longer the greatest constraint to watermelon production in most areas due to the high level of genetic resistance available to farmers. However, the status of Fusarium wilt is dynamic in that new races, which can attack previously resistant cultivars, can develop. If the new strains are ecologically competitive, they can become established in the field. Strains of F. *oxysporum* are separated into forma specialis and race strictly based on their ability to cause disease on certain hosts and cultivars (Snyder and Hansen, 1940). Propagules of the fungus may be spread by soil, plant debris, and farm implements. Fulton and Winston (1915) first noted that the Fusarium wilt pathogen can be carried on seeds. Several additional reports (Martyn, 1985, Martyn, 1987; Porter, 1928; Taubenhaus, 1935) have confirmed that the pathogen can be seed-transmitted. Consequently, infestation of new land may be through infested seed. Once the pathogen is established in a field, it may survive for 10 years or more in the absence of watermelon (Cirulli, 1972; Ioannou and Poullis, 1991).

Fusarium wilt symptoms include damping-off, seedling disease, or wilt during any stage of plant development. The specific symptom that may be exhibited is dependent on environmental conditions, age of plants when infected, and the density and virulence of the pathogen population. Plant symptoms following fruit-set may appear as a dull gray-green appearance of the leaves followed by yellowing of the crown foliage, wilting during the heat of the day, and eventual death.

Fusarium wilt is considered to be a vine decline disease of watermelon where vine vigor gradually deteriorates. As such, Fusarium wilt has symptoms similar to many of the other vine decline diseases of cucurbits. Bruton (1998) noted that Fusarium wilt has likely been one of the most misdiagnosed diseases of cucurbits because of its similarity with other vine decline diseases.

Fungal Virulence. Differences in virulence among isolates of *F. oxysporum* f. sp. *niveum* has been recognized for many years (McKeen, 1951). Crall (1963) reported the existence of two physiologic races in Florida. Cirulli (1972) also found two races in Italy and designated them as race 0 and race 1. Race 0 causes wilt only in cultivars with no known resistance genes. Race 2 is a highly aggressive pathogen (Netzer and Dishon, 1973) and appears to be fairly widespread in the eastern Mediterranean region including Greece and Turkey (Netzer, 1976) and Cyprus (Ioannou and Poullis, 1991). *F. oxysporum* f. sp. *niveum* race 2 has been found in watermelon production areas of Florida, Texas, and Oklahoma in the United States (Martyn and Bruton, 1989). Thus, three races have been described and designated as race 0, 1, and 2 which are presently identified using a set of differential cultivars (Table 1). Race 1 can induce slight to moderate wilt on most cultivars that are classified as resistant to Fusarium wilt, although, Armstrong and Armstrong (1978) concluded that differences between the strains were not sufficient to constitute distinct races. The distinction between race 0 and race 1 has been further questioned (Armstrong and Armstrong, 1978; Larkin et al., 1990; Martyn, 1987; McKeen, 1951; Reid, 1958; Sleeth, 1934). Furthermore, Larkin et al. (1990) demonstrated a continuum between race 1 and 2. The tests for race differentiation are laborious and often inconclusive. Results may be influenced by age of test plants, environmental factors, inoculum level, and inoculation method (Martyn and McLaughlin, 1983; Shimotsuma et al., 1972). Race identification using host differentials may be necessary, but limitations due to the virulence continuum exhibited among strains must be recognized. Actual wilt values used to distinguish specific races is rarely provided. Cirulli (1972) used both percentages and a wilt index to assign races but did not present specific criteria for placement. A wilt index (WI) was also used by Ioannou and Poullis (1991) to determine level of virulence in twelve isolates. However, they did not indicate the exact values for placement in highly virulent, moderately virulent, and less virulent categories. Furthermore, they did not assign race designations. Although differences in level of virulence were observed, Armstrong and Armstrong (1978) stated that there was insufficient evidence to demarcate seventeen watermelon isolates into races. They assigned resistance values to watermelon cultivars as follows: R = 0-46% wilt as resistant, I = 47-56% wilt as intermediate, and S = 57-100% wilt as susceptible. As noted by Martyn and Bruton (1989), a mean percentage wilt of >33% was rated susceptible, while <33% was rated resistant. Martyn and McLaughlin (1983) utilized a different system which included, highly resistant (≤20% wilt), moderately resistant (21-50% wilt), slightly resistant (51-80% wilt), and susceptible (>80% wilt) classifications. Approximately 12% of the isolates tested from watermelon growing areas of the United States did not conform to a specific race (Martyn and Bruton, 1989). Bruton (1998) recommended that isolates within the cucurbit forma specialis of *F. oxysporum* (especially watermelon races) be recognized as a continuum rather than discrete races. Languasco et al. (1998) recently reported that multivariate statistical analyses (i.e. cluster and discriminant analyses) could be used to assign cantaloupe isolates of *F. oxysporum* f. sp. *melonis* more reliably into races than the empirical criteria presently used. It is our view that virulence or race assessment of watermelon isolates may be more appropriately assigned using regression, cluster, and/or discriminant analyses. Additional analysis will be required to identify the most appropriate method for race determination and/or virulence assessment. The literature is laden with different methods for testing isolates for pathogenicity, relative virulence, race determination, and germplasm evaluation. It has been suggested that a "Universal Testing System" be developed to evaluate germplasm resistance and race differentiation (Bruton, 1998). Included in such a protocol should be a strict inoculation method (age of plants and inoculum concentration) and monitoring of environmental conditions (temperature and light intensity). Temperature and light intensity often play an important role in disease reaction. These variables cannot always be controlled, but, they can be monitored. Inoculum concentration has been shown over and over to have an effect on disease reaction of the host plant. Yet, different inoculum concentrations continue to be used by researchers. Seeds of the host genotype for differentiating races can be difficult to obtain without making additional increases. Armstrong et al. (1978) obtained the same cucumber genotype from different sources and found that those from one source were resistant while those from another source were susceptible. At present, there are a few public watermelon breeders in which one or more of the differentials may be obtained (Bruce Carle, University of Florida; Charles Johnson, Louisiana State University, Billy Rhodes, Clemson University; and Todd Wehner, North Carolina State University). The Watermelon Research and Development Working Group is attempting to establish a seed reserve for dispersal of genetically pure host differential genotypes.

Table 1. Watermelon genotypes used to differentiate races of *Fusarium oxysporum* f. sp. *niveum*[z].

	Disease reaction to:[y]		
Genotype	Race 0	Race 1	Race 2
Sugar Baby	S	S	S
Charleston Gray	R	S	S
Calhoun Gray	R	R	R
PI-296341-FR	R	R	R

[z]Developed from Cirulli (1972), Netzer (1976), and Martyn and Netzer (1991). Discrete wilt values placing isolates into a specific race classification are not standardized.

[y]R=resistant, S=susceptible.

Fungal Genetics. Vegetative compatibility grouping (VCG) has been useful in evaluating genetic diversity present in a fungal population. Members of one VCG are considered to be genetically interactive and genetically isolated from strains

in other VCGs. Kistler et al. (1998) recently provided guidelines for systematic numbering of VCGs within *F. oxysporum*. Larkin et al. (1990) demonstrated a relationship between aggressiveness and VCG. Although race 1 was placed in VCG 0080 and VCG 0081, race 2 was comprised solely in VCG 0082. Kim et al. (1992) reported six RFLP groups based on mitochondrial DNA (mtDNA) RFLP analysis, although, there was no relationship to VCG or race. Both cluster and parsimony analyses of mtDNA RFLPs indicated that all five *F. oxysporum* formae speciales in cucurbits are closely related (Kim et al., 1993). In some cases, isolates of different formae speciales were genetically more similar than isolates of the same forma specialis (Kim et al., 1993). They hypothesized that the genetic differences between the formae speciales were relatively small and that determinants for host specificity could be combined or lost in individual strains. In contrast, DNA fingerprinting of nuclear DNA clearly distinguished differences among Japanese formae speciales (Namiki et al., 1994). They suggested that the cucurbit-infecting formae speciales of *F. oxysporum* are intraspecific variants differing in nuclear DNA content and organization. Gordon and Martyn (1997) have recently reviewed the evolutionary biology of *F. oxysporum*. Concepts of isolate classification into formae specialis, VCGs, and race designations of *F. oxysporum* have also been addressed (Correll, 1991; Correll et al., 1987; Kistler, 1997; Kistler et al., 1991).

Distribution in Texas and Oklahoma. Watermelons exhibiting typical symptoms of Fusarium wilt have been observed in nearly all production areas of Texas and Oklahoma. However, sufficient surveys to quantify the prevalence of Fusarium wilt and the races involved have not been done. Fusarium wilt of watermelon was first described in 1894 from Georgia and South Carolina (Smith, 1894). The fungus is now well established in all watermelon growing areas with race 1 being the predominant race (Martyn and Bruton, 1989). Race 0 was first described in Florida (Crall, 1963) and more recently in Oklahoma (Bruton and Patterson, 1988). The first reported occurrence of race 2 of *F. oxysporum* f. sp. *niveum* in the United States was in a production field in Frio County in south-central Texas and in a seed-lot produced in Central Texas (Martyn, 1985a; Martyn, 1987). In 1988, race 2 was reported in Oklahoma (Bruton and Patterson, 1988; Bruton et al., 1988). Although all commercial cultivars are susceptible to race 2, spread throughout the watermelon areas of Texas and Oklahoma appears to be limited at present. In contrast, race 2 has become prominent in the eastern Mediterranean region of Greece and Turkey (Netzer, 1976) and Cyprus (Ioannou and Poullis, 1991). Hopkins et al. (1989) reported that cropping race 1 resistant cultivars in Florida appears to have increased the frequency of race 2 over race 1. Hadar et al. (1989) suggested the use of nitrate-nonutilizing (nit) mutants and VCG grouping to monitor the population of pathogenic strains of *F. oxysporum*. This could be an important application for ecological studies on race 2. There is no information as to the ecological fitness of race 2 in watermelon production areas of Texas and Oklahoma. Additional studies are required to determine the distribution and frequency of the watermelon wilt races in Texas and Oklahoma.

In 1998, *F. oxysporum* was isolated from numerous citron (wild watermelon plants) exhibiting stunting and xylem discoloration in central Texas (Comanche County). However, race determinations have not been made. Citron is widespread in the southern United States. Although the original African citron provided resistance genes in the development of Fusarium wilt resistant watermelon cultivars (Orton, 1913), the wild citron in the southern United States is probably hybridized with the cultivated watermelon. In Texas, over 200,000 ha of crop-land is infested with wild Cucurbitaceae (Smith et al., 1972). Smith and Cooley (1973) estimated that seed production may range from 0.2 to 1.3 million seed per hectare. Consequently, citron could play a major role in maintaining a high inoculum level in some areas and nullify the effects of crop rotation. Little is known about level of resistance possessed by wild citron in Texas and Oklahoma. Provided that wild citron possess a moderate to high level of resistance to races 0 and 1, it is conceivable that it can exert a selection pressure for the development of race 2 or additional pathogenic races of *F. oxysporum* f. sp. *niveum*.

Cultural and Chemical Control. Several approaches to control Fusarium wilt have been investigated with varying degrees of success. Smith (1899) first recommended crop rotation for the control of Fusarium wilt. Crop rotation is essential even when using cultivars classified as highly resistant because of high inoculum levels in infested fields (Elmstrom and Hopkins, 1981; Hopkins and Elmstrom, 1984; Hopkins et al., 1989). Control of Fusarium wilt is dependent upon the use of resistant cultivars and rotation of about 6 years (Elmstrom and Hopkins, 1981; Hopkins and Elmstrom, 1984). Continuous planting of susceptible cultivars can increase the inoculum density to exceed 2,000 colony forming units (CFU)/g of soil (Larkin et al., 1993). In fields previously cropped to watermelon, Netzer (1976) reported a range of 400 to 1,800 CFU/g of soil and a wilt severity in the susceptible cultivar Sugarbaby of 95-100%. Schenck (1966) noted that Fusarium-caused damping-off and the total *Fusarium* spp. population in the soil (4.6% damping-off per 1,000 colonies/gram of soil) was correlated with soil-plate counts. Transplants tend to have less wilt than do direct seeded watermelons, presumably due to stage of development at which infection occurs (D.L. Hopkins, personal communication).

Liming the soil to raise pH to 6.5-7.0 and use of a nitrate-nitrogen source have been recommended for control of Fusarium wilt (Everett and Blazquez, 1967; Jones et al., 1975). In contrast, Hopkins and Elmstrom (1976) reported that raising the soil pH to 7.0 and using nitrate nitrogen did not reduce wilt in 'old' watermelon land heavily infested with the wilt pathogen. Consequently, the effects of raising the soil pH and use of nitrate nitrogen are still not resolved in watermelon. Research is needed to identify and measure the effects of plant nutrition on Fusarium wilt of watermelon.

Soil solarization may have potential for controlling Fusarium wilt. Martyn and Hartz (1986) demonstrated a 32-fold reduction in inoculum density of *F. oxysporum* f. sp. *niveum* (race 1) in the top 10-cm of soil following 30 days of solarization, and a 50% reduction in wilt. However, wilt in

Sugarbaby and Charleston Gray was greater than 50% after 4 weeks as compared to nearly 100% wilt in non-solarized microplots. Further studies are required to evaluate the potential of soil solarization in fields with a history of Fusarium wilt.

Fumigation, using methyl bromide, has been used in Florida to control Fusarium wilt of watermelon for many years. Schenck (1966) noted that Vorlex and Vapam decreased *Fusarium* spp. populations and seedling infection, but populations were similar to non-treated soil by the end of the season. Hopkins and Elmstrom (1974) reported that benomyl (soil incorporated + weekly foliar sprays) provided adequate protection to damping-off of watermelon seedlings, especially if the cultivar has a moderate to high level of resistance.

Biological Control. The phenomenon of cross-protection or induced resistance to Fusarium wilt has been investigated as a method of control. Davis (1967) first reported cross-protection in watermelon with prior inoculation of an avirulent strain. However, tissue colonization by the non-pathogen was not responsible for the observed disease reduction. Shimotsuma et al. (1972) found that induced resistance was more effective at 20°C than at 27°C. Walker (1941) noted that temperatures around 27°C are most favorable for Fusarium wilt development in watermelon and that little infection occurs above 33°C. Huang et al. (1989) reported effective control of Fusarium wilt of watermelon using non-pathogenic isolates of *F. oxysporum*, *F. solani*, and *Trichoderma* sp. along with supplements of $CaCO_3$ and K_2HPO_4. The $CaCO_3$ and K_2HPO_4 compounds were inhibitory to chlamydospore germination. Martyn (1985b) noted that *F. oxysporum* f. sp. *cucumerinum* protected watermelon against *F. oxysporum* f. sp. *niveum*. However, *F. oxysporum* f. sp. *melonis* did not protect watermelon. The most effective inducer of resistance to race 2 of *F. oxysporum* f. sp. *niveum* in Calhoun Gray and Dixielee was race 1 of *F. oxysporum* f. sp. *niveum* (Biles and Martyn, 1989). Shimotsuma et al. (1972) noted that *Helminthosporium carbonum* was more effective in cross-protection of watermelon than were *F. oxysporum* f. sp. *lycopersici* or *Verticillium albo-atrum*. The phenomenon of cross-protection is encouraging, although its commercial practicality for control of Fusarium wilt is limited at this time.

Fusarium wilt suppressive soil has been induced through a 6-year monoculture to 'Crimson Sweet' watermelon (Larkin et al., 1993). The unique resistance of 'Crimson Sweet' to Fusarium wilt in monoculture appears to be the result of a cultivar specific promotion of a biological control factor in the soil (Hopkins et al., 1987). More recently, Larkin et al. (1996) reported that nonpathogenic isolates of *F. oxysporum* from suppressive soil were the only organisms consistently effective in reducing disease (35 to 75% reduction). They further noted that the mode of action was not directly related to the ability of the antagonist to colonize the roots or reduced colonization of the pathogen, but rather, the mechanism of induced systemic resistance.

Genetic Resistance. Orton (1913) developed the first wilt-resistant watermelon cultivar by crossing Eden with an African stock citron. Cultivars are typically described as "resistant" or "susceptible" although there is actually a continuum from resistant to susceptible (Elmstrom and Hopkins, 1981; Schenck, 1961). This was further illustrated by using different inoculum concentrations (Martyn and McLaughlin, 1983). Mohammed et al. (1981) suggested that wilt resistance in citron was due to a high level of preformed phenols and phytoalexins following infection. Ming and Xian (1988) noted that the distribution of vessels, the number of central vessels of the root system and the thickness of the cell wall of the xylem determined Fusarium wilt resistance. Resistance to race 1 of *Fusarium oxysporum* f. sp. *niveum* has been attributed to a single dominant gene (Henderson et al., 1970; Netzer and Weintall, 1980). Netzer and Martyn (1989) reported resistance to race 2 of *F. oxysporum* f. sp. *niveum* in the PI-296341-FR. Martyn and Netzer (1991) suggested that genes for resistance in PI-296341-FR are not fixed. Zhang and Rhodes (1993) further noted that the resistance genes to race 2 in PI-296341-FR are epistatic with one or more recessive genes interacting with a dominant gene. Larkin et al. (1993) noted no clear pattern of surface colonization of watermelon roots by *F. oxysporum* f. sp. *niveum*. with respect to resistant or susceptible cultivars. Resistance appears to be related to rate and/or extent of colonization in the xylem. Elmstrom and Hopkins (1981) evaluated a number of cultivars and noted moderate to high resistance in several cultivars. Paulus et al. (1976) reported that seedless watermelon cultivars tested under field conditions in California were very susceptible. Currently, most of the seedless cultivars are susceptible to Fusarium wilt. It would appear that many of the present-day triploids have a similar genetic background. With triploids commanding almost 30% of the watermelon market in 1998, Fusarium wilt resistance has again become a major emphasis for seed companies.

CONCLUSIONS

Much progress has been made in the understanding of soilborne diseases of cucurbits in the last 10 to 15 years, although many basic questions remain unanswered. We have attempted to review the existing knowledge on Fusarium wilt of watermelon and identify areas requiring additional research necessary for the implementation of sound management decisions. The farmer has been lulled into complacency in recent years because of the high level of resistance in many commercial cultivars. Most seedless cultivars are susceptible to Fusarium wilt at present placing a significant portion of the watermelon acreage at risk. If race 2 increases or additional races evolve that are ecologically competitive, Fusarium wilt could again become a critical and yield-limiting disease. Research and Extension personnel should aggressively identify and promote cultural control practices for the management of Fusarium wilt. Host resistance will provide only a temporary solution unless strategies are developed utilizing cultural practices that maximize the existing genetic resistance.

Mention of a trademark, propriety product, or vendor does not constitute a guarantee or warranty of the product by the USDA and does not imply its approval to the exclusion of other products of vendors that may also be suitable.

LITERATURE CITED

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

Biles, C.L. and R.D. Martyn. 1989. Local and systemic resistance induced in watermelons by formae speciales of *Fusarium oxysporum*. Phytopathology 79:856-860.

Bruton, B.D. 1998. Soilborne diseases in Cucurbitaceae: pathogen virulence and host resistance, p. 143-146. In: J. McCreight, (ed.) Cucurbitaceae'98. ASHS Press, Alexandria, Va.

Bruton, B.D. and C.L. Patterson. 1988. Status of Fusarium wilt of watermelon in Oklahoma, p. 173-175. In: B.D. McCraw (ed). Proc. 7th Ann. Okla. Hortic. Indust. Conf. Okla. State Univ., Stillwater. Bruton, B.D., C.L. Patterson, and R.D. Martyn. 1988. Fusarium wilt (*F. oxysporum* f. sp. *niveum* race 2) of watermelon in Oklahoma. Plant Dis. 72:734.

Bruton, B.D., V.M. Russo, J. Garcia-Jimenez, and M.E. Miller. 1998. Carbohydrate partitioning, cultural practices, and vine decline diseases of cucurbits, p. 189-200. In: J. McCreight, (ed.) Cucurbitaceae'98. ASHS Press, Alexandria, Va.

Cirulli, M. 1972. Variation of pathogenicity in *Fusarium oxysporum* f. sp. *niveum* and resistance in watermelon cultivars, p. 491-500. In: Actas Congr. Un. Fitopathol. Mediter. Oeiras, 3rd.

Correll, J.C. 1991. The relationship between formae speciales, races, and vegetative compatibility groups in *Fusarium oxysporum*. Phytopathology 81:1061-1064.

Correll, J.C., C.J.R. Klittich, and J.F. Leslie. 1987. Nitrate nonutilizing mutants of *Fusarium oxysporum* and their use in vegetative compatibility tests. Phytopathology 77:1640-1646.

Crall, J.M. 1963. Physiologic specialization in *Fusarium oxysporum* f. *niveum*. Phytopathology 53:873. (Abstr.)

Davis, D. 1967. Cross-protection in Fusarium wilt diseases. Phytopathology 57:311-314.

Elmstrom, G.W. and D.L. Hopkins. 1981. Resistance of watermelon cultivars to Fusarium wilt. Plant Dis. 65:825-827.

Everett, P.H. and Blazquez. 1967. Influence of lime on the development of Fusarium wilt of watermelon. Proc. Fla. St. Hort. Soc. 80:143-148.

Fulton, H.R. and J.R. Winston. 1915. Watermelon wilt spread by contaminated seed, p 48-51. In: Biennial Report. N. C. Agri. Exp. Sta.

Gordon, T.R. and R.D. Martyn. 1997. The evolutionary biology of *Fusarium oxysporum*. Ann. Rev. Phytopathol. 35:111-128.

Gordon, T.R. and D. Okamoto. 1992. Population structure and the relationship between pathogenic and nonpathogenic strains of *Fusarium oxysporum*. Phytopathology 82:73-77.

Hadar, E. and J. Katan. 1989. The use of nitrate-nonutilizing mutants and a selective medium for studies of pathogenic strains of *Fusarium oxysporum*. Plant Dis. 73:800-803.

Henderson. W.R., S.F. Jenkins, and J.O. Rawlings. 1970. The inheritance of Fusarium wilt resistance in watermelon, *Citrullus lanatus* (Thumb.) Mansf. J. Amer. Soc. Hort. Sci. 95:276-282.

Hopkins, D.L. and G.W. Elmstrom. 1974. Chemical control of watermelon damping-off and seedling wilt. Plant Dis. Reptr. 58:114-117.

Hopkins, D.L. and G.W. Elmstrom. 1976. Effect of soil pH and nitrogen source on Fusarium wilt of watermelon on land previously cropped in watermelons. Proc. Fla. St. Hortic. Soc. 89:141-143.

Hopkins, D.L. and G.W. Elmstrom. 1984. Fusarium wilt in watermelon cultivars grown in a 4-year monoculture. Plant Dis. 68:129-131.

Hopkins, D.L., R.P. Larkin, and G.W. Elmstrom. 1987. Cultivar-specific induction of soil suppressiveness to Fusarium wilt of watermelon. Phytopathology 77:607-611.

Hopkins, D.L., R.P. Larkin, and R.J. Lobinske. 1989. Effect of watermelon cultivar monoculture on aggressiveness of *Fusarium oxysporum* f. sp. *niveum*. Phytopathology 78:1542. (Abstr.)

Huang, J.W., S.K. Sun, and T.F. Hsieh. 1989. Characteristics of suppressive soil and its application to watermelon Fusarium wilt disease management. Plant Prot. Bull., Taiwan 31:104-118.

Ioannou, N. and C.A. Poullis. 1991. Fusarium wilt of resistant watermelon cultivars associated with a highly virulent local strain of *Fusarium oxysporum* f. sp. *niveum*. Ministry of Agriculture and Natural Resources, Agr. Res. Inst., Tech. Bull. 129.

Jones, J.P., S.S. Woltz, and P.H. Everett. 1975. Effect of liming and nitrogen source on Fusarium wilt of cucumber and watermelon. Proc. Fla. State Hort. Soc. 88:200-203.

Kim, D.H., R.D. Martyn, and C.W. Magill. 1992. Restriction fragment length polymorphism groups and physical map of mitochondrial DNA from *Fusarium oxysporum* f. sp. *niveum*. Phytopathology 82:346-353.

Kim, D.H., R.D. Martyn, and C.W. Magill 1993. Mitochondrial DNA (mtDNA)-relatedness among formae speciales of *Fusarium oxysporum* in the Cucurbitaceae. Phytopathology 83:91-97.

Kistler, H.C. 1997. Genetic diversity in the plant-pathogenic fungus *Fusarium oxysporum*. Phytopathology 87:474-479.

Kistler, H.C., C. Alabouvette, R.P. Baayen, S. Bentley, D. Brayford, A. Coddington, J. Correll, M.-J. Daboussi, D. Elias, D. Fernandez, T.R. Gordon, T. Katan, H.G. Kim, J.F. Leslie, R.D. Martyn, Q. Migheli, N.Y. Moore, K. O'Donnell, R.C. Ploetz, M.A. Rutherford, B. Summerell, C. Waalwijk, and S. Woo. 1998. Systematic numbering of vegetative compatibility groups in the plant pathogenic fungus *Fusarium oxysporum*. Phytopathology 88:30-32.

Kistler, H.C. and V.P.W. Miao. 1992. New modes of genetic change in filamentous fungi. Annu. Rev. Phytopathol. 30:131-152.

Kistler, H.C., E.A. Momol, and U. Benny. 1991. Repetitive genomic sequences for determining relatedness among strains of *Fusarium oxysporum*. Phytopathology 81:331-336.

Languasco, L., S. Giosue, V. Rossi, and G. Mecenero. 1998. Statistic assignment of *Fusarium oxysporum* f.sp. *melonis* strains to races 0, 1, 2 and 1-2. J. Plant Pathol. 80:27-36.

Larkin, R.P., D.L. Hopkins, and F.N. Martin. 1990. Vegetative compatibility within *Fusarium oxysporum* f. sp. *niveum*

and its relationship to virulence, aggressiveness, and race. Can. J. Microbiol. 36:352-358.

Larkin, R.P., D.L. Hopkins, and F.N. Martin. 1993. Ecology of *Fusarium oxysporum* f. sp. *niveum* in soils suppressive and conducive to Fusarium wilt of watermelon. Phytopathology 83:1105-1116.

Larkin, R.P., D.L. Hopkins, and F.N. Martin. 1996. Suppression of Fusarium wilt of watermelon by nonpathogenic *Fusarium oxysporum* and other microorganisms recovered from disease suppressive soil. Phytopathology 86:812-819.

Martyn, R.D. 1985a. An aggressive race of *Fusarium oxysporum* f. sp. *niveum* new to the United States. Plant Dis. 69:1007.

Martyn, R.D. 1985b. Differential cross protection of watermelon to Fusarium wilt by related formae speciales. (Abstr.) Phytopathology 75:1304.

Martyn, R.D. and T.K. Hartz. 1986. Use of soil solarization to control Fusarium wilt of watermelon. Plant Dis. 70:762-766.

Martyn, R.D. 1987. *Fusarium oxysporum* f. sp. *niveum* race 2: A highly aggressive race new to the United States. Plant Dis. 71:233-236.

Martyn, R.D. and B.D. Bruton. 1989. An initial survey of the United States for races of *Fusarium oxysporum* f. sp. *niveum*. HortScience 24:696-698.

Martyn, R.D. and R.J. McLaughlin. 1983. Effects of inoculum concentration on the apparent resistance of watermelons to *Fusarium oxysporum* f. sp. *niveum*. Plant Dis. 67:493-495.

Martyn, R.D. and D. Netzer. 1991. Resistance to races 0, 1, and 2 of Fusarium wilt of watermelon in *Citrullis* sp. PI 296341-FR. HortScience 26:429-432.

McDermott, J.M. and B.A. McDonald. 1993. Gene flow in plant pathosystems. Annu. Rev. Phytopathol. 31: 353-373.

McKeen, C.D. 1951. Investigations of Fusarium wilt of muskmelons and watermelons in southwestern Ontario. Sci. Agric. 31:413-423.

Ming, W. and Z. Xian. 1988. Studies on watermelon germplasm sources resistant to Fusarium wilt disease at the seedling stage. Cucurbit Gen. Coop. 11:68.

Mohammed, M.A., A.A. Hassan, I.I. Oksh, and R. Hilal. 1981. Nature of resistance to Fusarium wilt in watermelon. Egypt J. Hort. 8:1-12.

Namiki, F., S. Izumi, T. Kayamura, and T. Shiomi. 1992. Specialization of pathogenic strains of *Fusarium oxysporum* in the Cucurbitaceae. Ann. Phytopathol. Soc. Jpn. 58:540-541.

Namiki, F., T. Shiomi, T. Kayamura, and T. Tsuge. 1994. Characterization of the formae speciales of *Fusarium oxysporum* causing wilts of cucurbits by DNA fingerprinting with nuclear repetitive DNA sequences. App. Environ. Microbiol. 60:2684-2691.

Netzer, D. 1976. Physiological races and soil population levels of Fusarium wilt of watermelon. Phytoparasitica 4: 131-136.

Netzer, D. and I. Dishon. 1973. Screening for resistance and physiological specialization of *Fusarium oxysporum* in watermelon and muskmelon. 2nd Intl. Congr. Plant Pathol. Minneapolis, Minn. (Abstr. 941)

Netzer, D. and R.D. Martyn. 1989. PI-296341, a source of resistance in watermelon to race 2 of *Fusarium oxysporum* f. sp. *niveum*. Plant Dis. 73:518.

Netzer, D. and C. Weintall. 1980. Inheritance of resistance in watemelon to race 1 of *Fusarium oxysporum* f. sp. *niveum*. Plant Dis. 64:853-854.

Orton. 1913. The development of disease-resistant varieties of plants. 4th Conf. Inter. Genetique, Paris 4:247-267.

Paulus, A.O., O.A. Harvey, J. Nelson, and F. Shibuya. 1976. Fusarium-resistant watermelon cultivars. Calif. Agric. 30:5-6.

Porter, D.R. 1928. Infection studies with watermelon wilt caused by *Fusarium niveum* EFS. Iowa Agr. Expt. Sta. Res. Bul. 112:347-368.

Reid, J. 1958. Studies on the Fusaria which cause wilt in melons I. The occurrence and distribution of races of the muskmelon and watermelon Fusaria and a histological study of the colonization of muskmelon plants susceptible or resistant to Fusarium wilt. Can. J. Bot. 36:393-410.

Schenck, N.C. 1961. Resistance of commercial watermelon varieties to Fusarium wilt. Proc. Fla. State Hort. Soc. 74:183-186.

Schenck, N.C. 1966. Effect of fumigants and fungicides on fungi in soil planted annually in watermelon. Proc. Soil Crop Sci. Soc. Florida 26:70-80.

Shimotsuma, M., J. Kuc, and C.M. Jones. 1972. The effects of prior inoculations with non-pathogenic fungi on Fusarium wilt of watermelon. HortScience 7:72-73.

Sleeth, B. 1934. *Fusarium niveum*, the cause of watermelon wilt, p.3-23. In: W. V. Agric. Exp. Stn. Bull. 257.

Smith, E.F. 1894. The watermelon disease of the South. Proc. Am. Assoc. Adv. Sci. 43:289-290.

Smith, E.F. 1899. Wilt disease of cotton, watermelon, and cowpea. U.S. Dept. Agr. Div. Veg. Physiol. Pathol. Bull. 17. 73 pp.

Smith, D.T. and A.W. Cooley. 1973. Wild watermelon emergence and control. Weed Sci. 21:570-573.

Smith, D.T., A.W. Cooley, and T.E. Hemphill. 1972. Distribution, growth, and competitiveness of wild watermelon as a weed. Tex. Agr. Exp. Sta. Mis. Pub. 1056.

Snyder, W.C. and H.N. Hansen. 1940. The species concept in *Fusarium*. Amer. J. Bot. 27:64-67.

Snyder, W.C. and H.N. Hansen. 1941. The species concept in *Fusarium* with reference to section Martiella. Amer. J. Bot. 28:738-742.

Taubenhaus, J.J. 1935. Seeds of watermelons and okra as possible carriers of Fusarium wilt. Phytopathology 25:969. (Abstr.)

Walker, M.N. 1941. Fusarium wilt of watermelons. I. Effect of soil temperature on the wilt disease and the growth of watermelon seedlings. Fla. Agric. Exp. Sta. Bull. 33:3-29.

Zhang, X. and B. Rhodes. 1993. Inheritance of resistance to races 0, 1, and 2 of *Fusarium oxysporum* f. sp. *niveum* in watermelon (Citrullus sp. PI 296341). Cucurbit Genetics Cooperative 16:77-78.