Disease Notes

Plant Disease "Disease Notes" are originally published on-line and are available several weeks before being reprinted here. A note's official date of publication is the date it was placed on-line. Information concerning on-line access appears at the front of this journal under *Editorial Policies.*

A Cowpea Seed Rot Disease Caused by *Fusarium equiseti* Identified in Nigeria. S. O. Aigbe, B. Fawole, Department of Crop Protection and Environmental Biology, University of Ibadan, Nigeria; and D. K. Berner, IITA, Ibadan, Nigeria. Plant Dis. 83:964, 1999; published on-line as D-1999-0803-02N 1999. Accepted for publication 22 July 1999.

Fusarium equiseti (Corda) Sacc., reported on cowpea (*Vigna unguiculata* (L.) Walp.) seeds in India (2), was isolated for the first time in Nigeria from naturally infected cowpea seeds. Cowpea, cv. IT90K-76, seeds (400) from plants grown in Nigeria were surface-disinfested in 0.05% NaOCl and placed on moist filter paper in petri dishes (10 seeds per dish) and then in a dark incubator for 4 days at 27°C. After incubation, some seeds had fungal mycelia growing on their surfaces. When cultured on potato dextrose (PDA) and Spezieller Nährstoffarmer (SNA) agars, the fungi produced macroconidia characteristic of *F. equiseti* (1). Septate macroconidia were three to six celled with extended apical and distinctive foot-shaped basal cells. *F. equiseti* was recovered from 4.25% of seeds, and incidence correlated positively with development of seed rot symptoms. To confirm pathogenicity, 80 cowpea seeds were surface-disinfested with NaOCl, and 40 were soaked for 6 h in a suspension of 3×10^5 conidia of *F. equiseti* per ml of water. The remaining seeds were soaked in sterile distilled water. After incubation, white mycelia developed on 87.5% of seeds soaked in the conidial suspension and rotted without germinating. Only 5% of seeds soaked in sterile water developed seed rot symptoms. When cultured on PDA and SNA, fungi isolated from artificially infested seeds with rot symptoms again were identified as *F. equiseti*.

References: (1) P. E. Nelson et al. 1983. *Fusarium* species: An illustrated Manual for Identification. Pennsylvania University Press, University Park. (2) O. K. Sinha and M. N. Khare. Seed Sci. Technol. 5:721, 1977.

Evaluation of *Colletotrichum gloeosporioides* for Biological Control of *Miconia calvescens* in Hawaii. E. M. Killgore and L. S. Sugiyama, Hawaii Department of Agriculture, P.O. Box 22159, Honolulu 96823-2159; R. W. Barreto, Departamento de Fitopatologia, Universidade Federal de Viçosa MG, 36571-000, Brazil; and D. E. Gardner, Pacific Island Ecosystems Research Center, Biological Resources Division, USGS, Department of Botany, University of Hawaii at Manoa, Honolulu 96822. Plant Dis. 83:964, 1999; published on-line as D-1999-0804-02N 1999. Accepted for publication 26 July 1999.

Miconia calvescens (Melastomataceae), from the Neotropics, is a noxious forest weed in Hawaii. We evaluated an isolate of *Colletotrichum gloeosporioides* that causes leaf spots on *Miconia* spp. in Brazil for its potential in biological control. Hawaii has no native Melastomataceae genera but does have members of 12 introduced genera. Following Wapshere's centrifugal phylogenetic method (2), eight species of Melastomataceae genera in Hawaii were inoculated in addition to *Miconia* spp. Naturalized and native Hawaiian members of the order Myrtales also were inoculated to determine host specificity, including *Terminalia catappa* (Combretaceae); *Cuphea hysopifolia* and *C. ignea* (Lythraceae); *Arthrostema ciliatum, Clidemia hirta, Dissotis rotundifolia, Heterocentron subtriplinervium, Medinilla scortechenii, Melastoma candidum, Pterolepsis glomerata,* and *Tibouchina herbaceae* (Melastomataceae); *Eucalyptus grandis, Eucalyptus microcorys, Eugenia reinwardtiana, Eugenia uniflora, Leptospermum laevigatum, Melaleuca quinquenervia, Metrosideros polymorpha, Psidium guajava,* and *Syzgium malaccanse* (Myrtaceae); *Fuchsia magellanica* and *Oenothera stricta* (Onagraceae); and *Wikstroemia oahuensis* and *W. uva-ursi* (Thymelaeaceae). All *M. calvescens* plants were grown from seed collected in Hawaii. Other test plants were grown from seeds or cuttings in artificial potting medium in a greenhouse. Plants had 6 to 8 mature leaves when inoculated. *C. gloeosporioides* was cultured on 10% potato dextrose agar supplemented with plain agar (35 g/liter) and incubated under constant fluorescent illumination at 20°C. Conidia were harvested by flooding 10- to 14-day-old cultures with sterile tap water, followed by light scraping with a scalpel. Conidial suspensions were adjusted to 10^6 conidia per ml and applied to both leaf surfaces with a hand-held sprayer. Inoculated plants were kept at 100% relative humidity and 16 to 25°C for 48 h. Four replicate plants and one plant of *M. calvescens* per species were inoculated. Plants were observed for symptom development for up to 6 weeks. The entire test was repeated once. Lesions were visible after 7 to 10 days. Young lesions had chlorotic halos and expanded in a roughly circular pattern to diameters of 5 to 10 mm. Mature lesions developed necrotic centers, coalesced, and became dry and brittle with age, resulting in extensive leaf necrosis. Defoliation of moderately to severely infected leaves occurred ≈30 days after inoculation. With the exception of *M. calvescens*, *C. gloeosporioides* did not produce visible symptoms on test plants. The failure of *Clidemia hirta*, the taxonomic species most closely related to *M. calvescens*, to become symptomatic was particularly significant relative to the centrifugal phylogenetic concept. The results demonstrate that our pathogen (VIC 19306) is distinct from *C. gloeosporioides* f. sp. *clidemiae* (1), which did not infect *M. calvescens*. We designate our pathogen *C. gloeosporioides* f. sp. *miconiae*. Voucher specimens (VIC 19306, Sana, RJ, 24.II.1998, and R. W. Barreto) and cultures are maintained at the Departamento de Fitopatologia, Universidade Federal de Viçosa MG, Brazil.

References: (1) E. E. Trujillo et al. Plant Dis. 70:974, 1986. (2) A. J. Wapshere. Ann. Appl. Biol. 77:201, 1974.

First Report of Copper-Tolerant *Pseudomonas syringae* pv. *tomato* in Virginia. S. A. Alexander, Eastern Shore AREC, VPI & State University, Painter, VA 23420; S. H. Kim, Plant Disease Diagnostic Laboratory, Pennsylvania Department of Agriculture, Harrisburg 17110; and C. M. Waldenmaier, Eastern Shore AREC, VPI & State University, Painter, VA 23420. Plant Dis. 83:964, 1999; published on-line as D-1999-0722-01N 1999. Accepted for publication 20 July 1999.

Bacterial speck of tomato, caused by *Pseudomonas syringae* pv. *tomato*, is an important disease of fresh-market tomatoes along the Eastern Shore of Virginia. *P. syringae* pv. *tomato* was first identified in Northampton County on 17 May 1993, using the Biolog software program. During the spring of 1998, a field of tomato plants showed symptoms of bacterial speck. Three isolations on tryptic soy agar were made from symptomatic leaves and fruit tissues taken from young transplanted tomato plants, cv. Sunpride. The isolates were identified as *P. syringae* pv. *tomato*, using Biolog. A representative isolate was sent to the Pennsylvania Department of Agriculture, Plant Disease Diagnostic Laboratory, Harrisburg, for confirmation. The Virginia isolate was transferred to King's medium B, and identification of *P. syringae* pv. *tomato* was confirmed by fulfilling Koch's postulates, matching with the Biolog database, and testing for levan production (+), oxidase reaction (–), potato soft rot (–), arginine dihydrolase production (–), and tobacco hypersensitivity (+). In vitro growth inhibition of the 1998 Virginia *P. syringae* pv. *tomato* isolate required anhydrous cupric sulfate at 368 µg/ml compared with only 175 µg/ml for a known copper-sensitive 1998 Pennsylvania isolate. Therefore, the 1998 Virginia isolate was considered a copper-tolerant strain of *P. syringae* pv. *tomato*. For field evaluation, three copper treatments and two noncopper treatments were established in a randomized complete block design replicated four times. Treatments were initiated on 19 May and reapplied every 7 days for a total of 10 applications. Three disease ratings were taken every 7 days beginning on 30 June. For copper hydroxide (2.24 kg/ha; Kocide DF) plus mancozeb (2.24 kg/ha; Dithane); chlorothalonil (2.34 liters/ha; Bravo) plus copper salts of fatty and rosin acids (2.34 liters/ha; Tenncop); 2% maneb plus 66% copper sulfate (6.72 kg/ha; Cuprofix); and an untreated control, area under the disease progress curve (AUDPC) values were 319, 468, 478, and 438, respectively. There was no significant difference ($P = 0.05$) between the copper treatments and untreated control, confirming laboratory findings. In contrast, noncopper treatments of acibenzolar (21g/ha; Actigard), and acibenzolar (21 g/ha) plus mancozeb (2.24 kg/ha) (Actigard plus Dithane) were significantly different ($P = 0.05$) from the untreated control and copper treatments with AUDPC values of 116 and 160, respectively. This is the first report of copper-tolerant *P. syringae* pv. *tomato* in Virginia.

(*Disease Notes* continued on next page)

Two Field Isolates of Tomato Spotted Wilt Tospovirus Overcome the Hypersensitive Response of a Pepper (*Capsicum annuum*) Hybrid with Resistance Introgressed from *C. chinense* PI152225. P. Roggero, V. Melani, and M. Ciuffo, Istituto di Fitovirologia Applicata, CNR, Str. delle Cacce 73, I-10135 Torino, Italy; L. Tavella and R. Tedeschi, Di.Va.P.R.A. Entomologia e Zoologia Applicate all'Ambiente "C. Vidano," Università, Via L. da Vinci 44, I-10095 Grugliasco, Torino, Italy; and V. M. Stravato, Peto Italiana srl, Via Canneto di Rodi, I-04010 Borgo Sabotino, Latina, Italy. Plant Dis. 83:965, 1999; published on-line as D-1999-0805-01N 1999. Accepted for publication 4 August.

The hypersensitive response to tomato spotted wilt tospovirus (TSWV) present in *Capsicum chinense* PI152225 (1) was introgressed into *C. annuum* cultivars. During the summer of 1998, a hybrid with good agronomic performance was grown in glasshouses in Albenga, Liguzia Region of northwestern Italy, an area where infection by TSWV in pepper has been severe since 1992. In August, observations of different susceptible cultivars revealed that >50% of plants had TSWV-like symptoms, whereas the resistant hybrid remained healthy, except for two plants that showed virus-like symptoms on apical leaves and fruits. From the infected plants, tospoviruses (coded P164/6 and P166) were transmitted by sap-inoculation to *Nicotiana benthamiana*. Triple-antibody sandwich enzyme-linked immunosorbent assay with a panel of monoclonal antibodies against the TSWV nucleocapsid, but with different reactivity to the related species groundnut ringspot (GRSV) and tomato chlorotic spot (TCSV) viruses, indicated the isolates were TSWV. The host ranges of the isolates were wide and typical of normal TSWV isolates. Thus, they incited typical symptoms in all 50 TSWV-susceptible *C. annuum* cv. Quadrato d'Asti plants. However, isolate P164/6 also systemically infected 12 of 27 *C. chinense* PI152225 and 14 of 19 *C. chinense* PI159236 plants. These accessions are normally resistant to TSWV (1). Isolate P166 systemically infected 7 of 17 *C. chinense* PI152225 and 6 of 11 *C. chinense* PI159236 plants. Systemically infected plants showed severe necrosis, and some plants died. Other plants showed only necrotic local lesions. The response by *C. chinense* differed from that caused by typical TSWV, which causes only local lesions, and from both GRSV and TCSV, which cause mosaic but no necrosis in 100% of plants. The two new TSWV isolates were tested for transmission using a local population of *Frankliniella occidentalis* in a leaf disk assay with susceptible *C. annuum*. Transmission rates were high: 93.7% (63 thrips) for isolate P164/6 and 89.9% (49 thrips) for P166. Thus, the fitness of the two TSWV resistance-breaking isolates (a wide experimental host range and high transmission rates by the natural vector) was as high as that of typical TSWV. The absence of systemic infection in some *C. chinense* PI152225 and PI159236 plants that are resistant to typical TSWV suggests the possibility of selecting plants resistant to these pathotypes. This is the first report of field tospovirus isolates typed as TSWV (according to the current taxonomy based on nucleocapsid serology) overcoming the hypersensitive response of *C. chinense* PI152225 and PI159236, an ability previously found only in closely related viruses: TCSV and GRSV (2). Other TSWV-like isolates systemic on *C. chinense* were not typed further (3,4).

References: (1) L. L. Black et al. Plant Dis. 75:863, 1991. (2) L. S. Boiteux and A. C. DeAvila. Euphytica 75:139, 1994. (3) H. A. Hobbs et al. Plant Dis. 78:1220, 1994. (4) B. Moury et al. Euphytica 94:45, 1997.

First Report of Black Rot of Cauliflower and Kale Caused by *Xanthomonas campestris* pv. *campestris* in Yugoslavia. A. Obradović, Centre for Vegetable Crops, 11420 Smed. Palanka, Yugoslavia; and M. Arsenijević, Institute for Plant Protection, T. Drajzera 9, 11000 Belgrade, Yugoslavia. Plant Dis. 83:965, 1999; published on-line as D-1999-0802-01N 1999. Accepted for publication 20 July 1999.

In Yugoslavia, *Xanthomonas campestris* pv. *campestris* was isolated from forage kale in 1964 and cabbage in 1997 (1). Recently, the incidence and severity of black rot symptoms on cabbage, cauliflower, and kale have increased. Gram-negative, rod-shaped, motile bacteria were isolated from the diseased leaf and vascular tissues of cauliflower and kale plants collected from 1995 to 1998. The isolates formed yellow, convex, mucoid colonies on yeast dextrose chalk medium, metabolized glucose oxidatively, grew at 37°C, hydrolyzed gelatin and esculin, produced acids from D-arabinose, glucose, sucrose, and trehalose, and did not reduce nitrates. They were nonfluorescent, amylolytic and pectolytic, oxidase negative and catalase positive, and tolerant to 5% NaCl but not to 0.1% triphenyl tetrazolium chloride. Koch's postulates were completed by injecting bacterial suspensions (10^8 CFU/ml) into leaf petioles of cabbage, cauliflower, and kale seedlings (2- to 3-leaf stage). Dark green watersoaking of petioles and leaf veins followed by yellowing and collapse of inoculated plants was observed after 3 to 5 days. When compared with published information (2), the isolates were identified as *X. campestris* pv. *campestris*. This is the first occurrence of this bacterium in cauliflower and kale in Yugoslavia.

References: (1) O. Jovanovic et al. Plant Prot. Belgrade 221:175, 1997. (2) N. W. Schaad. 1988. Laboratory Guide for Identification of Plant Pathogenic Bacteria. 2nd ed. The American Phytopathological Society, St. Paul, MN.

First Report of *Bipolaris sorghicola* on Johnsongrass in Argentina. H. Acciaresi and C. Mónaco, Cerealicultura, Centro de Estudios Fitopatológicos, Facultad de Cs. Agr. y Ftales (UNLP), CC 31 (1900), La Plata, Argentina, CIC. Plant Dis. 83:965, 1999; published on-line as D-1999-0803-03N 1999. Accepted for publication 14 July 1999.

Target leaf spot was found on johnsongrass (*Sorghum halepense* (L.) Pers.) in La Plata, Argentina (34°54′S, 58°30′W). Up to 80% disease severity was observed in adult plants. In most diseased plants, spots were well defined, delimited by leaf veins, and elongated. Individual lesions ranged in size from small spots (2 to 3 mm) to large lesions (10 to 15 mm). Lesions sometimes coalesced to produce extensive areas of necrosis. Isolates identified as *Bipolaris sorghicola* (Lefebre & Scherwin) Alcorn (1) grew slowly on potato dextrose agar medium, producing a compact dark brown mycelium. Conidia were 40 to 80 µm long, slightly curved and golden brown, with three to seven pseudosepta. Although germination of conidia was mainly bipolar, lateral germination sometimes was observed. Twenty monoconidial isolates were inoculated at 10^5 conidia per cm^3 on johnsongrass at the 4-leaf stage to confirm the pathogenicity of *B. sorghicola*. After inoculation, plants were placed in a high-humidity chamber for 24 h; thereafter, they were kept at 25°C day and 18°C night temperatures. Typical lesions developed after 10 days. The pathogen was reisolated from lesions after 15 days. Johnsongrass is one of the most noxious weeds in the world. Due to its potential as a biocontrol agent, further studies are needed to determine the effect of *B. sorghicola* on johnsongrass.

Reference: (1) J. L. Alcorn. Mycotaxon 27:1, 1983.

First Report of *Acidovorax avenae* subsp. *citrulli* as a Pathogen of Cucumber. H. L. Martin and R. G. O'Brien, Queensland Horticulture Institute, P.O. Box 591, Ayr Qld 4807, Australia; and D. V. Abbott, Bowen Crop Monitoring Services Pty. Ltd., 67 Field St., Queen's Beach Qld 4805, Australia. Plant Dis. 83:965, 1999; published on-line as D-1999-0729-01N 1999. Accepted for publication 20 July 1999.

In March 1999, a foliar bacterial disease was observed in a commercial crop of cucumber (*Cucumis sativus* L.) cv. Jetset in Gumlu in northern Queensland, Australia. Initial symptoms consisted of angular, chlorotic, watersoaked lesions that later dried to necrotic areas of light brown, dead tissue. White bacterial ooze was commonly found on the undersides of young water-soaked lesions. Lesions were delimited by veins and distributed uniformly over leaf surfaces, and more than 20% of the crop was affected. No symptoms were observed on plant stems or fruits. Bacterial streaming from the edges of freshly cut young lesions was clearly visible in a droplet of water under ×100 magnification in the laboratory. Isolations were made from young lesions on King's medium B (1). A slow-growing, white, gram-negative, nonfluorescent bacterium was consistently isolated. Three isolates of the bacterium were identified, using the Biolog software program (Biolog, Hayward CA), and in each instance, the bacterium was confirmed as *Acidovorax avenae* subsp. *citrulli*, with a similarity of >0.80. Koch's postulates were completed with 8-day-old glasshouse-grown cucumber (cv. Jetset) seedlings. Seedlings were misted until runoff with a bacterial suspension of 3×10^8 CFU/ml and enclosed in plastic bags for ≈30 h at 22°C. Water-soaked lesions were observed on cucumber cotyledons 4 days after inoculation. This is the first report of *A. avenae* subsp. *citrulli* as a pathogen of cucumber.

Reference: (1) E. O. King et al. J. Lab. Clin. Med. 44:301, 1954.

(*Disease Notes* continued on next page)

Bacterial Blight, a New Disease of *Lobelia ricardii* Caused by *Pseudomonas cichorii*. M. L. Putnam, Oregon State University, Botany and Plant Pathology, Corvallis 97331-2902. Plant Dis. 83:966, 1999; published online as D-1999-0824-01N 1999. Accepted for publication 20 August 1999.

Lobelia ricardii is a small perennial bedding plant grown for its abundant purple flowers. During April 1998, young lobelia plants with severe leaf blight were observed in a commercial nursery in Washington State. Symptoms consisted primarily of watersoaking of leaves, followed by collapse of leaves, watersoaking of stems, and eventual dieback of all foliage. Isolations from affected tissues were made on King's medium B (KMB). A fluorescent, gram-negative, oxidase-positive, arginine dihydrolase- and potato rot-negative, levan-producing bacterium was recovered that produced a hypersensitive response in tobacco. The bacterium was identified as *Pseudomonas cichorii* by carbohydrate utilization profiling (Biolog, Hayward, CA). A single-colony isolate of the bacterium was raised on KMB. A bacterial suspension was made from a 24-h agar culture of the isolate, using 0.1 M phosphate buffer, pH 6.2, with 0.2% gelatin (PBG). The suspension concentration was adjusted to 1×10^7 cells per ml by direct enumeration. The suspension (3 ml) was atomized onto leaves of five potted 5-month-old lobelia plants that were then bagged in plastic and maintained at 22°C. Bags were removed after 2 days. Eight days after inoculation, foliage was extensively watersoaked, and plants showed symptoms similar to those observed in the nursery. The plants later died. *P. cichorii* was reisolated from all inoculated plants. Control plants, which were treated with PBG, did not develop any symptoms nor was *P. cichorii* isolated from them. This is the first report of *P. cichorii* as a pathogen of *L. ricardii*.

First Report on Natural Occurrence of Tomato Spotted Wilt Tospovirus in Basil (*Ocimum basilicum*). G. E. Holcomb, R. A. Valverde, J. Sim, and J. Nuss, Department of Plant Pathology and Crop Physiology, Louisiana Agricultural Experiment Station, Louisiana State University Agricultural Center, Baton Rouge 70803. Plant Dis. 83:966, 1999; published on-line as D-1999-0728-01N 1999. Accepted for publication 22 July 1999.

Virus-like symptoms were observed on basil plants (*Ocimum basilicum* L. 'Mrs. Burns Lemon' [MBL]) growing in containers and a demonstration plot at the Louisiana State University Burden Research Plantation, Baton Rouge, during July 1998. Symptoms consisted of ring spots, leaf distortion, and severe mosaic. Mechanical transmission of the suspect virus by sap inoculation from infected MBL to basil cvs. MBL, Aussie Sweet, Cinnamon, Siam Queen, and Sweet Dani was successful. Symptoms were similar to those on infected MBL. *Nicotiana benthamiana* Domin. reacted with local chlorotic spots followed by severe yellows, necrosis, and death. Electron microscopy of thin sections of infected basil revealed virus inclusions but no virus particles. However, infected *N. benthamiana* revealed the presence of 82-nm membrane-bound particles in the cytoplasm. The virus was identified from basil and *N. benthamiana* as the common strain of tomato spotted wilt tospovirus (TSWV) by enzyme-linked immunosorbent assay (Agdia, Elkhart, IN). An outbreak of thrips insects during the summer drought in 1998 was probably responsible for the occurrence of TSWV in basil. This is the first report of the occurrence of TSWV in basil (1).

Reference: (1) A. A. Brunt et al., eds. 1996. Plant Viruses Online: Descriptions and Lists from the VIDE Database. Published online by Australian National University, Canberra.

Outbreak of *Erwinia carotovora* on *Zantedischia* spp. in South Africa. E. L. Mansvelt, ARC-Fruit, Vine and Wine Research Institute, Private Bag X5013, Stellenbosch 7599, South Africa; and E. Carstens, Directorate Plant Production, Health and Quality, Private Bag X5015, Stellenbosch 7599, South Africa. Plant Dis. 83:966, 1999; published on-line as D-1999-0730-02N 1999. Accepted for publication 28 July 1999.

In South Africa, summer-flowering Arum lilies are grown for the tuber, potted plant, and cut flower markets. In 1998, an outbreak of soft rot was detected on *Zantedischia oculata* cv. Black Magic and *Z. elliottiana* plants from several nurseries. Crop losses of up to 25% were incurred. The initial symptom was wilting of leaves. When plants were lifted from the soil, soft rot of the tuber was found. Tuber rot usually developed on one side, and plants developing from affected tubers wilted and died. No discoloration of leaf or tuber tissues was found. Isolations from diseased tissues consistently yielded bacterial colonies that were translucent, white, and glistening and that had entire margins on nutrient agar. Ten representative isolates were chosen for further characterization. *Erwinia carotovora* subsp. *carotovora* strain B56 was included as a reference strain. All isolates were gram-negative rods, oxidase and arginine dihydrolase negative, catalase positive, and facultatively anaerobic. They degraded pectate and rotted potato slices but did not hydrolyze starch. All isolates fermented glucose, reduced nitrates to nitrites, and grew at a maximum temperature of 37°C. Isolates produced acids from D(+)-glucose, D(+)-cellobiose, melibiose, amygdalin, L(+)-arabinose, D-mannitol, L(+)-rhamnose, sucrose, ribose, D(–)xylose, and D(–)glucose but not from D-arabinose, D-sorbitol, or maltose. Isolates liquefied gelatin and used citrate, arbutine, esculin, salicin, and cellobiose as the sole carbon source. Pathogenicity to *Zantedischia* spp. was tested by injection of tubers with an inoculum suspension containing 10^8 CFU/ml. Control plants were inoculated with sterile distilled water. Inoculated plants were kept in a greenhouse at 24°C. Symptoms developed 2 days after inoculation with the pathogen and appeared to be identical to those observed on diseased material in nurseries. Control plants did not rot. The bacterium was readily reisolated from diseased plants, confirmed to be the inoculated pathogen, and identified as *E. carotovora*, based on morphological, biochemical, and physiological characteristics and pathogenicity. *E. aroideae* has been reported to cause soft rot of rhizomes of winter-flowering Arum lilies (*Z. aethiopica*) in South Africa (1). However, this is the first report of soft rot caused by *E. carotovora* subsp. *carotovora* on tubers of *Z. oculata* and *Z. elliottiana* plants in South Africa.

Reference: (1) V. Wager. 1970. Flower Garden Diseases and Pests. Purnell, Cape Town, South Africa.

First Report of Natural Infection of Peanut (Groundnut) by Impatiens Necrotic Spot Tospovirus (Family *Bunyaviridae*). S. S. Pappu, Department of Entomology, University of Georgia, Coastal Plain Experiment Station, Tifton 31793; M. C. Black, Department of Plant Pathology and Microbiology, Texas A&M University, Uvalde 78802; H. R. Pappu, T. B. Brenneman, and A. K. Culbreath, Department of Plant Pathology, and J. W. Todd, Department of Entomology, University of Georgia, Coastal Plain Experiment Station, Tifton 31793. Plant Dis. 83:966, 1999; published on-line as D-1999-0730-01N 1999. Accepted for publication 27 July 1999.

Impatiens necrotic spot tospovirus (INSV) of the family *Bunyaviridae* is an important viral pathogen of ornamentals and a major constraint in the greenhouse industry (2). Evidence of natural infection of peanut (groundnut, *Arachis hypogaea* L.) by INSV was found in samples collected from three sites in Frio County, TX, and one site each in Mitchell and Tift counties, GA, during October 1998. Roots from several plants were tested by enzyme-linked immunosorbent assay for tomato spotted wilt tospovirus (TSWV) and INSV. Symptoms on individual mature plants positive for INSV were the same as those associated with late-season TSWV infections: plants appeared yellow and wilted, internal taproot and crown were necrotic, and plant death resulted (1). At one Texas site, three of five composite samples were positive only for INSV. One composite sample at a second Texas site was positive for both TSWV and INSV. Double infections were found in three of four TSWV-positive samples at a third Texas site. In Mitchell County, GA, three of four samples tested were positive only for TSWV, and one was positive for both TSWV and INSV. In Tift County, GA, 11 of 23 samples tested were positive only for INSV, whereas 4 were positive only for TSWV. Double infections were found in 5 of 23 samples. The presence of INSV in the sample from Mitchell County was verified by immunocapture-polymerase chain reaction (PCR) (4). The apparently low titer of INSV in the doubly infected plant necessitated two cycles of PCR for detection of INSV sequences. A primer pair that can amplify most tospoviruses was used for the first PCR cycle (3). Using the PCR product obtained, a second PCR cycle was performed with one tospovirus-specific and one INSV-specific primer. This approach resulted in a product of the expected size (≈298 bp). The PCR product was cloned in a pGEM-T vector and sequenced. Comparisons indicated the sequence obtained from the infected peanut sample from Georgia was 99% identical to the respective S RNA region from known INSV isolates. Serological and molecular sequence data suggest the peanut samples were infected by INSV. Future surveys and screenings of peanut plants for spotted wilt disease should include a test for INSV.

References: (1) A. K. Culbreath et al. Plant Dis. 75:863, 1991. (2) M. Daughtrey et al. Plant Dis. 81:1220, 1997. (3) R. Dewey et al. Virus Genes 13:255, 1996. (4). R. K. Jain et al. Plant Dis. 82:900, 1998.

(*Disease Notes* continued on next page)

Additional Ornamental Species as Hosts of Impatiens Necrotic Spot Tospovirus in Italy. P. Roggero, M. Ciuffo, and G. Dellavalle, Istituto di Fitovirologia Applicata, CNR, Str. delle Cacce 73, I-10135 Torino, Italy; P. Gotta and S. Gallo, Settore Fitosanitario Regionale, Regione Piemonte, Torino, Italy; and D. Peters, Department of Virology, Wageningen University, The Netherlands. Plant Dis. 83:967, 1999; published on-line as D-1999-0804-01N 1999. Accepted for publication 3 August 1999.

Impatiens necrotic spot (INSV) and tomato spotted wilt (TSWV) tospoviruses are among the most important viral pathogens of glasshouse ornamental plants worldwide (1). Tospovirus infections drastically reduce the market value of plants and create certification problems for international traders. As with TSWV, the number of natural host species recorded for INSV is steadily increasing (2). In 1998, severe INSV infections were found on different ornamental plants in glasshouses in the Piedmont Region of northwestern Italy, together with heavy infestations of the thrips *Frankliniella occidentalis*. A high proportion of plants were infected with INSV, as shown by enzyme-linked immunosorbent assay (ELISA) with polyclonal antisera and monoclonal antibodies against its nucleocapsid protein. Results were confirmed by sap-inoculation to indicator hosts. Some species were already known to be susceptible to INSV, but others are apparently new hosts (2): *Ageratum houstonianum* (Asteraceae), showing small necrotic rings and leaf malformation; *Cordyline terminalis* (Agavaceae), showing chlorotic-necrotic ringspots and leaf malformation; *Dianthus chinensis* (Caryophyllaceae), showing stunting, mosaic, and leaf malformation (some plants had symptomless infections); *Episcia capreata* (Gesneriaceae), showing necrotic spots on stems and leaves; *Godetia grandiflora* (Onagraceae), showing necrotic rings; *Maranta leuconeura* (Marantaceae), showing chlorotic-necrotic spots and apical malformation; *Peperomia obtusifolia* (Piperaceae), showing necrotic ringspots and leaf malformations; *Scindapsus aureus* (Araceae), showing necrotic spots and rings; *Torenia fournieri* (Scrophulariaceae), showing necrosis on stems and apexes. Thrips feeding damage was high on some species, particularly those showing necrotic ringspot symptoms. It often was difficult to distinguish between true systemic symptoms and local INSV infection at thrips feeding sites. *Capsicum* sp., *Coleus blumei*, and *Dahlia* sp., which also were infected in our study, are species known to be infrequent hosts of INSV (2).

References: (1) M. L. Daughtrey et al. Plant Dis. 81:1220, 1997. (2) D. Peters. 1998. Pages 107-110 in: Abstr. 4th Int. Symp. Tospovirus Thrips Floral Vegetable Crops. University of Wageningen, The Netherlands.

First Report of *Fusarium oxysporum* f. sp. *lycopersici* Race 2 on Tomato in Italy. V. M. Stravato, Peto Italiana, Centro di Ricerca di Latina, Via Canneto di Rodi, 04010 Borgo Sabotino, Italy; R. Buonaurio and C. Cappelli, Dipartimento di Arboricoltura e Protezione delle Piante, Borgo XX Giugno, 74, 06121 Perugia, Italy. Plant Dis. 83:967, 1999; published on-line as D-1999-0803-01N 1999. Accepted for publication 27 July 1999.

During the summer of 1997, symptoms of Fusarium wilt were observed on tomato (*Lycopersicon esculentum* Mill.) cvs. Monica F1 and PS 110, which bear the *I* gene for resistance to race 1 of *Fusarium oxysporum* Schlechtend.:Fr. f. sp. *lycopersici* (Sacc.) W.C. Snyder & H.N. Hans., in two commercial production greenhouses in Latium (Fondi) and one greenhouse in Sardinia (Oristano). Infected plants showed yellowing, stunting, vascular discoloration, and premature death. A fungus from tomato stems with discolored vascular tissue was consistently isolated on potato dextrose agar (PDA) and, based on morphological features, was identified as *F. oxysporum*. To verify the pathogenicity of four fungal isolates, cv. Bonny Best tomato plants, which do not carry genes for Fusarium wilt resistance, were inoculated by dipping roots of 2-week-old seedlings in a suspension of 10^5 microconidia per ml for 30 s. Inocula were obtained from 1-week-old fungal cultures grown on PDA. Roots of control plants were dipped in water. Seedlings were transplanted to pots containing peat and river sand (1:1, vol/vol) and placed in a greenhouse at 20 to 25°C. One month after inoculation, all fungal isolates provoked wilting of inoculated plants. No symptoms were observed on control plants. The morphological features of the fungus reisolated from diseased plants were similar to those of the original isolates. Based on the pathogenicity test, we concluded that the fungal isolates belong to *F. oxysporum* f. sp. *lycopersici*. To determine the races of the fungal isolates, differential tomato lines VFN8 (*I* gene for resistance to race 1), Florida MH-1 (*I* and *I2* genes for resistance to races 1 and 2), and I3R (*I*, *I2*, and *I3* genes for resistance to races 1, 2, and 3) were inoculated with the four fungal isolates, using the same procedure described for the pathogenicity test. Because disease symptoms were detected on VFN8 but not on Florida MH-1 and I3R, we deduced that the fungal isolates belong to *F. oxysporum* race 2. This is the first report of *F. oxysporum* f. sp. *lycopersici* race 2 in Italy. Previous research indicated that race 1 is present in Italy (1). Currently, many commercially acceptable cultivars resistant to races 1 and 2 are available to Italian greenhouse growers.

Reference: (1) M. Cirulli. Phytopathol. Mediterr. 4:63, 1965.

First Report of Clover Proliferation Phytoplasma in Strawberry. R. Jomantiene, USDA-ARS, Fruit Laboratory, Beltsville, MD 20705, and Institute of Botany, Vilnius, Lithuania; J. L. Maas, USDA-ARS, Fruit Laboratory, Beltsville, MD 20705; E. L. Dally and R. E. Davis, USDA-ARS, Molecular Plant Pathology Laboratory, Beltsville, MD 20705; and J. D. Postman, USDA-ARS, National Clonal Germplasm Repository, Corvallis, OR 97333. Plant Dis. 83:967, 1999; published on-line as D-1999-0810-01N 1999. Accepted for publication 3 August 1999.

In 1996, diseased plants of *Fragaria virginiana* Duchesne were collected from a native population in Quebec, Canada, and sent to the National Clonal Germplasm Repository in Corvallis, OR, where grafting onto disease-free plants of *F. chiloensis* (L.) Duchesne (4) was performed. Plants of both species were sent to Beltsville, MD, for identification of a phytoplasma possibly associated with the disease symptoms of dwarfing and multibranching crowns. A phytoplasma was found in both species and characterized as the strawberry "multicipita" (SM) phytoplasma, which is representative of subgroup 16SrVI-B, a new subgroup of the clover proliferation (CP) group (2). In 1999, we observed commercial strawberry (*Fragaria* × *ananassa* Duchesne) plants collected in California and Maryland that were stunted and chlorotic or exhibited these symptoms in addition to small, distorted leaves. Infected *F.* × *ananassa* plants, as well as diseased *F. virginiana* and grafted *F. chiloensis* plants previously infected by the SM phytoplasma, were assessed for phytoplasma infection by nested polymerase chain reactions primed by phytoplasma universal primer pairs R16mF2/R1 and F2n/R2 (1) or P1/P7 (3) and F2n/R2 for amplification of phytoplasma 16S rDNA (16S rRNA gene) sequences. Phytoplasma-characteristic 1.2-kbp DNA sequences were amplified from all diseased plants. No DNA sequences were amplified from healthy plants. Restriction fragment length polymorphism patterns of rDNA digested with *Alu*I, *Kpn*I, *Hha*I, *Hae*III, *Hin*fI, *Hpa*II, *Mse*I, *Rsa*I, and *Sau*3A1 endonucleases indicated that all plants were infected by a phytoplasma that belonged to subgroup 16SrVI-A (CP phytoplasma subgroup) and that diseased *F. virginiana* and grafted *F. chiloensis* plants were infected by both SM and CP. This is the first report of the CP phytoplasma, subgroup 16SrVI-A, infecting strawberry. This report also indicates that the occurrence of the CP phytoplasma in strawberry may be widespread in North America and that *F. chiloensis*, *F. virginiana*, and *F.* × *ananassa* plants are susceptible to infection by the CP phytoplasma.

References: (1) D. E. Gunderson and I.-M. Lee. Phytopathol. Mediterr. 35:144, 1996. (2) R. Jomantiene et al. HortScience 33:1069, 1998. (3) R. Jomantiene et al. Int. J. Syst. Bacteriol. 48:269, 1998. (4) J. D. Postman et al. Acta Hortic. 471:25, 1998.