Disease Notes

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First Report of *Fusarium oxysporum* **f. sp.** *radicis-cucumerinum* **on Cucumber in Spain.** A. Moreno and A. Alférez, Centro Investigación Agrícola Torre de la Reina, Aventis CropSciences, Sevilla, Spain; M. Avilés, Departamento Ciencias Agroforestales, Universidad de Sevilla, Evita Ctra. Utrera Km. 1 s/n, 41013 Sevilla, Spain; and F. Diánez, R. Blanco, M. Santos, and J. C. Tello, Departamento de Producción Vegetal, Universidad de Almería, Ctra. de San Urbano s/n, 04120 Almería, Spain. Plant Dis. 85:1206, 2001; published on-line as D-2001-0904-01N, 2001. Accepted for publication 17 July 2001.

During December 1999, root and stem rot was observed on greenhouse-grown cucumber (cvs. Albatros, Brunex, Acapulco, and Cerrucho) plants in Almería, Spain, using rock wool cultures. The disease caused severe damage, estimated at a loss of up to 75% of the plants, in the first greenhouse affected; afterward, the disease was found in eight additional greenhouses (14 ha) in 1999 and 2000. Stem lesions extended up to 10 to 12 cm above the crown in mature plants, although no fruit damage was observed. In the advanced stages, abundant development of orange sporodochia was evident on crown and stem lesions, without vascular discoloration. Root, crown, and stem pieces that were placed on potato dextrose agar (PDA) after surface-disinfection with 5% sodium hypochlorite, rinsed, and dried resulted in pure fungal colonies. Based on morphological characteristics of conidia, phialides, and chlamydospores from the isolations, the fungus was identified as Fusarium oxysporum Schlechtend.:Fr. Pathogenicity tests were conducted on cucumber (cvs. Marketmore 76 and Cerrucho [F1 hybrid]), melon (cvs. Amarillo oro, Perlita, Piboule, Tania, and Nipper [F1]), watermelon (cvs. Sugar Baby, Sweet Marvel, Jubilee, and Pata Negra and hybrid Crimson sweet), Cucurbita maxima × Cucurbita moschata, zucchini (cv. Senator), and loofah (Luffa aegyptiaca) at several stages: (i) pregermination; (ii) 1 or 2 true leaves; and (iii) more than 10 true leaves. Five fungal isolates were grown on PDA or shaken potato dextrose broth at 25°C for 8 days. Inoculation was performed in pots (10 seeds or plants of each cultivar or hybrid and isolate) by drenching with 100 ml of a fungal suspension (10⁴ to 10⁶ CFU/ml). Sterile water was applied to noninoculated control plants. Tests were repeated in growth chambers at 25°C (night) and 28°C (day) with a 16-h photoperiod. Fifteen to fifty days after inoculation, cucumber and melon plants at all three stages developed symptoms of root and crown rot in 100% of inoculated plants, with no observed vascular discoloration. Fifty days after inoculation, all three stages of C. maxima × C. moschata and zucchini remained symptomless. Loofah and watermelon germinated poorly or not at all when inoculated at the pregermination stage. Fifteen to fifty days after inoculation, 100% of inoculated cucumber and melon plants developed symptoms. Watermelon plants inoculated at the 10 or more true-leaf stage did not develop disease symptoms. No symptoms developed on noninoculated control plants. F. oxysporum was reisolated from infected roots, crowns, and stems of inoculated plants, confirming Koch's postulates. The main symptoms on cucumber infected by F. oxysporum f. sp. cucumerinum are wilt, yellowing, and vascular discoloration. In contrast, based on inoculation of the host differentials and the resulting disease symptoms found in this study, the fungus was identified as F. oxysporum f. sp. radiciscucumerinum (1). To our knowledge, this is the first report of F. oxysporum f. sp. radicis-cucumerinum causing root and crown rot in cucumber in Spain.

Reference: (1) D. J. Vakalounakis. Plant Dis. 80:313, 1996.

Occurrence and Virulence of *Bipolaris hawaiiensis* on Bermudagrass (*Cynodon dactylon*) on Poultry Waste Application Sites in Mississippi. R. G. Pratt, USDA, ARS, WM&FRU, P.O. Box 5367, Mississippi State 39762. Plant Dis. 85:1206, 2001; published on-line as D-2001-0824-01N, 2001. Accepted for publication 2 August 2001.

Bipolaris hawaiiensis has been reported on bermudagrass (*Cynodon dactylon*) and other *Cynodon* spp. from subtropical areas around the world (2). This pathogen has not previously been reported on bermudagrass in North America (1) nor has its virulence been compared

with that of other Bipolaris spp. on this host. In July and October 1999, frequencies of dematiaceous hyphomycetous pathogens in live but symptomatic leaves of bermudagrass were determined on two poultry waste application sites in Smith and Covington counties, MS, where foliar disease symptoms were widespread. Common bermudagrass was being grazed in Covington County, and cv. Alicia was being grown for hay in Smith County. At each date and site, 100 stems with leaves exhibiting symptoms of chlorosis and necrosis were collected, and a single leaf with well-developed symptoms from each stem was assayed for pathogens by surface-disinfesting, plating on water agar, and observing fungal sporulation. Multiple species of pathogens were detected on most leaves. Identities and mean frequencies of observed pathogen species across both sites and sampling dates were Exserohilum rostratum (62%), Bipolaris cynodontis (98%), Curvularia lunata (28%), C. geniculata (20%), B. spicifera (3%), and B. hawaiiensis (3%). B. hawaiiensis was detected at both sites and on both sampling dates. It was distinguished from *B. cynodontis* by smaller conidia (14 to 28 µm long) and from B. spicifera by more than three pseudosepta per conidium. Virulence of B. hawaiiensis on bermudagrass, compared with B. cynodontis and B. spicifera, was assessed in two identical inoculation experiments using three pathogen-inoculated treatments plus an uninoculated control. In each experiment, foliage of 12-week-old plants in five replicate pots per treatment was sprayed with 4×10^4 conidia per ml of water of each pathogen. The pots were incubated under 12-h plantgrowth lights at 25°C for 3 days in a water-saturated atmosphere to initiate infection and then grown for seven additional days in ambient air under plant-growth lights at 25°C. All three pathogens induced symptoms of chlorosis and necrotic lesions. Symptoms induced by B. hawaiiensis were similar in severity to those produced by B. spicifera and less severe than those produced by *B. cynodontis*. To our knowledge, this is the first report of B. hawaiiensis on bermudagrass in North America. The site in Smith County also apparently represents its northernmost known point of occurrence on this continent (2).

References: (1) D. F. Farr et al. Fungi on Plants and Plant Products in the United States. The American Phytopathological Society, St. Paul, MN, 1989. (2) A. Sivanesan. Graminicolous Species of *Bipolaris, Curvularia, Drechslera, Exserohilum* and their Teleomorphs. Mycol. Pap. No. 158, CAB International Mycological Institute, Wallingford, U.K., 1987.

First Report of Resistance to Benomyl Fungicide in *Sclerotinia sclerotiorum.* B. D. Gossen and S. R. Rimmer, Agriculture and Agri-Food Canada, Saskatoon, SK S7N 0X2; and J. D. Holley, Alberta Agriculture Food and Rural Development, Brooks, AB T1R 1E6. Plant Dis. 85:1206, 2001; published on-line as D-2001-0914-04N, 2001. Accepted for publication 19 August 2001.

Benomyl fungicide (Benlate) is used worldwide to control ascomycete pathogens, but resistance has developed in several pathogen populations (1). On the Canadian prairies, benomyl is used to reduce injury caused by Sclerotinia sclerotiorum (Lib.) de Bary on canola (Brassica napus, B. rapa) and alfalfa (Medicago sativa) seed crops. To determine if populations are resistant to benomyl, isolates of S. sclerotiorum collected from 15 fields (12 alfalfa and 3 canola, one isolate per field) in 2000 were grown on potato dextrose agar amended with benomyl at 0, 0.05, 0.5, 5, 50, and 500 mg/liter. Plugs of mycelium from the margin of an actively growing colony were placed in the center of a 10-cm-diameter petri dish containing 15 ml of test medium and incubated on a laboratory bench. Linear growth (mean of maximum width and right angle) of each colony (three replicates each) was measured after 5 to 6 days. The growth of isolates from 13 fields was inhibited by low concentrations of benomyl $(EC_{50} < 8 \text{ mg/liter})$, but two isolates were very resistant $(EC_{50} > 200 \text{ ms})$ mg/liter). Resistant cultures were isolated from infected canola plants in the only two fields in the study in which reduced efficacy of benomyl was suspected. The distribution and importance of benomyl-resistant populations of S. sclerotiorum in the region remains to be determined.

Reference: (1) T. R. Pettitt et al. Mycol. Res. 97:1172, 1993.

A New Bacterial Leaf Spot Disease of Broccolini, Caused by *Pseudomonas syringae* pathovar *maculicola*, in California. N. A. Cintas and C. T. Bull, U.S. Agricultural Research Station, Salinas, CA 93905; S. T. Koike, University of California Cooperative Extension, Salinas 93901; and H. Bouzar, Sakata Seed, Salinas, CA 93907. Plant Dis. 85:1207, 2001; published on-line as D-2001-0905-01N, 2001. Accepted for publication 5 August 2001.

In 1998, a new disease was detected on 3-week-old commercial broccolini (Brassica oleracea L. var. botrytis × B. alboglabra) transplants in a Salinas Valley, Monterey County, CA greenhouse. Initial symptoms were small (2 to 4 mm diameter) circular to angular, water-soaked spots. As the disease progressed, spots remained relatively small, but turned tan to brown. When diseased tissues were macerated and streaked on King's medium B, a blue-green fluorescent pseudomonad was consistently isolated. Strains were levan positive, oxidase negative, and arginine dihydrolase negative. Strains did not rot potato slices, but induced a hypersensitive reaction on tobacco (Nicotiana tabacum L. 'Turk'). Fatty acid methyl ester analysis (MIS-TSBA, version 4.10, MIDI Inc., Newark, DE) indicated that strains had a high similarity index (0.82 or higher) to Pseudomonas syringae, and GN (version 3.50, Biolog, Inc., Hayward, CA) profiles also identified strains as P. syringae. The bacterium associated with the disease, therefore, was identified as P. syringae van Hall. Pathogenicity was demonstrated by growing inoculum in nutrient broth shake cultures for 48 h, misting the broth cultures (1×10⁶ CFU/ml) onto broccolini (cv. Aspabrock), and subjecting the plants to 48 h of high humidity. Control plants were misted with sterile nutrient broth. After 4 to 5 days in a greenhouse, leaf spot symptoms developed on all inoculated broccolini plants, and reisolated strains were characterized and found to be P. syringae. Control plants remained symptomless. The results of two sets of pathogenicity tests were the same. Repetitive sequence-based polymerase chain reaction using the BOXA1R primer resulted in identical banding patterns for the broccolini pathogen and for known isolates of P. syringae pv. maculicola from crucifers. In host range testing, P. syringae pv. maculicola was pathogenic to broccolini plants. The broccolini isolates and P. syringae pv. maculicola isolates had the same pathogenicity results when crucifers and tomatoes were tested as hosts; broccoli and cauliflower (B. oleracea var. botrytis) were infected, and tomato results were variable. These tests suggest that the broccolini pathogen is the bacterial leaf spot pathogen, Pseudomonas syringae pv. maculicola, that occurs on broccoli and cauliflower transplants (1). To our knowledge, this is the first report of this pathogen causing a disease on commercially grown broccolini.

Reference: (1) S. T. Koike et al. Plant Dis. 82:727, 1998.

First Report of *Erysiphe sedi* **on** *Sedum spectabile* **in North America.** L. Kiss, Plant Protection Institute, Hungarian Academy of Sciences, P.O. Box 102, Budapest, H-1525; and Margery L. Daughtrey, Department of Plant Pathology, Cornell University, Riverhead, NY 11901. Plant Dis. 85:1207, 2001; published on-line as D-2001-0914-01N, 2001. Accepted for publication 16 August 2001.

Since 1997, powdery mildew infections have been repeatedly observed on Sedum spectabile plants, cv. Autumn Joy, grown as ornamentals in commercial greenhouses in New York. Circular patches of gray mycelia appeared and spread on upper and occasionally on lower leaf surfaces followed by necrosis of the leaf tissues and defoliation. The new disease reduced the market value of the infected ornamentals and required chemical control. The pathogen produced conidia singly on 2- to 3-celled conidiophores occurring on the ectophytic hyphae. Conidia were subcylindrical, measured 27 to 36 μ m × 13 to 17 μ m, and contained no fibrosin bodies. Germinating conidia produced a short germ tube, 5 to 30 µm, terminating in a lobed appressorium. Hyphal appressoria were lobed to multi-lobed, opposite or spread along the hyphae. Cleistothecia were not found. Based on conidial characteristics, the pathogen was identified as Erysiphe sedi Braun. To confirm pathogenicity, potted healthy S. spectabile plants were placed near infected plants in the greenhouse. In addition, detached S. spectabile leaves were inoculated with the pathogen by touching them to powdery mildew colonies and then placed in plates filled with one layer of polystyrene balls floated in water. Plates were covered and kept in the laboratory. Uninfected potted plants kept in another greenhouse and noninoculated detached leaves served as controls. After 1 week, powdery mildew appeared on all infected plants and leaves exposed to or inoculated with the pathogen. The pathogen was

morphologically identical to the original fungus. No symptoms were observed on the controls. *E. sedi* is a common Asiatic powdery mildew species infecting many crassulaceous plants (1,2) and was introduced to Eastern Europe from Asia (2). To our knowledge, this is the first report of *E. sedi* in North America.

References: (1) U. Braun. Beih. Nova Hedwigia 89:1, 1987. (2) U. Braun. The Powdery Mildews (Erysiphales) of Europe. Gustav Fisher Verlag, Jena, 1995.

Identification of an Isolate of *Potato virus Y*-Tuber Necrotic Strain on Clary Sage (*Salvia sclarea*). M. G. Bellardi and C. Rubies-Autonell, DiSTA, Istituto di Patologia Vegetale, Università degli Studi, Via F.Re, 8, 40126 Bologna, Italy; and C. Cerato, ISCI, Istituto Sperimentale per le Colture Industriali, Via di Corticella, 133, 40129 Bologna, Italy. Plant Dis. 85:1207, 2001; published on-line as D-2001-0911-01N, 2001. Accepted for publication 1 August 2001.

In 1999, clary sage plants (Salvia sclarea L.) at the Herb Garden of Casola Valsenio (Emilia-Romagna Region, northern Italy) exhibited malformed leaves with yellow spots and line patterns. Sap from leaves of symptomatic sage plants caused symptoms in inoculated Chenopodium amaranticolor Coste et Reyn. plants (local chloro-necrotic lesions developed 7 to 10 days after inoculation) and Nicotiana tabacum L. 'White Burley' and 'Samsun' plants (systemic veinal necrosis developed ≈2 weeks after inoculation). Leaves from symptomatic sage plants tested positive for Potato virus Y (PVY) based on immunoelectron microscopy, gold-labeled decoration, and protein A sandwich enzyme-linked immunosorbent assay (ELISA), using antiserum to PVY (PVAS 50a, American Type Culture Collection, Manassas, VA). Double-antibody sandwich-ELISA, using specific monoclonal antibodies (BioReba AG, Reinach, Switzerland) to the tobacco veinal necrosis strain group of PVY (PVY-N), revealed that the PVY isolate from sage belonged to this group. Immunocapture-reverse transcription-polymerase chain reaction, using specific primers for PVY and the tuber necrotic strain of PVY (PVY-NTN), further classified the sage isolate as PVY-NTN (1). PVY-NTN causes serious damage to potato in Europe. Clary sage, one of the most important aromatic plants cultivated worldwide as a source of essential oils, represents a new natural host of PVY-NTN.

Reference: (1) H. L. Weidemann and E. Maiss. J. Plant Dis. Prot. 103:337, 1996.

First Report of *Sclerotinia sclerotiorum* **on** *Gazania* **sp. Hybrid in Italy.** A. Garibaldi, A. Minuto, G. Gilardi, and M. L. Gullino, DIVAPRA - Patologia Vegetale, Via Leonardo da Vinci 44, 10095 Grugliasco, Italy. Plant Dis. 85:1207, 2001; published on-line as D-2001-0914-03N, 2001. Accepted for publication 16 August 2001.

Gazania sp. hybrid is produced in pots in the Albenga Region of northern Italy for export to central and northern Europe. During fall 2000 to spring 2001, sudden wilt was observed in commercial plantings of this ornamental. Initial symptoms included stem necrosis at the soil level and yellowing and tan discoloration of leaves. As stem necrosis progressed, infected plants wilted and died. Wilt followed by soft rot occurred within a few days on young plants after the first leaf symptoms. Necrotic tissues became covered with white mycelia that produced dark, spherical (2 to 6 mm diameter) sclerotia. Sclerotinia sclerotiorum was consistently recovered from infected stem pieces of Gazania disinfested for 1 min in 1% NaOCl, plated on potato dextrose agar amended with streptomycin sulfate at 100 mg/liter. Pathogenicity of three fungal isolates was confirmed by inoculating 45- to 60-day-old plants grown in containers (14 cm diameter). Inoculum that consisted of wheat kernels infested with mycelium and sclerotia of each isolate was placed on the soil surface around the base of each plant. Noninoculated plants served as controls. All plants were maintained outdoors where temperatures ranged between 8 and 15°C. Inoculated plants developed symptoms of leaf yellowing, followed by wilt, within 7 to 10 days, while control plants remained symptomless. White mycelia and sclerotia developed on infected tissues, and S. sclerotiorum was reisolated from inoculated plants. To our knowledge, this is the first report of wilt of Gazania sp. hybrid caused by S. sclerotiorum in Italy. A crown rot of Gazania caused by S. sclerotiorum has been reported from California in the United States(1).

Reference: (1) V. M. Muir and A. H. McCain. Calif. Plant Pathol. 16:1, 1973.

(Disease Notes continued on next page)

Disease Notes (continued)

First Report of *Fusarium crookwellense* **Causing Tip Blight on Cones of Hop.** S. J. Pethybridge, F. S. Hay, and C. R. Wilson, University of Tasmania, P.O. Box 447, Burnie, 7320, Tasmania, Australia; and L. J. Sherriff and G. W. Leggett, Australian Hop Marketers, GPO Box 104A, Hobart, 7001, Tasmania. Plant Dis. 85:1208, 2001; published on-line as D-2001-0910-01N, 2001. Accepted for publication 10 August 2001.

Hop (Humulus lupulus L.) is grown primarily for the alpha and beta acids produced in the strobile (cone) and used for bittering beer. In late summer (March) 2001, necrotic lesions covering the tips of cones of cvs. Agate, Nugget, and Willamette at hop farms in Tasmania, Australia, were observed. The necrotic lesions encompassed the proximal tips and affected between 5 and 60% of the cone; however, all bracts in the whorl were always affected. Diseased cones were observed in all seven gardens included in the survey. The incidence of plants with cone tip blight in 'Nugget' ranged from 5 to 30% in three gardens, in 'Agate' ranged from 3 to 10% in three gardens, and in the only 'Willamette' garden 30% of cones were affected. Pieces of infected hop cones (N = 55) were surfacetreated for 1 min in 2% sodium hypochlorite, placed on 2% water agar, and incubated at 22 ± 2°C. Fusarium crookwellense Burgess, Nelson, & Toussoun was isolated from 95% of the cones (1). F. crookwellense was identified on carnation leaf agar by L. Burgess, University of Sydney, Australia. Koch's postulates were fulfilled by inoculating detached mature hop cones of cvs. Nugget and Willamette (N = 20 for each cultivar) with an atomized conidial suspension $(3.5 \times 10^5 \text{ spores of a})$ single F. crookwellense isolate per milliliter) until runoff and incubated at $20 \pm 2^{\circ}$ C in a sealed container on plastic mesh over tissue wetted with sterile distilled water. Symptoms first appeared 5 days after inoculation and were identical to those found in the field. No disease symptoms were observed on cones subjected only to sterile distilled water. The pathogen was reisolated from diseased tissue on inoculated cones, completing Koch's postulates. Similar disease symptoms on hop cones have been described in Oregon and were associated with infection by F. sambucinum and F. avenaceum (C. Ocamb, personal communication). To our knowledge, this is the first report of the infection of hop cones by F. crookwellense

Reference: (1) L. W. Burgess et al. Laboratory Manual for Fusarium Research, 3rd ed. University of Sydney, Australia, 1994.

Occurrence of Bacterial Leaf Spot of Escarole Caused by *Pseudomonas cichorii* in the Everglades Agricultural Area of Southern Florida. K. Pernezny and R. N. Raid, University of Florida, Everglades Research and Education Center, P.O. Box 8003, Belle Glade 33430. This research was supported by the Florida Agricultural Experiment Station. Plant Dis. 85:1208, 2001; published on line as D-2001-0912-01N, 2001. Accepted for publication 16 July 2001.

In the fall of 1997, 1998, and 2000, a leaf spot disease of escarole (Cichorium endivia L.) was widespread among commercial plantings in the Everglades Agricultural Area (EAA), south and east of Lake Okeechobee, FL. Symptoms consisted of dry, dark gray-to-black lesions that expanded to ≈ 4 cm in diameter. Concentric rings were often observed in mature lesions. Growers and scouts in the area consistently identified this disease as Alternaria leaf spot, because the symptoms closely resembled Alternaria leaf spots seen on a number of other vegetables. Prolific bacterial streaming occurred when cut portions of lesions were observed microscopically. A fluorescent bacterium was consistently isolated when a sterile inoculation needle was pushed through lesions. Eight bacterial strains were isolated, restreaked to obtain pure cultures, and characterized. All strains were aerobic, gram-negative rods that were oxidase positive and arginine dihydrolase negative. Negative reactions were recorded for levan formation and rotting of potato slices. All strains utilized glucose, mannitol, and m-tartrate and were negative for sucrose, sorbitol, benzoate, D-arabinose, L-rhamnose, and cellobiose. Results for utilization of D-aspartate were variable. Based on these results, the causal agent of bacterial leaf spot of escarole was identified as Pseudomonas cichorii. Greenhouse-grown plants of escarole, cv. Full Heart, and Cos lettuce, cv. Tall Guzmaine, were mistinoculated with a suspension (107 CFU/ml) of each test strain from escarole and P. cichorii strain Pc28, originally isolated from celery (1). Plants were bagged for 3 days after inoculation. Symptoms characteristic of this disease were evident on escarole inoculated with all test strains and Pc28 6 days after inoculation. Pure cultures of P. cichorii were recovered from lesions on King's B medium. Three test strains produced

mild leaf spot symptoms in Cos lettuce, but the symptoms were distinctly different from those associated with the common bacterial leaf spot of lettuce in Florida caused by *Xanthomonas campestris* pv. *vitians* (2). To our knowledge, this is the first report of *P. cichorii* causing this unusual target spot symptom on escarole in the EAA.

References: (1) K. Pernezny et al. Plant Dis. 78:917, 1994. (2) K. Pernezny et al. Plant Dis. 79:359, 1995.

First Report of *Tomato chlorosis virus* **in Italy.** G. P. Accotto, A. M. Vaira, and M. Vecchiati, Istituto di Fitovirologia Applicata, CNR, Strada delle Cacce 73, 10135 Torino, Italy; M. M. Finetti Sialer and D. Gallitelli, Dip. Protezione Piante e Microbiologia Applicata, Università degli Studi, Via Amendola 165/A, 70126 Bari, Italy; and M. Davino, Dip. Scienze e Tecnologie Fitosanitarie, Università degli Studi di Catania, Via Valdisavoia 5, 95123 Catania, Italy. Plant Dis. 85:1208, 2001; published on-line as D-2001-0918-01N, 2001. Accepted for publication 25 August 2001.

During winter 2000-2001, an unusual disease of tomato was observed in some greenhouses in Sardinia, Sicily, and Apulia, in southern Italy. Plants were chlorotic and reduced in size, expanded leaves showed interveinal yellowing, and older leaves developed interveinal reddishbronze necrosis and downward rolling. The symptoms resembled those recently reported from Portugal (1) as induced by Tomato chlorosis virus (ToCV) (family Closteroviridae, genus Crinivirus), a whiteflytransmitted virus new to Europe. Symptomatic leaf tissues were extracted and analyzed by reverse transcription-polymerase chain reaction as described by Louro et al.(1). The 439-bp ToCV-specific DNA fragment was amplified in samples collected from 6 of 14 greenhouses in Sardinia, 2 of 5 greenhouses in Sicily, and 1 of 1 greenhouse in Apulia. The sequence of the fragment obtained from a Sicilian isolate (GenBank Accession No. AY048854) showed more than 99% identity to ToCV isolates (Accession Nos. AF024630 and AF234029) from the United States and Portugal, respectively. Infestations of Trialeurodes vaporariorum and Bemisia tabaci have been reported in autumn. To our knowledge, this is the first report of ToCV in Italy. Although we found the virus in three regions of the country, its distribution is likely to be wider, since the symptoms can be mistaken for those of a physiological disorder or of Tomato infectious chlorosis virus, another crinivirus infecting tomato.

Reference: (1) Louro et al. Eur. J. Plant Pathol. 106:589, 2000.

Detection of *Poinsettia mosaic virus* Infecting Poinsettias (*Euphorbia pulcherrima*) in Venezuela. O. Carballo, M. L. Izaguirre, and E. Marys, IVIC, Centro de Microbiología y Biología Celular, Laboratorio de Virus de Plantas, Apdo Postal 21827, Caracas 1020A, Venezuela. Plant Dis. 85:1208, 2001; published on-line as D-2001-0920-02N, 2001. Accepted for publication 25 August 2001.

Poinsettia mosaic virus (PnMV), a putative member of the tymoviruses, was detected in several cultivars of vegetatively propagated poinsettias grown in commercial nurseries in Estado Miranda, Venezuela. Symptoms associated with the affected plants consisted of severe mottling and distortion of leaves and bracteoles. The suspect virus was mechanically transmitted to Nicotiana benthamiana. Leaf extracts and thin sections of affected leaf tissue were analyzed by transmission electron microscopy. Spherical virus particles (30 nm diameter) were observed in samples from symptomatic poinsettia plants. Ultrastructural analyses of virus-infected cells revealed aggregates of virus particles in the cytoplasm and central vacuole. The virus was purified twice from infected N. benthamiana, resulting in yields as high as 12 mg/100 g. Dissociated coat protein contained a single 24-kDa protein species. The virus was not serologically related to Carnation mottle, Bean rugose mosaic, Cowpea mosaic, Cucumber mosaic, Pea enation mosaic, Prunus necrotic ringspot, Apple mosaic, Tobacco streak, Maize rayado fino, Tomato ringspot, Bean southern mosaic, Sowbane mosaic, Andean potato latent, Belladona mottle, Scrophularia or Turnip yellow mosaic viruses, but did react positively in enzymelinked immunosorbent assay and western blot analysis with antiserum (ATCC PVAS-476) to PnMV. Based on these results, the virus is considered to be PnMV. To our knowledge, this is the first report of PnMV infecting poinsettias in Venezuela.

Three Previously Unidentified Begomoviral Genotypes from Tomato Exhibiting Leaf Curl Disease Symptoms from Central Sudan. A. M. Idris and J. K. Brown, Department of Plant Sciences, University of Arizona, Tucson 85721. Plant Dis. 85:1209, 2001; published on-line as D-2001-0920-03N, 2001. Accepted for publication 14 August 2001.

Field tomato plants exhibiting upward curling of leaflets, chlorosis, and stunting symptoms described for tomato leaf curl disease in Sudan (2) were collected in 1996 from Gezira (GZ) and Shambat (SH), Sudan. Disease symptoms were reproduced following experimental transmission of the causal agent(s) by the whitefly Bemisia tabaci from field tomato to virus-free tomato seedlings in a glasshouse at Gezira Research Station, Wad Medani, Sudan. Total nucleic acids were extracted from symptomatic tomato test plants. An ≈1.3-kbp fragment, diagnostic for begomovirus, was obtained from extracts by polymerase chain reaction using degenerate primers that amplify the coat protein gene (CP) and the respective flanking sequences for most begomoviuses (1). A second pair of degenerate primers was used to amplify a 2.3-kbp begomoviral fragment that overlaps both ends of the (CP) amplicon by >200 nt (1). At least 10 amplicons for each were cloned, and their sequences were determined, revealing three unique, tomato-infecting begomoviruses genotypes, two from GZ and one from SH. No B component was detected using degenerate primers that direct the amplification of a diagnostic fragment of the B component (1.4 kbp) for most bipartite begomoviruses. The organization of the three, apparently full-length viral genomes, was typical of other monopartite begomoviruses. A GenBank search revealed that the three viruses were previously undescribed. The GZ and SH tomato isolates are herein provisionally named ToLCV-GZ1 (GenBank Accession No. AY044137), ToLCV-GZ2 (GenBank Accession No. AY044138), and ToLCV-SH (GenBank Accession No. AY044139), respectively. All three tomato-infecting begomoviruses have identical stem-loop structures containing the conserved nonanucleotide motif characteristic of all members of the family Geminiviridae; however, the predicted Rep binding element located in the common region is unique for each virus. Phylogenetic analysis of the three viral sequences placed them in a large clade containing all other Old World begomoviruses. Distance comparisons among these and other well-studied begomoviruses indicated that ToLCV-GZ1 and ToLCV-SH shared an overall 90% nucleotide sequence identity, with ~83% nucleotide sequence identity to ToLCV-GZ2. ToLCV-GZ1 and ToLCV-SH were 83% identical, with their closest relative, Tomato yellow leaf curl virus (TYLCV), while ToLCV-GZ2 shared 93% identity with TYLCV. The genomes of all three Sudan viruses contained regions of homologous nucleotide sequences, suggesting intermolecular exchange among these viruses. Exclusion of the homologous sequences (>800 nt) from the phylogenetic analysis indicated even lower shared nucleotide identities (<90%, the arbitrary cut-off for distinct species), which may warrant their classification as separate species. These three newly described begomoviruses are indigenous to central Sudan, and comprise a unique Old World lineage distinct from previously described begomoviruses associated with leaf curl disease of tomato in Africa and the Mediterranean Region.

References: (1) A. M. Idris and J. K. Brown. Phytopathology 83:548, 1998. (2) A. M. Yassin. Trop. Pest Manage. 29:253, 1983.

Report of Leaf Spot of Spinach Caused by *Stemphylium botryosum* in **Maryland and Delaware.** K. L. Everts and D. K. Armentrout, University of Maryland Lower Eastern Shore Research and Education Center, Salisbury 21801. Plant Dis. 85:1209, 2001; published on-line as D-2001-0917-01N, 2001. Accepted for publication 16 August 2001.

In October 2000, leaf spot symptoms were observed on spinach (*Spinacia oleracea* L. 'Seven R') at the University of Maryland Lower Eastern Shore Research and Education Center in Salisbury. In April 2001, a similar leaf spot disease was observed in two commercial spinach fields (cv. Vancouver) in Dorchester County, MD, and Sussex County, DE. Symptomatic plants occurred in foci, and overall disease incidence in the research and commercial fields was <10% of plants with lesions. However, low disease incidence may reduce the value of a spinach rop by requiring additional hand-sorting (fresh market) or lowering the grade (processing). Leaf spot lesions were small (0.2 to 0.7 cm), circular, tan, and papery and lacked visual signs of fungal infestation. Lesions resembled a new leaf spot of spinach reported in California (1) caused by *Stemphylium botryosum* Wallr. Plating surface-disinfested lesion margins on 0.25-strength potato dextrose agar consistently yielded *S. botryosum*.

Single conidial cultures of three isolates were grown on V8 agar in a growth chamber with a 12 h light/dark regime at 21°C and were used for the pathogenicity test. Conidia were collected from 7-day-old colonies to test pathogenicity. Conidia were suspended in distilled water (1.1×10^5) conidia per milliliter), and sprayed on 4-week-old spinach plants (with four to six true leaves) of cvs. Seven R, Vancouver, and Melody. Noninoculated control plants were sprayed with deionized water. Plants were incubated for 72 h in a dew chamber (18°C, 9 to 15 h light/dark regime where dew formed during the dark periods) and then placed on a greenhouse bench (23°C) for 2 weeks. Plants that had been inoculated with any of the three isolates developed the aforementioned leaf spot lesions after 4 days in the greenhouse. Plants sprayed with deionized water were symptomless. One week after inoculation, more lesions were observed on 'Seven R' and 'Vancouver' than on 'Melody' (41, 39, and 1 lesion per plant, respectively; P < 0.0030), and the lesions were 1.5, 1.2, and 0.5 mm in diameter, respectively (P < 0.0001). S. botryosum was consistently reisolated from leaf spot lesions. The pathogenicity test was repeated with similar results. Isolates grown on V8 agar and incubated for ≈ 10 days produced conidia with mean dimensions of 31×19 µm. To our knowledge, this is the first report of leaf spot of spinach caused by S. botryosum in Maryland and Delaware.

Reference: (1) S. T. Koike et al. Plant Dis. 85:126, 2001.

First Report of Natural Infection by "*Candidatus* Phytoplasma brasiliense" in *Catharanthus roseus*. H. G. Montano, USDA-ARS, Molecular Plant Pathology Laboratory, Beltsville, MD 20705, and UFRRJ/IB, Departamento de Entomologia e Fitopatologia, Seropédica, RJ, Brazil; E. L. Dally and R. E. Davis, USDA-ARS, Molecular Plant Pathology Laboratory, Beltsville, MD 20705; J. P. Pimentel, and P. S. T. Brioso, UFRRJ/IB, Departamento de Entomologia e Fitopatologia, RJ, Brazil. Plant Dis. 85:1209, 2001; published on-line as D-2001-0914-02N, 2001. Accepted for publication 3 August 2001.

Catharanthus roseus (L.) G. Don (periwinkle) is well known as an experimental host for diverse phytoplasmas that are artificially transmitted to it through the use of dodder (Cuscuta sp.), laboratory vector insects, or grafting. However, few phytoplasma taxa have been reported in natural infections of C. roseus, and the role of C. roseus in phytoplasma dissemination and natural disease spread is not clear. In this study, naturally diseased plants of C. roseus exhibiting yellowing and witches' broom symptoms indicative of phytoplasma infection were observed throughout the year in the state of Rio de Janeiro, Brazil. Shoots and leaves of four diseased plants were assayed for the presence of phytoplasma DNA sequences by nested polymerase chain reactions (PCR) as previously described (2,3). Phytoplasma rDNA was amplified from diseased periwinkle plants in PCR primed by primer pair P1/P7 and was reamplified in nested PCR primed by primer pair R16F2n/R16R2 (F2n/R2). The results indicated the presence of phytoplasma in all four diseased plants. Phytoplasma identification was accomplished by restriction fragment length polymorphism (RFLP) analysis, using 11 restriction enzymes, of 16S rDNA amplified in PCR primed by F2n/R2. Phytoplasmas were classified according to the system of Lee et al. (1). On the basis of collective RFLP patterns of 16S rDNA, the phytoplasma infections in the four periwinkle plants could not be distinguished from one another. Furthermore, the collective RFLP patterns were indistinguishable from those reported previously for hibiscus witches' broom phytoplasma, "Candidatus Phytoplasma brasiliense" (2). The phytoplasma found in C. roseus, designated strain HibWB-Cr, was classified in group 16SrXV (hibiscus witches' broom phytoplasma group). HibWB-Cr is tentatively considered a new strain of "Ca. P. brasiliense". C. roseus is the first known, naturally diseased alternate plant host of "Ca. P. brasiliense". The present study identified strain HibWB-Cr in Rio de Janeiro State, where hibiscus witches' broom disease is prevalent (2). How this economically important disease of hibiscus spreads is not known. Our findings raise the possibility that a polyphagous insect vector is involved in the natural transmission of "Ca. P. brasiliense" and that C. roseus or other plant species serve as reservoirs for the spread of this phytoplasma taxon.

References: (1) I.-M. Lee et al. Int. J. Syst. Bacteriol. 48:1153, 1998. (2) H. G. Montano et al. Int. J. Syst. Evol. Microbiol. 51:1109, 2001. (3) H. G. Montano et al. Plant Dis. 84:429, 1999.

(*Disease Notes* continued on next page)

Disease Notes (continued)

First Report of *Rhizopycnis vagum* **Associated with Tomato Roots in Italy.** A. Porta-Puglia, N. Pucci, G. Di Giambattista, and A. Infantino, Istituto Sperimentale per la Patologia Vegetale, Via G.G. Bertero, 22, I-00156, Rome, Italy. Research funded by MiPA, National Program 'Orticoltura'. Plant Dis. 85:1210, 2001; published on-line as D-2001-0917-03N, 2001. Accepted for publication 22 August 2001.

Field surveys were made in several central and southern Italian tomato-growing areas for Pyrenochaeta lycopersici, the cause of corky root of tomato. In addition to P. lycopersici, a different fungus was frequently isolated from roots showing typical corky root symptoms, even after disinfestation of diseased roots with 0.1% (vol/wt) mercury chloride water solution for 1 min. The fungus was isolated from primary and secondary tomato roots in 8 of 21 fields visited. The isolates were grown on potato dextrose agar (PDA), with morphological features such as color and shape of mature conidia and pycnidia, type of conidiogenesis, presence of microsclerotia, and color of colony underside noted. Preliminary identification of the fungus was Rhizopycnis vagum Farr. To confirm the identification, the internal transcribed spacer (ITS) region of rDNA of one isolate (maintained at the ISPaVe collection at the authors' address and available on request as isolate ER 940) was amplified with two universal primers, ITS5 and ITS4. The ITS fragment was sequenced, and the nucleotide sequence compared with that of R. vagum deposited in GenBank (Accession No. AF022786). Both sequences were identical supporting the identification. R. vagum is a recently described species associated with the vine decline syndrome of melon in the United States, Guatemala, Honduras (2), and Spain (3). Eight isolates were tested for pathogenicity both on tomato (five cultivars) and melon (three cultivars) using two methods. In method 1, plantlets at the cotyledonary stage were grown on blotter in petri dishes and tested by placing a 6-mm plug of colonized PDA on the tap root (1). After 7 days, the plug was removed, and the roots were checked for symptoms. In method 2, 20-day-old seedlings were transferred to pots with infested soil (50,000 CFU/g of soil) and grown for 45 days before the roots were checked for each isolate-cultivar combination. Eight and four plants were used in tests 1 and 2, respectively. With the first method, rotten, pinkish lesions with different extensions from the inoculation point were observed on all the melon cultivars tested (Pamir, Cantalupo di Charentais, and Charme). On tomato, three of eight isolates caused root necrosis of limited extent, without pinkish discolorations at the inoculation site on cvs. Monalbo and Bonnie Best, the former showing the larger lesions. The tests on plants grown in infested soil confirmed pathogenicity on both host species, although the symptoms were of minor intensity (light, small brown lesions on secondary roots, no pinkish discoloration). The symptomatic plantlets ranged from 0 to 100% on both hosts in the petri dish tests and from 0 to 100% and 0 to 50%, respectively, for tomato and melon in the pot tests, varying according to the cultivar-isolate combination. The fungus was consistently reisolated from all symptomatic plants. To our knowledge, this is the first report of R. vagum associated with tomato roots. Although the isolates showed varying degrees of virulence with respect to host species (all being pathogenic at least on one host), the virulence of R. vagum on tomato was certainly low. Nevertheless, tomato may maintain or possibly increase inoculum for melon, which often follows tomato in Italian crop rotations.

References: (1) M. Clerjeau and M. Conus. Annu. Rev. Phytopatol. 5:143, 1973. (2) D. F. Farr et al. Mycologia 90:290, 1998. (3) J. García-Jiménez et al. EPPO Bull. 30:169, 2000.

First Report of the Prevalence of Benzimidazole-Resistant Isolates in a Population of *Cylindrocladium pauciramosum* **in Italy.** G. Polizzi, and A. Vitale, Dipartimento di Scienze e Tecnologie Fitosanitarie, University of Catania, Via Valdisavoia 5, 95123 Catania, Italy. Plant Dis. 85:1210, 2001; published on-line as D-2001-0828-01N, 2001. Accepted for publication 5 August 2001.

Cylindrocladium pauciramosum C.L. Schoch & Crous (teleomorph Calonectria pauciramosa C.L. Schoch & Crous), described as a member of the Cylindrocladium candelabrum Viégas complex (4), was recently reported from Europe (3). In southern Italy, the fungus has caused

extensive losses, and chemical control measures are necessary, especially in ornamental nurseries. Several researchers have found benzimidazole fungicides to be effective for control of different species of Cylindrocladium, however, in fungicide trials conducted on myrtle plants infected by C. pauciramosum, benomyl was ineffective (2). Another study showed that mycelial growth of six isolates was completely inhibited by carbendazim at a concentration of 1 µg a.i./ml whereas, concentrations of 10, 100, and 500 µg a.i./ml did not completely inhibit growth of four isolates (1). To examine benzimidazole resistance in C. pauciramosum, 200 single-conidia isolates were tested. These were collected during 1996 and 1997 from several symptomatic hosts in different nurseries located in Sicily and Calabria and identified through morphological characteristics as well as mating-type studies with tester strains. Sensitivity to benomyl was determined by plating mycelial plugs on potato dextrose agar (PDA) amended with benomyl at 1, 10, 100, and 500 µg a.i./ml. For 20 benomyl-resistant isolates, fungal growth was also determined at the same concentrations on carbendazim-amended PDA. Sensitivity was expressed as the minimum inhibitory concentration (MIC) (the lowest fungicide concentration that completely prevented fungal growth). Isolates that did not grow on benzimidazole-amended PDA were classed as sensitive. Isolates were considered resistant to benzimidazole if MIC values were greater than 1 µg a.i./ml. Of the 200 isolates tested, 58% were resistant to benomyl. The benomyl-resistant isolates tested for carbendazim sensitivity were cross-resistant to carbendazim. Most resistant isolates grew in the presence of benomyl at 500 µg a.i./ml. On agar culture, the isolates were either the fast-growing or slow-growing type. The slow-growing phenotype appears to be related to the higher level of resistance (500 µg a.i./ml). On the basis of these data, the use of benzimidazole for the control of this pathogen should be seriously questioned. To our knowledge, this is the first report of the prevalence of benzimidazole resistance in a population of C. pauciramosum.

References: (1) G. Polizzi. Inf. Fitopatol. 11:39, 2000. (2) G. Polizzi and A. Azzaro. Petria 6:117, 1996. (3) G. Polizzi and P. W. Crous. Eur. J. Plant Pathol. 105:407, 1999. (4) C. L. Schoch et al. Mycologia 91:286, 1999.

First Report of *Parietaria mottle virus* on Tomato in Spain. J. Aramburu, Institut de Recerca y Tecnología Agroalimentaria, Departamento de Protección Vegetal, Crta. de Cabrils s/n, 08348 Cabrils, Barcelona, Spain. Plant Dis. 85:1210, 2001; published on-line as D-2001-0919-01N, 2001. Accepted for publication 30 July 2001.

During spring 2001, plants of different tomato (Lycopersicon esculentum) cultivars grown in several commercial fields in the eastern Catalonia Region of Spain had fruit with brown patches and young leaves with rings and a bright necrotic mosaic that progressed to stem necrosis of the apex, which might die and later develop new symptomless shoots. The symptoms were similar to those of Cucumber mosaic virus (CMV) and Tomato spotted wilt virus (TSWV). Sap of tomato sample R1 (in buffered saline [0.02 M sodium phosphate, 0.15 M NaCl at pH 7.2, containing 0.2% 2-mercaptoethanol]) was infective to Cucumis sativus (local necrosis), tomato cv. Marmande (systemic infection consisting of chlorotic local lesions and necrotic mosaic), Nicotiana clevelandii and N. benthamiana (chlorosis and rosetting), and Chenopodium quinoa (chlorotic local lesions, systemic mottle, and leaf distortion). The sap was not infective to N. glutinosa, N. tabacum cv. Xanthi, Datura stramonium, or Gomphrena globosa. The host range data indicated that the infective agent in sample R1 could be Parietaria mottle virus (PMoV) (1). Symptomatic plants inoculated in a greenhouse with the R1 isolate and symptomatic from tomato plants from the field were analyzed by indirect enzyme-linked immunosorbent assay (ELISA) and had minimum ELISA values at least 10-fold higher than healthy controls, using a polyclonal antiserum (provided by P. Roggero) of a tomato strain of PMoV denoted tomato virus 1 (2). The R1 isolate of PMoV was negative in ELISA when analyzed with commercial antisera to TSWV, CMV, Tomato mosaic virus, Tomato bushy stunt virus, Potato Y virus, Tobacco etch virus, Pelargonium zonate spot virus, and Tobacco streak virus.

References: (1) P. Caciagli et al. Plant Pathol. 38:577, 1989. (2) P. Roggero et al. J. Plant Pathol. 82:159, 2000.

Occurrence of *Diaporthe phaseolorum* var. *meridionalis* on Soybean in Illinois. C. E. Gravert and S. Li, Department of Crop Sciences, University of Illinois; and G. L. Hartman, USDA-ARS, Department of Crop Sciences, University of Illinois, 1101 W. Peabody Dr., Urbana 61801. Plant Dis. 85:1211, 2001; published on-line as D-2001-0918-02N, 2001. Accepted for publication 13 August 2001.

Both Diaporthe phaseolorum var. caulivora and D. phaseolorum var. meridionalis cause stem canker on soybean, with D. phaseolorum var. caulivora reported in the northern regions and D. phaseolorum var. meridionalis reported in the southern regions of the United States (1). During the 1999 and 2000 growing seasons, fungi were isolated from soybean plants from growers' fields exhibiting stem canker symptoms. Stem tissue along the margin of the canker was cut into 1- to 5-mm³ pieces, surface-disinfected for 4 min in 0.5% NaOCl solution, rinsed twice, and plated on water or potato dextrose agar (PDA). Fungi of interest were hyphal tipped, grown on PDA at 21°C with 24 h of light, and identified by culture and spore morphology after 3 to 4 weeks. Typical D. phaseolorum var. meridionalis isolates produced white, lanose colonies that turned tan with age. Most of the D. phaseolorum var. meridionalis isolates produced pycnidia with alpha spores and beaked perithecia after 25 to 30 days (2). Brown to black stromata formed in irregular shapes. Of the 16 D. phaseolorum var. meridionalis isolates identified, 11 were from Illinois, 1 each from Indiana and Ohio, and 3 from Kentucky. In Illinois, four isolates were from the northern part of the state, and the rest were from the central and southern areas of the state. In addition to D. phaseolorum var. meridionalis, other isolates obtained from soybean plants included D. phaseolorum var. caulivora, D. phaseolorum var. sojae, and Phomopsis longicolla.

References: (1) J. A. McGee and D. C. Biddle. Plant Dis. 71:620, 1987. (2) A. W. Zhang et al. Phytopathology 88:1306, 1998.

First Occurrence of Panama Disease in Two Banana-Growing Areas of South Africa. E. J. Grimbeek and A. Viljoen, Department of Microbiology and Plant Pathology, Forestry and Agricultural Biotechnology Institute, University of Pretoria, Pretoria, South Africa; and S. Bentley, Cooperative Research Centre for Tropical Plant Protection, Indooroopilly Research Centre, Plant Pathology Building, 80 Meiers Road, Indooroopilly, Brisbane, Queensland, 4058, Australia. Plant Dis. 85:1211, 2001; published on-line as D-2001-0920-01N, 2001. Accepted for publication 30 August 2001.

Fusarium wilt (Panama disease) of bananas is well established in two of the five banana production regions in South Africa: Kiepersol (near Hazyview) and southern KwaZulu-Natal. The disease is caused by a soilborne fungus, Fusarium oxysporum Schlechtend.: Fr. f. sp. cubense (E.F. Sm.) W.C. Snyd. & H.N. Hans., which is most commonly introduced into an area by infected plant material or in contaminated soil attached to vehicles, farm machinery, or footwear. In September 2000, banana plants were observed dying at an experimental site in a commercial Cavendish plantation in the Tzaneen area of the Northern Province of South Africa. Symptoms included wilting of leaves (starting from the oldest foliage) and yellow-brown discoloration of vascular tissue in the rhizome and pseudostem. These symptoms are typical of those described for Panama disease of bananas (2). Similar symptoms were observed at another experimental site in a banana plantation in the Komatipoort region of the Mpumalanga Province in November 2000. Komatipoort is currently the largest banana production region in South Africa. Panama disease has not previously been reported in the Tzaneen and Komatipoort regions. Both are at least 200 km away from the other banana production areas in South Africa. Fungal isolations were made from four diseased plants in both Tzaneen and Komatipoort, and the discolored tissue of the pseudostem was placed on potato dextrose agar amended with novobiocin (0.2 g/liter). Single-spore cultures made from developing colonies were identified as F. oxysporum based on morphological characteristics. Isolates were sent to the Cooperative Research Centre for Tropical Plant Protection (CRCTPP) in Australia for identification by means of DNA amplification fingerprinting (DAF)

analysis (1). Based on DAF analysis, isolates from Tzaneen and Komatipoort were identical to those in vegetative compatibility group 0120 of F. oxysporum f. sp. cubense ("subtropical" race 4), the causal agent of Panama disease in Kiepersol and southern KwaZulu-Natal. Pathogenicity studies were performed in the greenhouse by inoculating 5cm Cavendish banana plants with two isolates of F. oxysporum f. sp. cubense from Tzaneen and two isolates from Komatipoort. Ten plants per isolate were inoculated by submerging their roots in a suspension of microconidia (10⁵ spores/ml). Roots of control plants were submerged in sterile distilled water. Within 6 weeks, wilting symptoms developed on the lower leaves of inoculated banana plants, and the central cylinder of the rhizomes turned reddish brown. F. oxysporum f. sp. cubense was reisolated from the diseased tissue to complete Koch's postulates. The outbreaks of Panama disease in Komatipoort and Tzaneen do not appear to have spread further. Both of the infected fields were placed under quarantine, and symptomatic plants were destroyed.

References: (1) S. Bentley et al. Phytopathology 88:1283, 1998. (2) R. H. Stover. Fusarial Wilt (Panama Disease) of Bananas and Other Musa Species. CMI, Kew, Surrey, UK, 1962.

First Report of *Frankliniella fusca* **as a Vector of** *Impatiens necrotic spot tospovirus.* R. A. Naidu, C. M. Deom, and J. L. Sherwood, Department of Plant Pathology, University of Georgia, Athens 30602. Plant Dis. 85:1211, 2001; published on-line as D-2001-0917-02N, 2001. Accepted for publication 24 August 2001.

Of more than a dozen members of the genus Tospovirus, Tomato spotted wilt virus (TSWV) and Impatiens necrotic spot virus (INSV) are among the most damaging viruses found in North America (3). TSWV is a major problem in vegetable and field crops, whereas INSV is commonly encountered in the floriculture and nursery industries. TSWV is transmitted by several thrips species, of which the western flower thrips (WFT, Frankliniella occidentalis Pergande) is the most predominant vector. INSV has been reported to be transmitted only by WFT (1). To determine if tobacco thrips (TT, F. fusca Hinds) can transmit INSV, a virus-free culture of TT was reared on detached peanut cv. Florunner leaves in 0.5-liter polypropylene cups with closed lids at 25 \pm 2°C with constant light. Fresh peanut leaves were exchanged every 2 to 3 days to maintain the thrips colony. For transmission studies, adult thrips were confined on peanut leaves for 24 h for oviposition and then the peanut leaves, sans adults thrips, were transferred to a new cup. Leaves were examined daily for larval emergence, and similarly aged first instar larvae (<12 h old) were given an acquisition access period of 24 to 48 h on INSV-infected detached leaves of Emilia sonchifolia. The larvae were subsequently transferred to healthy peanut leaves and reared until adult emergence. Groups of 10 adults per plant were given a 48-h inoculation access period on 10-day-old healthy E. sonchifolia seedlings. Thrips were subsequently killed, and the plants were maintained in a growth chamber at $28 \pm 2^{\circ}$ C, and with a 16/8 light/dark photoperiod. Transmission studies were repeated 10 times with different sources of infected plants and different batches of larvae following acquisition access periods. Seven to ten days after inoculation, plants developed symptoms consisting of chlorotic spots, mosaic, and mottling. The presence of INSV in these symptomatic plants was confirmed by ELISA using INSV ImmunoStrip Test (Agdia, Inc., Elkhart, IN) and by reverse transcription-polymerase chain reaction assay with primers specific to the INSV-NSs gene. Our results demonstrate that TT can serve as a vector of INSV. INSV has been reported in peanut in the southeastern United States (2). WFT and TT transmit TSWV in peanuts, with the latter being the predominant vector species in Georgia and other parts of the region. TT transmission of INSV is of concern because of the increased incidence in recent years of INSV in peanuts and the potential for synergistic or gene exchange between TSWV and INSV, since mixed infections with both viruses have been observed (4).

References: (1) M. L. Daughtrey et al. Plant Dis. 81:1220, 1997. (2) S. S. Pappu et al. Plant Dis. 83:966, 1999. (3). J. L. Sherwood et al. Pages 1034-1040 in: Encyclopedia of Plant Pathology. C. Maloy and T. D. Murray, eds. John Wiley and Sons, Inc., New York, 2001. (4) L. Wells et al. Phytopathology (Abstr.) 94:S94, 2001.