Disease Notes

Current Status and Newly Discovered Natural Hosts of *Tomato infectious chlorosis virus* and *Tomato chlorosis virus* in Spain. M. I. Font, Departamento de Ecosistemas Agroforestales, Universidad Politécnica de Valencia, Cno. de Vera s/n, 46020 Valencia, Spain; M. Juárez, Departamento de Producción Vegetal y Microbiología, Universidad Miguel Hernández, Carretera de Beniel km 3.2, 03312-Orihuela-Valencia, Spain; and O. Martínez and C. Jordá, Departamento de Ecosistemas Agroforestales, Universidad Politécnica de Valencia, Cno. de Vera s/n, 46020 Valencia, Spain. Plant Dis. 88:82, 2004; published on-line as D-2003-1024-01N, 2004. Accepted for publication 24 September 2003.

Tomato chlorosis virus (ToCV) and *Tomato infectious chlorosis virus* (TICV) are emergent whitefly-transmitted criniviruses. ToCV and TICV were detected in Spain in 2000 (2) and 2001 (1), respectively. Both viruses infect tomato (*Lycopersicon esculentum* Mill) crops and cause symptoms of foliar chlorosis. ToCV is prevalent along the southern and eastern regions of Spain (provinces of Sevilla, Málaga, Almería, Murcia, Alicante, and Castellón), Balearic (Mallorca), and the Canary Islands (Tenerife and Gran Canaria). However, TICV only has been detected in the provinces of Murcia, Alicante, and Castellón in Spain. During the summer and autumn of 2002, abnormal interveinal reddening, yellowing symptoms, or both, were observed in plants of *Chenopodium album* L., *C. murale* L., and *Solanum nigrum* L. growing in or around tomato fields in Murcia and Almería provinces. To study the alternative hosts that may serve as virus reservoirs in areas where these viruses are prevalent, 62 samples of 42 common weed species were analyzed by reverse transcription-polymerase chain reaction using specific primers for ToCV and TICV (1). The 439-bp ToCV-specific DNA fragment was amplified in two *S. nigrum* samples from Alicante and Murcia provinces, and the 501-bp TICV-specific DNA fragment was amplified in one *C. murale* sample from Murcia, as well as in three *C. album* samples from Murcia and Alicante provinces. The DNA fragment amplified from the ToCV isolate was sequenced and showed 99 to 98% identity with the ToCV isolates (GenBank Accession Nos. AY048854 and AF234029) from Italy and Portugal, respectively. The DNA fragment amplified from TICV isolates were sequenced and showed 98% identity with the TICV isolate from Spain (GenBank Accession No. AF479662), confirming the diagnosis. Although the number of samples is not sufficient to conclude that we know, in a precise way, the role of weed reservoirs in TICV and ToCV epidemics in Spain, this study might contribute to a better understanding of the epidemiology of these viruses. To our knowledge this is the first report of these weeds as natural hosts of ToCV and TICV in Spain.

References: (1) M. I. Font et al. Plant Dis. 86:696, 2002. (2) J. Navas-Castillo et al. Plant Dis. 84:835, 2000.

First Report of Alternaria Leaf Blight of *Aralia japonica* Caused by *Alternaria panax* in Europe. A. Garibaldi, G. Gilardi, and M. L. Gullino. DIVAPRA—Patologia vegetale, Via Leonardo da Vinci 44, 10095 Grugliasco, Italy. Plant Dis. 88:82, 2004; published on-line as D-2003-1031-01N, 2004. Accepted for publication 3 October 2003.

Aralia japonica (synonym *Fatsia japonica*), belonging to the Araliaceae family, is a foliage plant highly valued in Italy for landscape and interior decoration. In the fall of 2002, a leaf blight disease was observed on plants grown in pots that were maintained under shade at a density of 15 to 20 pots per m^2 at a nursery located in central Italy (Teramo Province). Typical symptoms were tan-to-dark brown leaf spots and rapid blighting of foliage under moist conditions. Chlorotic zones around necrotic lesions were common, and considerable leaf drop was associated with the disease. Affected plants were rarely killed, but the presence of lesions on mature plants reduced aesthetic quality and market value. The disease occurred on 70% of the plants. A fungus identified morphologically as *Alternaria panax* (2) was consistently isolated from infected leaves on potato dextrose agar (PDA). The fungus grows slowly and sparsely on PDA and produces a light brown mycelium, a characteristic red diffusible pigment in the agar medium, and rare conidia under 12-hr photoperiods. Measurements were carried out on conidia formed from single-spore isolates grown on autoclavated host tissue on water agar (LWA) at 24°C for 10 days. In LWA culture, conidia were borne singly or in chains of two to four conidia. Conidia produced in culture were smaller than those formed on the host and were highly variable in shape. They appeared obclavate, ellipsoidal, and obpyriform and pale to dark brown with relatively short or false beaks. Conidial bodies were 14.4 to 48.0 µm long (average 30.5 µm) and 7.2 to 12.0 µm wide (average 9.9 µm) with 3 to 10 transverse and a few longitudinal septa. Length of appendages was 9.6 to 26.0 µm (average 16.0 µm). Pathogenicity tests were performed by inoculating leaves of healthy *Aralia japonica* and *Schefflera actinophylla* plants by placing mycelial disks (5 mm in diameter) directly on wounded leaf tissues. Uninoculated, wounded plants served as controls. Four plants of each species were used. Plants were covered for 72 h with plastic bags and maintained in a growth chamber at 20°C (12 hours per day of fluorescent light). Control plants were maintained similarly. The first lesions developed on leaves of inoculated plants of both species after 7 days. *A. panax* was consistently reisolated from the lesions. The pathogenicity test was carried out twice. The presence of *A. panax* on *Aralia japonica* has been reported in Japan, Korea (2), and the United States (1) but to our knowledge, this is the first report of *A. panax* on *Aralia japonica* in Europe.

References: (1) S. Alfieri et al. Index of plant diseases in Florida. Bull. 11:52, Florida Department of Agriculture and Consumer Services, 1984 (2) S. H. Yu et al. Ann. Phytopathol. Soc. Jpn. 50:313, 1984.

Pumpkin (*Cucurbita maxima and C. pepo*), a new host of *Beet pseudo yellows virus* in California. W. M. Wintermantel, USDA-ARS, 1636 E. Alisal Street, Salinas, CA 93905. Plant Dis. 88:82, 2004; published on-line as D-2003-1031-02N, 2004. Accepted for publication 3 October 2003.

In the summer of 2002, pumpkin plants (*Cucurbita pepo* L. and *C. maxima* Duchesne) with extensive leaf chlorosis similar to those observed in crinivirus infections were found in fields at two locations in Monterey County, California. Leaves of diseased plants were observed to have large populations of the greenhouse whitefly (*Trialeurodes vaporariorum*) present. Double-stranded RNA was extracted from symptomatic leaves of these plants and tested by northern hybridization for numerous criniviruses. A positive signal was identified exclusively with probes against the *HSP70h* gene of *Beet pseudo yellows virus* (BPYV) and confirmed by reverse transcription-polymerase chain reaction (RT-PCR) amplification of a 335-nucleotide section of the BPYV minor coat protein (*CPm*) gene (3). Similar symptoms were observed in additional fields in 2003, and BPYV was again confirmed. In addition, the *CPm* RT-PCR product was cloned into a TOPO pCR2 vector (Invitrogen, Carlsbad, CA) and sequenced. BLAST analysis of the cloned *CPm* RT-PCR product sequence corresponded to the published sequence of the *CPm* gene of BPYV (98%) (3) and *Cucumber yellows virus* (CuYV), a recently sequenced crinivirus considered to be a strain of BPYV (97%) (2). Incidence of BPYV in pumpkin appears to be variable and probably corresponds to the incidence of viruliferous whiteflies. On the basis of foliar symptoms, BPYV incidence varied from less than 50% in these fields in 2002 to nearly 100% infection of a large commercial field in 2003. BPYV is transmitted semipersistently by the greenhouse whitefly and has an extensive host range (1). The virus causes economic losses worldwide for greenhouse vegetable production and is becoming an increasing problem for field crops in areas of high greenhouse whitefly incidence (3). The impact of BPYV on pumpkin production remains to be determined; however, grower data suggests an increased incidence of fruit abortion and a substantial decrease in fruit weight. To our knowledge, this is the first report of BPYV infecting pumpkin.

References: (1) J. E. Duffus. Phytopathology 55:450, 1965. (2) S. Hartono et al. J. Gen. Virol. 84:1007, 2003. (3) I. E. Tzanetakis et al. Plant Dis. 87:1398, 2003.

First Record of *Barley yellow striate mosaic virus* Affecting Wheat Summer-Nurseries in Syria. Khaled M. Makkouk, Safaa G. Kumari, Widad Ghulam, and Nouran Attar, Germplasm Program, Virology Laboratory, ICARDA, P.O. Box 5466, Aleppo, Syria. Plant Dis. 88:83, 2004; published on-line as D-2003-1102-01N, 2004. Accepted for publication 3 October 2003.

A limited survey to identify virus diseases affecting wheat in summer nurseries in agricultural stations in southern Syria was conducted during October 2002. A total of 94 bread and durum wheat samples with symptoms suggestive of virus infection (stripping, stunting, and yellowing) were collected. All samples were tested for the presence of four viruses by tissue-blot immunoassay (2) at the Virology Laboratory of ICARDA, Aleppo, Syria using the following polyclonal antibodies: *Barley stripe mosaic virus* (BSMV); *Barley yellow dwarf virus-PAV* (BYDV-PAV) and *Wheat streak mosaic virus* (WSMV) from the Virology Laboratory at ICARDA; and *Barley yellow striate mosaic virus* (BYSMV) isolated from Italy (BYSMV-Italy) and provided by M. Conti, Instituto di Fitovirologia applicata, Turino, Italy. Serological results obtained indicated that BYSMV was the most commonly encountered virus (78.7%) followed by BYDV-PAV (22.3%), whereas, BSMV and WSMV were not detected in any of the samples tested. In sodium dodecyl sulfate-polyacrylamide gel electrophoresis, followed by western blots, purified BYSMV preparations were observed to contain a 47-kDa structural protein typical of the N protein of Rhabdoviruses that reacted strongly with three BYSMV antisera (BYSMV-Italy, BYSMV-Lebanon [4], and BYSMV-Morocco [1]). Samples that reacted with BYSMV antisera were transmitted from wheat to wheat, barley, and oat plants by the planthopper *Laodelphax striatella* (Fallen) (Hemiptera: family Delphacidae) in a persistent manner, and the major symptoms of BYSMV on cereal crops were stripping and stunting. BYDV-PAV has been reported from Syria earlier (3) but to our knowledge, this is the first report of BYSMV affecting wheat in Syria.

References: (1) B. E. Lockhart et al. Plant Dis. 70:1113, 1986. (2) K. M. Makkouk and A. Comeau. Eur. J. Plant Pathol. 100:71, 1994. (3) K. M. Makkouk et al. Phytopathol. Mediterr. 28:164, 1989. (4) K. M. Makkouk et al. Plant Dis. 85:446, 2001.

Anastomosis Grouping of *Rhizoctonia solani* Associated with Black Scurf and Stem Canker of Potato in South Africa. M. Truter and F. C. Wehner, Department of Microbiology and Plant Pathology, University of Pretoria, Pretoria, 0002 South Africa. Plant Dis. 88:83, 2004; published on-line as D-2003-1103-01N, 2004. Accepted for publication 8 October 2003.

Rhizoctonia disease (black scurf of tubers and stem canker) of potato (*Solanum tuberosum* L.) caused by *Rhizoctonia solani* Kühn was first recorded in South Africa in 1918 (3). Although the sclerotial form on tubers is one of the most common potato diseases in the country, it is not known which anastomosis groups (AGs) of *R. solani* are involved. Between 1999 and 2001, *R. solani* was isolated from 28 plant and 56 soil samples collected in 7 (Eastern Free State, Gauteng, KwaZulu-Natal, Limpopo, Mpumalanga, Northern Cape, and Sandveld) of the 14 potato-production regions of South Africa and screened for hyphal anastomosis with tester strains of *R. solani* AG-1 to AG-10 according to Carling et al. (1). Of the 411 isolates from tubers with black scurf symptoms, 408 were AG-3 and three were AG-5. Symptomless tubers yielded two AG-3 isolates and three AG-5 isolates. Of 39 isolates from symptomatic stems and roots, 32 were AG-3, five were AG-4, and two were AG-5. Of the 127 isolates obtained from soil, 86, 28, 7, 3, and 3 were AG-3, AG-4, AG-5, AG-7, and AG-8, respectively. More than one AG was isolated from five of the seven regions. Virulence of 40 isolates representative of the above AGs was determined in triplicate on sprouts growing from seed tubers of potato cultivar Up-to-Date in a sand/soil mixture as described by Carling and Leiner (2) but using cultures grown in cornmeal/sand instead of colonized agar disks as inoculum. Damage to sprouts (lesions, girdling, and death) was assessed after 28 days at 16 to 28°C according to the 0 to 4 rating scale (2). Chi-square analysis of the data indicated that AG-3 was the most virulent, with isolates from sclerotia on tubers and lesions on stems more aggressive than those from symptomless tubers or soil. AG-4 and AG-5 caused significantly less disease than AG-3, but none of the AG-7 and AG-8 isolates showed any virulence to potato sprouts.

References: (1) D. E. Carling et al. Phytopathology 77:1609, 1987. (2) D. E. Carling and R. H. Leiner. Phytopathology 80:930, 1990. (3) E. M. Doidge. S. Afr. Fruit Growers 5:6, 1918.

First Report of *Fusarium oxysporum* Causing Vascular Wilt of Lamb's Lettuce (*Valerianella olitoria*) in Italy. A. Garibaldi, G. Gilardi, and M. L. Gullino, AGRINNOVA, Via Leonardo da Vinci, 44, 10095 Grugliasco, Italy. Plant Dis. 88:83, 2004; published on-line as D-2003-1103-02N, 2004. Accepted for publication 4 October 2003.

Lamb's lettuce (*Valerianella olitoria*), also known as corn salad, is increasingly grown in Italy and used primarily in the preparation of mixed processed salad. In the summer of 2003, plants of lamb's lettuce cvs. Trophy and Palmares exhibiting wilt symptoms were observed in several commercial greenhouses near Bergamo in northern Italy. Wilted 30-day-old plants were observed first during the month of June, at the time of thinning when temperatures ranged between 28 and 35°C. Disease was generally uniform in the greenhouses and 30 to 50% of the plants were affected. Vascular tissue of affected seedlings appeared red or brown but later turned brown or black. Affected plants were stunted and developed yellowed leaves. Vascular discoloration was continuous from the upper taproot through the crown to the leaf. *Fusarium oxysporum* was consistently isolated from symptomatic vascular tissue onto a Fusarium-selective medium (1). Seeds of the same cultivars (Trophy and Palmares) affected by the wilt in the field were artificially inoculated by dipping them for 15 min into spore suspensions (1×10^6 conidia per ml) of three isolates of *F. oxysporum* obtained from infected plants. Noninoculated seeds served as control treatments. Forty seeds per treatment were sown in pots (1-liter volume) containing steam-sterilized soil and maintained at 25°C in a growth chamber programmed for 12 hours of light per day. Wilt symptoms developed on both cultivars 20 days after seeding, and *F. oxysporum* was consistently reisolated from infected plants. The plants obtained from noninoculated seeds remained healthy. The pathogenicity test was carried out twice with similar results. To our knowledge, this is the first report of *F. oxysporum* causing vascular wilt of lamb's lettuce and may warrant a new *forma specialis* designation.

Reference: (1) H. Komada. Rev. Plant Prot. Res. 8:114, 1975.

First Report of Onion Rust Caused by *Puccinia allii* on *Allium pskemense* and *A. altaicum*. S. L. Lupien, B. C. Hellier, and F. M. Dugan, USDA-ARS Western Regional Plant Introduction Station, Washington State University, Pullman 99164. Plant Dis. 88:83, 2004; published on-line as D-2003-1103-03N, 2004. Accepted for publication 8 October 2003.

In June 2003, uredinial and telial pustules were seen on leaves of accession W6-12755 *Allium pskemense* B. Fedtsch. originating from Uzbekistan and grown for germplasm increase in Pullman, WA. W6-18947 *A. altaicum* Pall., originating from Mongolia, displayed similar symptoms in the same garden in June 2000. *A. altaicum* is a wild onion exploited for food in its native range and is ancestral to *A. fistulosum* L., bunching onion (2). *A. pskemense* is a wild perennial sometimes propagated under cultivation (2). Both species have been exploited for research in breeding and systematics of *Allium* and used to a lesser degree in screening for pest or disease resistance. Clustered, golden orange, amphigenous uredinia were approximately 1 × 0.5 mm and surrounded by stromatic, subepidermal, blackish telia of variable size. Urediniospores (thick-walled, pale orange, echinulate, (25-) 27 to 32 (-34) × (19-) 21 to 25 µm, with as many as 10 scattered, indistinct pores), teliospores (two-celled, smooth, golden brown, 42 to 65 × 18 to 26 µm), and mesospores (27 to 42 × 15 to 21 µm, and approximately 30% as frequent as teliospores) all approximated the description for *P. allii* Rudolphi (4), but were more strongly congruent with the description of *Puccinia blasdalei* Diet. & Holw. (1), now considered a synonym (4). Specimens are deposited with WSP, Washington State University, Pullman. *P. allii* or its synonyms have been recorded from over 30 species of *Allium* (1,3,4), but to our knowledge, this is the first report of this rust on *A. pskemense* or *A. altaicum*.

References: (1) J. C. Arthur. Manual of the Rusts in United States and Canada, Hafner Publishing, N.Y., 1962. (2) J. L. Brewster. Onions and Other Vegetable Alliums. CABI, Wallingford, Oxon, U.K, 1994. (3) D. F. Farr et al. Fungal Databases, Systematic Botany and Mycology Laboratory, On-line publication. ARS, USDA, 2003. (4) G. F. Laundon and J. M. Waterston. *Puccinia allii*. No. 52 in: Descriptions of Pathogenic Fungi and Bacteria. CMI, Kew, Surrey, U.K., 1965.

(*Disease Notes* continued on next page)

First Report of Stem Rot of Guiana Chestnut (*Pachira aquatica*) Caused by *Pythium splendens*. M. Tojo, Graduate School of Agriculture and Biological Sciences, Osaka Prefecture University, Gakuen-cho, Sakai, Osaka, 599-8531, Japan; and K. Kuroda and H. Suzuki, Mie Prefectural Science and Technology Promotion Center, Kawakita, Ureshino-cho, Mie 515-2316, Japan. Plant Dis. 88:84, 2004; published on-line as D-2003-1104-01N, 2004. Accepted for publication 6 October 2003.

Guiana chestnut is a perennial tropical plant that has recently become popular as a potted ornamental in Japan. In October 2001, severe stem rot occurred on Guiana chestnut plants grown in a greenhouse in Mie Prefecture, Japan. Water-soaked lesions appeared initially at the base of the stems and enlarged gradually toward the tops of plants. The affected tissues were softened and turned dark brown. Rotting was observed in the vascular bundles with advanced disease development. Globose hyphal swellings were numerous on diseased stems. Sections from diseased stems were cleaned by washing with running tap-water, placed on water agar, and incubated at 25°C. A species of *Pythium* was identified on the basis of morphological and cultural characteristics (1) and isolated consistently from the rotted stems of diseased plants. All isolates produced abundant hyphal swellings that were globose, smooth, 12 to 39 µm in diameter, mostly terminal, dark colored, and with dense granulated contents. Zoospores were absent. All isolates were of the compatibility '+ type' with production of sexual organs when paired with cultures of the '– type' tester isolate of *Pythium splendens* Braun (CBS462.48). Oogonia produced by crossings between Guiana chestnut isolates and isolate CBS462.48 were terminal or intercalary, globose, smooth-walled, and 32 to 38 µm in diameter. Antheridia were terminal, one to three per oogonium, sac-like, and diclinous. Oospores were single, aplerotic, globose, and 28 to 32 µm in diameter. The thickness of the oospore wall ranged from 1 to 2 µm. The internal transcribed spacer rDNA sequences of representative isolate OPU591 from Guiana chestnut matched those of CBS462.48 (similarity 99.2%) and have been deposited in GenBank under the Accession No. AY375242. Pathogenicity tests were conducted on potted Guiana chestnut plants (30 cm high and 7 to 10 cm in diameter at base of the stem) using isolate OPU591. A mycelial suspension from one culture, grown at 25°C for 7 days on water agar, was inoculated onto a single plant. Prior to inoculation, a wound (10 mm deep and 30 mm long) was made on the surface at the stem base on five plants. The mycelial suspension was poured onto the base of the stems of five wounded and five nonwounded plants. In addition, five wounded and five nonwounded, noninoculated plants were used as controls. Plants were maintained in a greenhouse for 8 weeks after inoculation. The temperature and relative humidity in the greenhouse ranged from 25 to 30°C and 65 to 75%, respectively. Dark-brown rotting developed on the stems of all wounded, inoculated plants by 20 days after inoculation. *P. splendens* was isolated from diseased tissues and found to be morphologically identical to the original isolate. This confirmed *P. splendens* as the causal agent of the disease. Disease did not develop on nonwounded inoculated plants or noninoculated plants. To our knowledge, this is the first report of *P. splendens* on Guiana chestnut. Potted plants of Guiana chestnut are often injured by frequent transplanting and transferring. Such injuries may have predisposed the plant to infection by *P. splendens.*

Reference: (1) A. J. van der Plaats-Niterink. Page 1 in: Monograph of the Genus *Pythium*. Studies in Mycology Vol. 21, Centraalbureau Voor Schimmelcultures, Baarn, the Netherlands, 1981.

First Report of *Monosporascus cannonballus* on Melon in Brazil. R. Sales Jr., I. J. Bezerra do Nascimento, and L. de Souza Freitas, Escola Superior de Agricultura de Mossoró, ESAM, Caixa Postal 137, 59.600-970, Mossoró-RN, Brazil; and R. Beltrán, J. Armengol, A. Vicent, and J. García-Jiménez, Instituto Agroforestal Mediterráneo, Universidad Politécnica de Valencia, Camino de Vera s/n, 46022-Valencia, Spain. Plant Dis. 88:84, 2004; published on-line as D-2003-1105-01N, 2004. Accepted for publication 10 October 2003.

Approximately 15,000 ha of melon (*Cucumis melo* L.) are grown in the northeastern section of Brazil, mostly for export to Europe during the winter months. Surveys for melon vine decline diseases were carried out in farms in the municipalities of Mossoró (Rio Grande do Norte) and Quixeré (Ceará) during 2002 and 2003. Symptoms typical of vine decline were observed in several fields and included yellowing of crown leaves just prior to harvest and collapse of many of the vines. Affected plants exhibited necrotic root systems and lacked most of the secondary and tertiary feeder roots. Numerous perithecia were observed on roots which, when examined with a microscope, showed characteristic asci and ascospores of the fungus *Monosporascus cannonballus* Pollack & Uecker (2). Isolations were made from the crown region and primary and secondary roots of affected plants by excising 4- to 6-mm pieces that were surface sterilized for 30 to 60 s with 1.5% active chlorine solution. Seven tissue pieces from each plant part were placed on potato dextrose agar (PDA) containing 0.5 g liter^{-1} of streptomycin sulfate. Plates were examined daily for fungal growth for 7 days, and hyphal tips from all colonies were transferred to PDA for subsequent growth and sporulation. *M. cannonballus* was isolated from 50% of the root sections. All isolates produced only one ascospore per ascus. Pathogenicity of four isolates was confirmed in the greenhouse on the muskmelon cv. Temprano Rochet. Inoculum was produced in a sand-oat hulls (*Avena sativa* L.) medium (0.5 liter of sand, 46 g of ground oat hulls, and 37.5 ml of distilled water) and incubated at 25°C for 1 month. Colony forming units (CFU) were quantified by serial dilution using 1% hydroxyethyl cellulose. A sterilized mixture of equal portions (vol/vol) of sand and peat moss was used to fill plastic pots (17 cm in diameter), and inoculum was added to produce an inoculum concentration of 20 CFU g^{-1}. Five melon seeds were planted in each pot and after germination, were thinned to one seedling per pot. There were five replicated pots for each treatment with an equal number of uninfested pots. Plants were evaluated for disease 45 days after sowing. Roots were exposed by carefully washing the potting mix away. All isolates of *M. cannonballus* tested were highly aggressive and caused severe root necrosis compared with the noninoculated control plants. *M. cannonballus* was reisolated from symptomatic plants, confirming Koch's postulates. Double cropping in the same fields for several years has created serious problems in Brazil, which are related to this soilborne pathogen that also causes root rot and vine decline of watermelon (*Citrullus lanatus* (Thunb.) Matsum. & Nakai) worldwide (1). To our knowledge this is the first report of *M. cannonballus* in Brazil and South America.

References: (1) R. D. Martyn and M. E. Miller. Plant Dis. 80:716, 1996. (2) F. G. Pollack and F. A. Uecker. Mycologia 66:346, 1974.

First Report of *Peronospora farinosa* f. sp. *spinaciae* Race 5 Causing Downy Mildew on Spinach in Florida. B. M. Irish and J. C. Correll, Department of Plant Pathology, University of Arkansas, Fayetteville 72701; R. N. Raid, Department of Plant Pathology, University of Florida, Belle Glade 33430; and T. E. Morelock, Department of Horticulture, University of Arkansas, Fayetteville 72701. Plant Dis. 88:84, 2004; published on-line as D-2003-1112-02N, 2004. Accepted for publication 24 October 2003.

Downy mildew, caused by *Peronospora farinosa* f. sp. *spinaciae*, is an economically important disease in most areas where spinach is grown. This disease has become increasingly important in production fields for prepackaged salad mixes where plant densities typically are very high. In Florida, spinach production for these markets has reached approximately 200 ha. Currently, seven physiological races of the downy mildew pathogen have been described (1). Downy mildew was observed in several commercial spinach fields in the Everglades agricultural area of Palm Beach County, Florida in January 2003 on cvs. Unipak 151 and Merlo Nero. Symptoms appeared as chlorotic and necrotic leaf spots. Disease incidence reached approximately 25% in some field locations. Economic losses were significant, since entire plantings in several fields were not harvested as a result of diminished quality. The race of a field isolate recovered from the cv. Unipak 151 was determined following greenhouse inoculation procedures and using differentials outlined by Irish et al (1). Greenhouse inoculation tests were conducted twice. Disease reactions on a U.S. and international set of differentials indicated that the isolate was race 5. To our knowledge, this is the first report of race 5 occurring outside of the California/Arizona spinach production area in the United States. There are commercial spinach lines with resistance to race 5, as well as the other described races (1).

References: (1) B. M. Irish et al. Plant Dis. 87:567, 2003.

First Report of Basal Stem Rot of Golden Barrel Cactus Caused by *Fusarium oxysporum* f. sp. *opuntiarum* in Italy. G. Polizzi and A. Vitale, Dipartimento di Scienze e Tecnologie Fitosanitarie, University of Catania, Via S. Sofia 100, 95123 Catania, Italy. Plant Dis. 88:85, 2004; published on-line as D-2003-1107-01N, 2004. Accepted for publication 15 October 2003.

Golden barrel cactus (*Echinocactus grusonii* Hildm.) is the most common landscape cactus in southern Italy. During 2000, 2001, and 2002, a basal stem rot of golden barrel cactus was observed in several plastichouses located in eastern Sicily with disease levels of nearly 100% on young plants (up to 15 cm in diameter). On the basal crown area, the plants showed pale brown or yellow-orange, sunken lesions bordered by a reddish orange strip up to 1 mm wide. A water-soaked rot or white mycelium at the soil line was also observed. Thirty pieces (0.5 to 1 cm) from the edge of symptomatic tissues were surface disinfected for 2 min in 0.8% (wt/vol) NaOCl, washed with sterile distilled water (SDW), and placed on potato dextrose agar (PDA). In addition, 20 pieces of affected tissue were ground in 400 µl of SDW (1:3, wt/wt), and the resulting suspensions were streaked by loops on PDA supplemented with 1.1 µl/ml of lactic acid (pH 4.4). A *Fusarium* sp. was consistently isolated from affected tissue pieces and streaks. Koch's postulates were performed at 25°C by inoculating 24 golden barrel cactus plants in 12-cm-diameter pots (12 plants previously sterile needle wounded) with 10 ml per plant of three suspensions (10^6 CFU/ml) of three isolates sprayed onto the basal stem. One milliliter per ten g of soil of each suspension was also added in the crown portion of golden barrel cacti. Twelve control cacti (six wounded) were sprayed only with SDW. Further pathogenicity tests were carried out on Thanksgiving cactus (*Schlumbergera truncata* (Haw.) Moran), devil's tongue barrel cactus (*Ferocactus latispinus* (Haw.) Britton & Rose), peruvian old man cactus (*Espostoa lanata* (Kunth) Britton & Rose), and *Parodia* spp. by inoculating eight plants for each host (four wounded) by placing 9-day-old 6-mm mycelial plugs at the base of the healthy cacti. An equal number of plants (four wounded) was inoculated only with a PDA plug. All cacti were maintained in polyethylene bags (90 to 95% for 72 hr) at 25°C. After 12 to 15 days, all wounded inoculated golden barrel, devil's tongue barrel, and peruvian old man cacti exhibited similar symptoms observed in the plastichouses. Typical symptoms were visible also in nonwounded and inoculated cacti 15 days later. Yellow-orange, tan, sunken, and roughly circular lesions were observed on the wounded and inoculated Thanksgiving and *Parodia sp.* cacti. Control plants were symptomless. The causal fungus was always reisolated from infected cacti. On the basis of 3-septate macroconidia (27 to 35 µm long × 3 to 4 µm wide [average 31.45 × 3.18 µm]), microconidia aseptate, single or double chlamydospores, and monophialide conidiophores observed on carnation leaf agar, and considering the susceptibility of all other inoculated hosts, the fungus was identified as *F. oxysporum* Schlechtend. f. sp. *opuntiarum* (Speg.) (1). To our knowledge, this is the first report of basal stem rot of golden barrel cactus in Italy.

Reference: (1) W. Gerlach. Phytopathol. Z. 74, 197, 1972.

First Report of *Melampsora larici-populina* on *Populus* spp. in Eastern North America. L. Innes and L. Marchand, Direction de la conservation des forêts, ministère des Ressources naturelles, Québec, Canada; P. Frey, INRA, Nancy, France; and M. Bourassa and R. C. Hamelin, Canadian Forest Service, Natural Resources Canada, Sainte-Foy, Canada. Plant Dis. 88:85, 2004; published on-line as D-2003-1107-02N, 2004. Accepted for publication 16 October 2003.

In September 2002, yellow spots were observed on the leaf surface of a hybrid poplar (*Populus maximowiczii* Henry × *P. balsamifera* L.) grown at the Berthier forest nursery (46°2′N, 73°11′W) in the St. Lawrence Valley (Lanaudière Region, Québec, Canada). Disease severity was low, but the pathogen was present on a hybrid that was previously thought to be resistant to *Melampsora medusae* Thuem, the only reported poplar rust in eastern North America. Uredinia typical of a *Melampsora* sp. were observed on the abaxial leaf surface. The observed urediniospores were longer (32 to 48 µm) than the expected range for *M. medusae* (23 to 35 µm) and possessed an apical bald spot; thick paraphyses were also observed. These characteristics are diagnostic of *M. larici-populina* Kleb (2). Samples were deposited in the National Mycological Herbarium of Canada (DAOM 232107 and 232108) and in the Quebec Forest Biology Herbarium (QFB14703 and 14704). DNA was extracted from uredinia, and the internal transcribed spacer (ITS) of the ribosomal RNA gene was amplified and sequenced (GenBank Accession Nos. AY429656 and AY429657). There was a 100% match between the two sequences obtained and that of *M. larici-populina* (GenBank Accession No. AY375267), but there was approximately 12% divergence with the ITS sequence of *M. medusae* (GenBank Accession No. AY375273-5). This is the first report of *M. larici-populina* in eastern North America. This fungus was reported on *P. trichocarpa* × *P. deltoides* hybrids in the western United States in the early 1990s (1). It appears that *M. larici-populina* can overwinter in Québec because it was observed again at the nursery in September 2003. The occurrence of *M. larici-populina* in eastern North America has direct implications for the poplar industry since the host specificities of *M. medusae* and *M. larici-populina* differ; *P. balsamifera* and *P. maximowiczii* are sensitive to *M. larici-populina* (3). Hybrids with *P. balsamifera* or *P. trichocarpa* components may be particularly at risk.

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Bacterial Spot of Tomato and Pepper Caused by *Xanthomonas axonopodis* pv. *vesicatoria* in the Western Mediterranean Region of Turkey. H. Basim, University of Akdeniz, Faculty of Agriculture, Department of Plant Protection, 07050, Antalya, Turkey; E. Basim, University of Suleyman Demirel, Faculty of Agriculture, Department of Plant Protection, 32260, Isparta, Turkey; and J. B. Jones, G. V. Minsavage, and E. R. Dickstein, University of Florida, Department of Plant Pathology, Gainesville 32611. Plant Dis. 88:85, 2004; published on-line as D-2003-1112-01N, 2004. Accepted for publication 22 October 2003.

Xanthomonas axonopodis pv. *vesicatoria*, causal agent of bacterial spot of tomato (*Lycopersicon esculentum* L.) and sweet pepper (*Capsicum annuum* L.) was isolated from tomato and pepper plants in greenhouse production in the Province of Antalya, in southwestern Turkey. Disease incidence was less than 4% of plants observed in 2001 and ranged from 1 to 20% in 2002. Eleven seedling-producing companies and 26 greenhouses that produce tomato and pepper were surveyed during the rainy seasons of 2001 and 2002. The increase in disease incidence in 2002 is an indication that this disease is becoming more prevalent on tomato and pepper plants grown in greenhouses in southwestern Turkey. A gram-negative bacterium producing yellow-pigmented colonies on nutrient agar was consistently isolated from brown, circular spots on leaflets of tomato and sweet pepper seedlings. Five isolates were pathogenic on commercial cultivars of tomato and pepper when bacterial suspensions (10^8 CFU/ml) were infiltrated into the intercellular spaces of leaves to determine race by using procedures described by Bouzar et al. (1). All the isolates produced hypersensitive reaction responses on tomato genotype cv. Hawaii 7998 and pepper genotype cvs. 20 R and 30 R and were designated tomato race 1 pepper race 1 (T1P1) (1). Fatty acid analysis of the strains identified them as *X. axonopodis vesicatoria* with similarity index values of 0.872 to 0.933. In addition, the strains were tested with *X. axonopodis vesicatoria*-specific polymerase chain reaction primers (RST 2/3 and RST 9/10) (2). The isolates were determined to be *X. axonopodis vesicatoria*. Although bacterial spot of tomato has been suspected in Turkey for a number of years, to our knowledge, this is the first report of the bacterium on tomato.

References: (1) H. Bouzar et al. Phytopathology 84:663, 1994. (2) R. P. Leite, Jr. et al. Appl. Env. Microbiol. 60:1068, 1994.

(*Disease Notes* continued on next page)

Root and Foot Rot on Tomato Caused by *Phytophthora infestans* Detected in Belgium. B. Lievens, I. R. M. Hanssen, and A. C. R. C. Vanachter, Scientia Terrae Research Institute, Fortsesteenweg 30A, B-2860 Sint-Katelijne-Waver, Belgium; B. P. A. Cammue, Centre of Microbial and Plant Genetics (CMPG), Katholieke Universiteit Leuven, Kasteelpark Arenberg 20, B-3001 Leuven, Belgium; and B. P. H. J. Thomma, Wageningen University, P.O. Box 8025, 6700 EE Wageningen, the Netherlands. Plant Dis. 88:86, 2004; published on-line as D-2003-1110-01N, 2004. Accepted for publication 21 October 2003.

In January 2003, a severe root and foot rot was observed on 2-month-old wilted tomato (*Lycopersicon esculentum* Mill.) plants in a large-scale (2.5 ha) commercial greenhouse setting in Belgium. Tomato plants (10%) produced from healthy nursery-grown seedlings and planted to new, clean rockwool and drip irrigation with UV-disinfected water developed symptoms. Symptom development was restricted to lower plant parts with severe rotting of the entire root system and dark lesions girdling the stem base. No symptoms of disease were observed on the foliage or upper stems. Cross sections of the stem base revealed brown discoloration of internal tissue, including the vascular tissue and pith. Dark brown lesions also occurred on the roots. Sections of the stem base, the upper roots (sampled near to the stem base), and the lower roots (sampled on roots deeper in the rockwool) were plated separately on corn meal agar. The oomycete pathogen *Phytophthora infestans* (Mont.) de Bary was identified in each sample on the basis of morphological characteristics observed directly with light microscopy. Branched sporangiophores with slight swellings and characteristic lemon-shaped sporangia (35 × 20 µm and ratio length/width of 1.75 µm) at their tips were obvious after incubation in darkness at 24°C. Oospores and chlamydospores were not observed. After multiple soil treatment with oomycete-specific fungicides, the plants recovered. Since the occurrence of *P. infestans* on roots is unusual, the identity of the pathogen on the diseased plant tissues was confirmed with three techniques, DNA array identification, internal transcribed spacer (ITS) sequencing, and a polymerase chain reaction (PCR) amplification using *P. infestans*-specific primers. DNA was directly processed from separate samples of upper and lower root and stem base tissue. The DNA array used was originally developed to detect and identify the key fungal pathogens of tomato (2). Among detector probes for other tomato pathogens, this array contains oligonucleotide detector probes for *P. infestans* (PIN1: 5′-GGT TGT GGA CGC TGC TAT T and PIN2: 5′-AAT GGA GAA ATG CTC GAT TC). These probes are based on ITS sequences (ITS I and ITS II). Using conserved ribosomal primers OOMUP18Sc (5′-TGC GGA AGG ATC ATT ACC ACA C) and ITS4, oomycete DNA was amplified by PCR and simultaneously labeled with alkaline-labile digoxigenin (2). All generated amplicons strongly hybridized to the oligonucleotide detector probes for *P. infestans* and not to any other pathogen-specific detector probe present on the array. The pathogen could not be detected in roots and stem bases of symptomless plants. In addition, the ITS-region was sequenced and showed 100% homology with multiple GenBank accessions of *P. infestans* sequences. As a third confirmatory test, a PCR was performed on DNA extracts from infected root and stem base tissues using a primer set specific to *P. infestans* (O8-3/O8-4 [1]). A band of the expected size was produced for the infected stem base and root samples. Until now, this pathogen was known worldwide to cause late blight on potatoes and tomatoes. To our knowledge, this is the first report of root and foot rot of tomato caused by *P. infestans*.

References: (1) H. S. Judelson and P. W. Tooley. Phytopathology 90:1112, 2000. (2) B. Lievens et al. FEMS Microbiol. Lett. 223:113, 2003.

First Report of Petunia Root Rot Caused by *Rhizoctonia solani* in Argentina. E. R. Wright, M. C. Rivera, and K. Asciutto, Cátedra de Fitopatología, Facultad de Agronomía, Universidad de Buenos Aires, Av. San Martín 4453 (1417), Capital Federal, Argentina; and L. Gasoni, V. Barrera, and K. Kobayashi, IMYZA-INTA, C.C. 25 (1712) Cautelar, Prov. Buenos Aires, Argentina. Plant Dis. 88:86, 2004; published on-line as D-2003-1110-02N, 2004. Accepted for publication 16 September 2003.

Common garden petunias (*Petunia* × *hybrida* Hort. Vilm.-Andr.) are herbaceous annual plants with brightly colored flowers up to 10 cm in diameter. During the winter of 2002, crown and root rot were observed on plants (cv. Ultra) growing in five greenhouses in Buenos Aires. Affected plants were randomly distributed in the greenhouses, and mean disease incidence in all the greenhouses was 26%. Basal leaves turned yellow and gradually became necrotic, and infected plants were often killed. Small pieces of affected tissues were disinfested in 2% sodium hypochlorite for 1 min and plated on 2% potato dextrose agar (PDA). Fifteen isolates identified to the genus *Rhizoctonia* were obtained. Fungal colonies were initially white, turned brown with age, and produced irregularly shaped, brown sclerotia. Hyphal branched at right angles, were constricted at the base of the branch near the union with main hyphae, and septate near the constriction. Basidia were not observed in the greenhouses or on the plates. Isolates were cultivated on water agar and incubated at 25°C for 3 days. Hyphal cells were determined to be multinucleate when stained with 1% aniline blue solution (2) and examined at ×400. Anastomosis group of one isolate was determined by using AG-4 HG II, AG-1 IA, AG-1 IB, AG-1 IC, AG-2 2-1, and AG-2 2IIIB tester strains of *Rhizoctonia solani* that includes isolates reported to be pathogenic on ornamentals (1). Anastomosis was observed only with strains of AG-4 HG II. Pathogenicity on this isolate was conducted on potted, healthy, adult plants that were 10 to 22 cm high and flowering. Thirty-five plants were inoculated by placing 1 cm^2 pieces of PDA from 7-day-old mycelial cultures near the base of the stem. Twelve control plants were treated with 1 cm^2 PDA plugs. Plants were kept at 22 to 24°C, >95% relative humidity, and 12 h of fluorescent light. Wilt symptoms due to basal stem rot appeared 7 days after inoculation, and all the inoculated plants died within 27 days. Control plants remained disease free. The pathogen was reisolated from symptomatic tissues, completing Koch's postulates. To our knowledge, this is the first report of *R. solani* causing disease on petunia in Argentina.

References: (1) D. M. Benson and D. K. Cartwright. Ornamental diseases incited by *Rhizoctonia* spp. Pages 303-314 in: *Rhizoctonia* species: Taxonomy, Molecular Biology, Ecology, Pathology and Disease Control. B. Sneh et al., eds. Kluwer Academic Publishers, London, England, 1996. (2) C. C. Tu and J. W. Kimbrough. Mycologia 65:941, 1973.

***Strawberry latent ringspot virus* Infecting Roses in India.** S. Kulshrestha, V. Hallan, G. Raikhy, R. Ram, and A. A. Zaidi, Floriculture Division, IHBT (CSIR) P.O. Box 6, Palampur-176 061 (H.P.), India. IHBT Communication No. 0337. Plant Dis. 88:86, 2004; published on-line as D-2003-1117-01N, 2004. Accepted for publication 27 September 2003.

Rose is an economically important crop of India and the world. A survey of rose plantations in and near the Kangra Valley of Himachal Pradesh, India, showed virus-like symptoms, including yellow flecking in young leaves and reduction in leaflet size, while some were symptomless. These symptoms are similar to those for *Strawberry latent ringspot virus* (SLRSV) (1). Sap inoculation from symptomatic and some symptomless leaves to *Chenopodium amaranticolor* resulted in chlorotic local lesions followed by systemic chlorosis. SLRSV was detected in this indicator host and six rose cultivars (Happiness, Iceberg, First Prize, Ganga, Pink Panther, and Oklahoma) showing characteristic symptoms of SLRSV using enzyme-linked immunosorbent assay (ELISA) with ELISA kit (DSMZ, Braunschweig, Germany). Reverse transcription-polymerase chain reaction was performed with SLRSV-specific primers (2), and a product of the expected size of ~181 bp was amplified. The authenticity of the fragment was confirmed by sequencing. Isolated SLRSV was also inoculated to seed-grown rose seedlings and after 20 days postinoculation the same symptoms (yellow flecking in young leaves) were observed. These results established the identity of the virus that caused yellow flecking on rose leaves in India as SLRSV. To our knowledge, this is the first report of SLRSV infecting rose in India.

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Detection of *Phytophthora ramorum* Blight in Oregon Nurseries and Completion of Koch's Postulates on *Pieris, Rhododendron, Viburnum,* and *Camellia.* J. L. Parke, Oregon State University, Corvallis; R. G. Linderman, USDA-ARS, Corvallis, OR; and N. K. Osterbauer and J. A. Griesbach, Oregon Department of Agriculture, Salem. Plant Dis. 88:87, 2004; published on-line as D-2003-1111-01N, 2004. Accepted for publication 22 October 2003.

Phytophthora ramorum, the cause of sudden oak death in California and Oregon coastal forests and ramorum blight in European nurseries and landscapes (1), was detected in six Oregon nurseries in Jackson, Clackamas, and Washington counties from May to June 2003. The pathogen was isolated from: *Viburnum bodnantense* 'Dawn', *V. plicatum* var. *tomentosum* 'Mariesii', *Pieris japonica* × *formosa* 'Forest Flame', *P. japonica* 'Variegata' and 'Flaming Silver', *P. floribunda* × *japonica* 'Brouwer's Beauty', *Camellia sasanqua* 'Bonanza' and other cultivars, *C. japonica*, and *Rhododendron* × 'Unique'. Samples of symptomatic tissues were plated on a *Phytophthora*-selective medium (PARP) and tested by polymerase chain reaction (PCR) (3). All samples positive for *P. ramorum* with PCR yielded *P. ramorum* isolates in culture. The isolates have the European genotype, mating type A1, except for the *Camellia* spp. isolates, which have the North American genotype, mating type A2 (2). Isolates are deposited in the American Type Culture Collection. Koch's postulates for this pathogen have been completed on *V. bodnantense* and *C. japonica* (1). To confirm pathogenicity on the new hosts, isolates from *V. plicatum* var. *tomentosum* 'Mariesii', *Pieris* × 'Forest Flame', *Pieris* × 'Brouwer's Beauty', and *P. japonica* 'Variegata' and 'Flaming Silver' were used to inoculate healthy plants of the same cultivars. For isolates from *Rhododendron* × 'Unique' and *C. sasanqua* 'Bonanza', pathogenicity was tested on *Rhododendron* × 'Nova Zembla' and *C. sasanqua* 'Sutsugekka' and 'Kanjiro'. Three to five plants of each cultivar were inoculated and three to five were noninoculated. Zoospore inoculum was prepared on dilute V8 agar for one isolate from each host. Foliage of plants growing in 10-cm pots was dipped for 5 sec in a zoospore suspension (3×10^4 zoospores per ml) or sprayed to runoff with a hand mister (6×10^4 zoospores per ml). Control plants were dipped in or sprayed with sterile water. *C. sasanqua* plants were also inoculated by placing 6-mm mycelial plugs on individual leaves that had been wounded by piercing with a pin. Control leaves were wounded but not inoculated. Foliage was enclosed in plastic bags to retain humidity and the pathogen, and plants were incubated in a locked growth chamber (21 to 23°C). After 21 days, plants were examined for symptoms, and isolations onto PARP were made. All inoculated plants showed foliar symptoms, and *P. ramorum* was consistently isolated from inoculated plants, but not from asymptomatic control plants. On *Rhododendron* × 'Nova Zembla', nearly all leaves were wilted and dead, as were terminal buds and stems. *Pieris* spp. cultivars exhibited leaf and stem necrosis and defoliation. On *V. plicatum* var. *tomentosum* 'Mariesii', necrotic leaf lesions and defoliation of the lower leaves were observed. On *C. sasanqua*, necrotic lesions developed only on wounded leaves inoculated with mycelial plugs; these leaves abscised. Our results confirm the pathogenicity of Oregon nursery isolates of *P. ramorum* on *V. plicatum* var. *tomentosum* 'Mariesii', *P. japonica* × *formosa* 'Forest Flame', *P. japonica* 'Variegata' and 'Flaming Silver', *P. floribunda* × *japonica* 'Brouwer's Beauty', *C. sasanqua* and *Rhododendron* and complete Koch's postulates for several new hosts.

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