

Disease Notes

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

Xanthomonas Blight of Onion in South Africa. J. J. Serfontein, Plant Protection Research Institute, Agricultural Research Council, Private Bag X134, Pretoria 0001, South Africa. *Plant Dis.* 85:442, 2001; published on-line as D-2001-0201-03N, 2001. Accepted for publication 18 December 2000.

During April 1999, a foliar blight of onion (*Allium cepa* L. 'Granex 33') was reported in an early commercial planting under center pivot irrigation in the Limpopo Valley of the Northern Province of South Africa. Regular fungicide sprays failed to inhibit the progress of the disease. Foliar symptoms started as water-soaked lesions that elongated and turned chlorotic followed by tissue collapse in some leaves. Leaves often collapsed at the point of infection. Bulb size was severely reduced and premature leaf death caused irregular maturation and bulb size in the field. The symptoms were similar to those of *Xanthomonas* blight, described on the same cultivar in Hawaii (1). Microscopic examination of hand cut sections through lesion margins showed bacterial streaming. Isolation on semi-selective diagnostic milk Tween agar (2) yielded almost pure cultures of a typical xanthomonad. The mucoid, yellow pigmented bacterium was rod shaped, gram negative, catalase positive, oxidase negative, utilized glucose oxidatively, and was lypolytic (Tween 80), proteolytic (skimmed milk), and amolytic. Biolog GN Microplate profiles as read by the MicroLog database release 3.50 (Biolog, Hayward, CA) were similar to those of a pathovar (similarity indices of 0.29 to 0.71). Symptoms were successfully reproduced on glasshouse grown Granex 33 seedlings at the five-leaf stage by spray and syringe inoculations (1) and the pathogen reisolated as described above. Ten seedlings were used in the pathogenicity test, of which five served as controls. After inoculation, seedlings were covered overnight with plastic bags, after which bags were removed and seedlings grown in the greenhouse at 24 to 30°C and natural light until symptom development. Attempts to isolate the pathogen from the seed lot used to plant the affected field were unsuccessful. The disease re-occurred in early plantings of Granex 33 on the same farm in April 2000 toward the end of an unusually wet summer rainy season. Damage caused by the disease was so severe in one early planting that it was plowed under. High temperatures and humid conditions combined with overhead irrigation could have enhanced disease development and spread during the early growth of the crop. No further spread was observed during cooler and drier weather later in the season.

References: (1) A. M. Alvarez et al. *Phytopathology* 68:1132, 1978. (2) T. Goszczynska and J. J. Serfontein. *J. Microbiol. Methods* 32:65, 1998.

First Report of Tomato Spotted Wilt Virus on Potatoes in Iran. R. Pourrahim, Sh. Farzadfar, A. A. Moini, and N. Shahraeen, Plant Virology Department, Plant Pests and Diseases Research Institute, P.O.Box 19395-1454, Tehran, Iran; and A. Ahoonmanesh, Plant Protection Department, Isfahan University of Technology, Isfahan, Iran. *Plant Dis.* 85:442, 2001; published on-line as D-2001-0206-01N, 2001. Accepted for publication 25 January 2001.

Severe leaf and stem necrosis before flowering was observed in potato (*Solanum tuberosum*) fields of Firouzkoh Province, Iran, during the summer of 1998. Infected plants died before the end of the growing season. Necrosis was more severe in cv. *Agria* than in cvs. *Ajax* and *Arinda*. A high population of *Thrips tabaci* was observed in August and September. *Tomato spotted wilt virus* (TSWV) (1) was detected in affected potatoes by using specific TSWV-IgG (from Bioreba) in double-antibody sandwich enzyme linked immunosorbent assay and by indicator plant reactions. Mechanical inoculation of indicator plants with leaf extracts of symptomatic potatoes produce necrotic local lesions in *Chenopodium quinoa*, *C. amaranticolor*, *Gomphrena globosa*, *Vicia faba*, *Vigna sinensis*, *Phaseolus aureus* var. *Gohar*, *P. vulgaris*, and *Petunia hybrida*. The virus caused systemic necrosis in *Capsicum frutescens*, *Datura stramonium*, *D. metel*, *Nicotiana glutinosa*, *N. rustica*, and *Trapaolum majus*, preceded by systemic chlorotic spots. TSWV was reported from ornamental crops in Tehran and Absard areas near to

Firouzkoh province (2), but this is the first report of TSWV occurrence on potatoes in Iran.

References: (1) T. S. Ie. *Descriptions of Plant Viruses*. No. 39, 1970. (2) A. A. Moeini, et al. *Iran. J. Plant Pathol.* (In press.)

New Hosts of the Parasitic Flowering Plant, *Alectra vogelii*, in Malawi. P. Subrahmanyam, International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), P.O. Box 1096, Lilongwe, Malawi. *Plant Dis.* 85:442, 2001; published on-line as D-2001-0202-01N, 2001. Accepted for publication 15 January 2001.

Alectra vogelii Benth. (Family: Scrophulariaceae) is a vascular hemiparasite of various leguminous crops in Africa, including peanut (*Arachis hypogaea*), bambara groundnut (*Vigna subterranea*), cowpea (*Vigna unguiculata*), common bean (*Phaseolus vulgaris*), soybean (*Glycine max*), and mung bean (*Vigna radiata*) (1). It is a common parasite of peanut in Angola, Burkina Faso, Malawi, Mozambique, Nigeria, Swaziland, Zambia, and Zimbabwe (2). During April and May 2000, *A. vogelii* was observed parasitizing several wild *Arachis* species in a field at the Chitedze Agricultural Research Station near Lilongwe, Malawi. These species were part of a germ plasm enhancement program that included *A. appressipila* (ICRISAT Groundnut Accession number [ICG] 8127), *A. batizocoi* (ICG 8124), *A. benensis* (ICG 13215), *A. cardenasii* (ICG 13164 and 13166), *A. correntina* (ICG 8918), *A. duranensis* (ICG 13200), *A. helodes* (ICG 8955 and 14917), *A. hoehnei* (ICG 13228), *A. magna* (ICG 8960), *A. pintoii* (ICG 13222 and 14914), *A. stenosperma* (ICG 13172 and 13223), and *A. valida* (ICG 13230). In addition, *A. vogelii* was observed on four unidentified *Arachis* species (ICG 13231, 14875, 14888, and 14907). Parasitized plants were less vigorous and connections between *A. vogelii* and host plants could be observed by carefully removing the soil in the root zone. Mature *A. vogelii* plants were 0.3 to 0.5 m and had multiple stems branching at the base. Subsoil plant parts were a deep orange color. Flowers were prominent lemon yellow with horseshoe-shaped stigmata and leaves were light green. This is the first report of *A. vogelii* parasitizing wild *Arachis* species.

References: (1) C. Parker. *Crop Prot.* 10:6-22, 1991. (2) P. Subrahmanyam. 1997. Parasitic flowering plants. Pages 70-71 in: *Compendium of Peanut Diseases*, 2nd Ed. N. Kokalis-Burelle, D. M. Porter, R. Rodriguez-Kabana, D. H. Smith, and P. Subrahmanyam, eds. American Phytopathological Society, St. Paul, MN.

First Report of Columbia Root Knot Nematode (*Meloidogyne chitwoodi*) in Potato in Texas. A. L. Szalanski, P. G. Mullin, T. S. Harris, and T. O. Powers, Department of Plant Pathology, University of Nebraska, Lincoln, 68583. *Plant Dis.* 85:442, 2001; published on-line as D-2001-0201-01N, 2001. Accepted for publication 18 December 2000.

Columbia root-knot nematode, *Meloidogyne chitwoodi* Golden et al. (1) was identified from potatoes, *Solanum tuberosum* L., collected from Dallam County, Texas in October 2000. Seed potatoes are the most likely source for this introduction. This nematode is currently found infecting potatoes grown in California, Colorado, Idaho, New Mexico, Nevada, Oregon, Utah, and Washington. Some countries prohibit import of both seed and table stock potatoes originating in states known to harbor *M. chitwoodi*. Lesions on the potatoes had discrete brown coloration with white central spots in the outer 1 cm of the tuber flesh. Female nematode densities averaged 3 per square centimeter of a potato section beneath the lesions. Nematodes were morphologically identified as *M. chitwoodi* based on the perineal pattern of mature females and the tail shape of juveniles per Golden et al. (1). Using polymerase chain reaction-RFLP of the rDNA ITS1 region and the mtDNA COII-16S rRNA region (2), individual juveniles were identified as *M. chitwoodi* based on their restriction fragment patterns. This is the first report of Columbia root-knot nematode infecting potatoes in Texas. The distribution of this nematode in potato fields throughout central United States should be determined.

References: (1) A. N. Golden et al. *J. Nematol.* 12:319, 1980. (2) T. O. Powers and T. S. Harris. *J. Nematol.* 25:1, 1993.

Outbreaks of Soybean Frogeye Leaf Spot in Iowa. X. B. Yang, M. D. Uphoff, and S. Sanogo, Department of Plant Pathology, Iowa State University, Ames 50011. *Plant Dis.* 85:443, 2001; published on-line as D-2001-0213-03N, 2001. Accepted for publication 16 January 2001.

Frogeye leaf spot of soybean, caused by *Cercospora sojina*, is typically a disease of warm and humid regions (2). Although the disease was reported in the Midwest in the 1920s (1), no outbreaks have been recorded in Iowa. Outbreaks of frogeye leaf spot occurred during 1999 in soybean fields in Ames and Grand Junction in central Iowa. During the 2000 growing season, the disease occurred in southwestern, south-central, central, southeastern, and east-central Iowa. Occurrences of the disease with severity (reduction of green leaf area) greater than 50% were observed in production soybean fields at Grand Junction in central Iowa and Central City in eastern Iowa. In a 12-ha no-till field planted with cv. Asgrow 2501, the disease was noticeable and uniformly distributed in the entire field in mid July. Disease severity in this field was greater than 70% by the end of August. Disease incidence, however, was less than 10% in three adjacent soybean fields. In a soybean performance test at a central Iowa location where the disease occurred in 1999 and 2000, the disease was observed on all 80 varieties, with four having a severity equal to or greater than 40%. Fourteen entries had less than a 10% disease severity and 19 entries had a disease severity equal to or greater than 30%. Infected leaves in these locations had typical lesions of frogeye leaf spot, which appeared as reddish brown margins surrounding light brown or ash gray centers. On the infected tissues, hyaline, straight, and multiseptate conidia from clustered conidiophores were found, isolated, and identified to *C. sojina*. The relatively warm winter temperatures in 1998 to 1999 and 1999 to 2000 were associated with frogeye leaf spot epidemics. Because of the seedborne nature of *C. sojina*, efforts are warranted to monitor and survey the occurrence of frogeye leaf spot in Iowa, an important seed production state in the northern soybean production region.

References: (1) K. Athow and A. H. Probst. *Phytopathology* 42:660-662, 1952. (2) D. V. Phillips. 1999. Pages 20-21 in: *Soybean Disease Compendium*. Hartman et al. eds, American Phytopathological Society, St. Paul, MN.

Isolation of an Isometric Virus Causing Sunflower Necrosis Disease in India. M. Ramiah, A. I. Bhat, R. K. Jain, R. P. Pant, Y. S. Ahlawat, K. Prabhakar, and A. Varma, Advanced Centre for Plant Virology, Division of Plant Pathology, Indian Agricultural Research Institute, New Delhi 110 012, India. *Plant Dis.* 85:443, 2001; published on-line as D-2001-0205-03N, 2001. Accepted for publication 18 December 2000.

Sunflower necrosis disease (SND) is becoming a potential threat to sunflower (*Helianthus annuus* L.) cultivation in the Indian subcontinent. The disease was first recorded in parts of Karnataka state in 1997. Since then the disease has become increasingly important in Andhra Pradesh, Karnataka, Maharashtra, and Tamil Nadu, the four major sunflower-growing states of India, and is a limiting factor in sunflower production; up to 80% of the plants of some open pollinated and hybrids were affected during the 1999 survey in sunflower growing areas. Field symptoms of the disease include extensive necrosis of leaf lamina, petiole, stem and floral calyx and severe stunting with malformation of flowering head when plants are infected early. The association of a tospovirus, antigenically related to groundnut bud necrosis (GBNV) and watermelon silver mottle (WSMV) viruses, with the disease has been reported (1). However, the etiology of the disease remains unaddressed. In this study a sap-transmissible isometric virus was transferred to cowpea (cvs. Pusa Komal and C152) inciting localized chlorotic and necrotic lesions and systemic veinal necrosis. Electron-microscopic studies of leaf-dip preparations from field samples revealed two types of particles (isometric measuring 25 to 28 nm in diameter and flexuous rods with a length of about 600 nm). The sap-inoculated cowpea and sunflower contained only the isometric particles. Some preparations also showed the presence of tubules containing virus particles. The presence of flexuous particles in field samples could be due to mixed infection as the mosaic disease, known to be caused by a flexuous virus, was common in the sunflower fields surveyed in the present investigations. Extracts

from the field collected samples or sap-inoculated plants did not react with antisera to cucumber mosaic (CMV) or potato Y (PVY) viruses in direct antigen-coated (DAC)-ELISA and immunosorbent electron microscopy tests. The isometric virus isolated from sunflower was purified from sap-inoculated cowpea plants by differential and sucrose density-gradient centrifugations. The virus was sap transmitted back to sunflower (cv. Morden), which developed symptoms identical to those observed under field conditions. Disease symptoms were also reproduced on sunflower upon mechanical inoculation with the purified virus. Polyclonal antiserum raised in rabbits using purified virus preparations, detected the virus from field and glasshouse collected sunflower plants in DAC-ELISA tests. This will help in epidemiological studies and breeding for disease resistance. The particle size and structure and the presence of tubule containing virus particles in plant extracts suggest that the virus belongs to ILAR group. An ILAR virus is reported to infect sunflower (2), but details of its natural occurrence are not known. This is the first report on the etiology of the sunflower necrosis disease in India. Further studies are in progress.

References: (1) Anon. 2000. Annual Report (1999-2000), Indian Agricultural Research Institute, New Delhi, India. (2) A. A. Brunt et al. CAB International, Wallingford, UK, 1210, 1996.

First Report of *Fusarium solani* Causing Stunt on Lisianthus. S. Wolcan, G. Lori, and L. Ronco, CIC, CIDEFI, Facultad de Cs. Agrarias y Forestales, UNLP, 60 y 119, (1900) La Plata, Bs. As., Argentina. *Plant Dis.* 85:443, 2001; published on-line as D-2001-0201-02N, 2001. Accepted for publication 4 January 2001.

Fusarium solani Mart. (Sacc.) is the causal agent of stem rot and damping-off of lisianthus (*Eustoma grandiflorum* (Raf.) Shinn.) (1). Since the end of the 1980s, when this flower crop was introduced in Argentina, it has been affected by a basal stem rot (2). A previously undescribed disease was observed in 100% of the greenhouses in the Buenos Aires Province that grow lisianthus. Symptoms that developed after seedlings were transplanted included stunting, shortened internodes with reduced stem diameter, and small narrow leaves that were a dull green color. Some affected plants turned yellow-brownish and died 2 to 3 months after transplanting. Other plants recovered but produced low quality flowers later than normal. A third group of plants remained stunted (5 to 10 cm high) until the last flower harvest (about 8 to 10 months). *F. solani* was consistently isolated from basal stems and roots of diseased plants. For pathogenicity tests, inoculum was produced by culturing the fungus for 10 days in petri dishes containing sterile moistened rice. Inoculum was air dried, crushed, and mixed with soil that had been autoclaved at 112°C for 40 min on each of two consecutive days. The propagules in the soil were estimated by soil plate dilutions on the Nash & Snyder-PCNB medium at a ratio of about 10⁴ CFU/g soil. Twenty plants of each cultivar Echo White and Echo Blue, whose roots had been pruned, were planted in both infested and noninfested soil. After about 40 days, stunting was observed in 85% of the inoculated plants, while controls remained asymptomatic. *F. solani* was reisolated from symptomatic plants, thus fulfilling Koch's postulates. A test also was conducted in a commercial greenhouse that produced lisianthus for several years, in which healthy plants were planted in three plots fumigated with methyl bromide and in three nonfumigated plots. The mean cfu/g soil of *F. solani* in the methyl-bromide treated plots was 5 × 10² and 1.6 × 10⁴ CFU/g in the nontreated plot. After 120 days, the incidence of stunting in the treated plots was 0.6 and about 88% in the control plots. *F. solani* was recovered from symptomatic plants. Because disinfestation of soil is generally practiced in flower production, stunted plants are limited and can be confused with root problems. This is the first report of *F. solani* causing stunt on lisianthus.

References: (1) J. J. Taubenhaus and W. N. Ezekiel. *Phytopathology* 24:19, 1934. (2) S. M. Wolcan and G. A. Lori. *Invest. Agr. Prot. Veg.* 11:465, 1996.

(Disease Notes continued on next page)

Disease Notes (continued)

First Report of *Arceuthobium hondurense* in Mexico. R. Mathiasen, School of Forestry, Box 15018, Northern Arizona University, Flagstaff, 86011; D. Nickrent, Department of Plant Biology and Center for Systematic Biology, Southern Illinois University, Carbondale 62901; C. Parks, Pacific Northwest Research Station, USDA Forest Service, LaGrande, OR 97850; J. Beatty, Forest Insects and Diseases, USDA Forest Service, Sandy, OR 97055; and S. Sennie, School of Forestry, Box 15018, Northern Arizona University, Flagstaff, AZ 86011. Plant Dis. 85:444, 2001; published on-line as D-2001-0202-04N, 2001. Accepted for publication 25 January 2001.

Honduran dwarf mistletoe (*Arceuthobium hondurense* Hawksw. & Wiens) is a rare dwarf mistletoe previously known only from Honduras (1,2). In March 2000 we collected a dwarf mistletoe from approximately 7 km west of San Cristobal de las Casas, Chiapas, Mexico near Route 190 (elevation 2,440 m), which was morphologically similar to *A. hondurense* (1). This population had initially been classified as *A. nigrum* Hawksw. & Wiens (1). However, our morphological measurements and analysis of nuclear rDNA ITS sequences of *A. hondurense* plants from Honduras (GenBank No. AF325969) and the plants from Chiapas (AF325970) have confirmed that the Chiapan population is *A. hondurense* and not *A. nigrum*. An additional population of *A. hondurense* was discovered in Chiapas approximately 11 km west of Oxchuc near Route 186 (elevation 2160 m). Both of the Chiapan populations of *A. hondurense* were parasitizing *Pinus tecunumanii* (Schw.) Eguiluz et Perry. Specimens of *A. hondurense* from Chiapas were deposited at the Deaver Herbarium, Northern Arizona University, Flagstaff. This is the first report of *A. hondurense* in Mexico and extends its known distribution from northwestern Honduras (3) by approximately 500 km. Although *A. hondurense* has not been observed in the pine forests of Guatemala, it is probable that it also occurs there (1).

References: (1) F. G. Hawksworth and D. Wiens. 1996. Dwarf Mistletoes: Biology, Pathology, and Systematics. USDA Agric. Handb. 709. (2) R. Mathiasen et al. Phytologia 36:211, 1998. (3) R. Mathiasen et al. Plant Dis. 84:372, 2000.

Outbreak of Clover yellow vein virus in a Bean Field in Colusa County, California. R. Crnov and R. L. Gilbertson, Department of Plant Pathology, University of California, Davis 95616. Plant Dis. 85:444, 2001; published on-line as D-2001-0123-01N, 2001. Accepted for publication 21 December 2000.

In 1999, a severe outbreak (i.e., 100% infection) of a virus disease was observed in a single field of common bean in Colusa County, CA. The symptoms included a yellow mosaic, leaf epinasty and, in some plants, a systemic necrosis. This field was adjacent to a clover field that had been harvested early in the development of the bean plants. A preliminary serological test (enzyme-linked immunosorbent assay, ELISA) suggested that the virus infecting these bean plants was *Peanut mottle virus* (PeMoV). This would represent the first report of this virus in California. A range of common bean cultivars (Black Turtle Soup, Topcrop, California Early Light Red Kidney, and Sutter Pink) were inoculated with sap prepared from symptomatic leaves collected from this field. Symptoms developing on these plants ranged from systemic necrosis (cvs. Sutter Pink and Black Turtle Soup) to strong yellow green mosaic and leaf distortion (cvs. Topcrop and California Early Light Red Kidney). Furthermore, inoculated primary leaves of cv. Topcrop failed to develop local lesions, which is characteristic of PeMoV. ELISAs on all symptomatic plants with antisera against PeMoV, BYMV, BCMV, and BCMNV as well as reverse transcription polymerase chain reaction (RT-PCR) analysis with primer pairs specific for PeMoV, BYMV, BCMV, and BCMNV were negative. To further investigate the nature of this virus, a minipurification method was used to purify virions from symptomatic leaves of all four cultivars. Sodium dodecyl sulfate polyacrylamide gel electrophoresis analysis of purified virions from these cultivars revealed a 32-kDa band consistent with infection by a potyvirus. Transmission electron microscopy analysis of these preparations revealed the presence of potyvirus-like flexuous rods (approximately 750 nm long and 10 nm wide). We next designed a primer pair specific for the coat protein gene of *Clover yellow vein virus* (CIYVV) and RT-PCR with these primers resulted in the amplification of a 630-bp DNA fragment from four

isolates of the unknown potyvirus. No fragments were amplified from an uninfected control. The PCR-amplified fragments were direct-sequenced, and sequence comparisons revealed that the sequences of all four isolates were 95% identical to that of CIYVV (Genbank accession number D89541). Subsequently, a CIYVV antiserum was obtained from Simon Scott (Department of Plant Pathology, Clemson University), and ELISAs performed on leaves infected with all four isolates were positive. Finally, to assess whether the virus was seed-transmitted, seed harvested from this field was planted in a greenhouse (two lots of 400 seed each). None of the plants from these seeds developed virus symptoms, suggesting that the virus was not seed-transmitted. Together, these results indicate that the virus disease outbreak in this bean field was caused by CIYVV rather than PeMoV. The inoculum source for the virus was probably the adjacent clover field. This is the first report of CIYVV infecting common bean in California.

***Pythium aphanidermatum* Causing Collar Rot on Papaya in Baja California Sur, Mexico.** G. Rodríguez-Alvarado, Centro de Investigaciones Biológicas del Noroeste (CIBNOR), Apdo. Postal 128, La Paz 23090, Mexico; S. P. Fernandez-Pavia, PICTIPAPA/CEEM, Apdo. Postal 3-12, Metepec 52176, Mexico; J. A. Geraldo-Verdugo, Sueño Tropical, Inc., Pescadero, Mexico; and L. Landa-Hernandez, CIBNOR. Plant Dis. 85:444, 2001; published on-line as D-2001-0213-01N, 2001. Accepted for publication 23 January 2001.

Demand from international markets for organically grown papaya (*Carica papaya* L.) from Baja California Sur is increasing. Occasional rains during the summer of 2000 provided extra moisture to the soil in most papaya farms in the state. Collapsed plants of the Hawaiian type cv. Sunset were observed in 20 commercial orchards in Pescadero, Baja California Sur from July through September 2000. The average disease incidence per orchard was 2%, although there was one orchard with 25% of diseased plants. The initial symptoms were soft, watery lesions at the soil line. As collar rot progressed, foliage wilted. In plants with severe collar rotting, lesions girdled the stem, causing the foliage to be completely wilted and the plants to collapse. Root rot was not observed in plants with collar rot. To isolate the pathogen, 15-cm-long portions of the stem with rot lesions were excised, washed with soap and brush, and rinsed with tap water. Transverse sections of the stem were lightly sprayed with 95% ethanol, and the ethanol was ignited. The superficially burned tissue was removed aseptically, and 1-cm-square sections were cut from the remaining tissue. These sections were plated on potato dextrose agar. The fungus consistently isolated from disease stems grew optimally at 37°C, producing lobate sporangia, antheridia mostly intercalary, and aplerotic oospores characteristic of *Pythium aphanidermatum* (Edson) Fitzp. (3). Pathogenicity studies were conducted twice in a screened house on a total of 36 4 to 6 month-old Sunset papaya plants 85 to 100 cm tall. Two longitudinal wounds (0.5 cm long and 0.2 cm deep) were made on opposite sides at the base of the stem using sterile razor blades. Pathogen inoculum was obtained from 7-day-old V8 agar cultures. Thirty milliliters of an oospore suspension (200 oospores per ml) and V8 agar plugs containing mycelia and oospores were applied next to the crown of wounded and nonwounded plants. Initial symptoms were observed 3 days after inoculation and were similar to those observed on diseased plants in the field. Wounded, pathogen-inoculated plants were dead 6 days after inoculation. *P. aphanidermatum* was reisolated from diseased plants. Nonwounded pathogen-inoculated plants, wounded water-inoculated plants, and nonwounded water-inoculated plants remained healthy throughout the experiments. Pathogenicity experiments suggest that field grown papaya plants might be predisposed to infection by *P. aphanidermatum* due to mechanical damage to the base of the stem caused by abiotic factors such as wind driven sand. *P. aphanidermatum* has been reported to cause root rot in *C. papaya* in Tabasco, Mexico (2), and the United States (1). This is the first report of *P. aphanidermatum* causing collar rot on *C. papaya* in Baja California Sur.

References: (1) Anonymous. 1960. Index of Plant Diseases in the United States. USDA. Handbook. No. 165. Washington, DC. (2) M. I. Saldaña et al. Rev. Mex. Fitopatol. 3:14, 1985. (3) A. J. Van der Plaats-Niterink. Studies Mycol. 21:1, 1981.

Geographic and Host Range of *Meloidogyne* spp. in North Central Mexico. R. Velásquez-Valle, Campo Experimental Pabellón INIFAP, Apdo. Postal 20, Pabellón, Aguascalientes, México, CP20660. Plant Dis. 85:445, 2001; published on-line as D-2001-0122-02N, 2001. Accepted for publication 17 January 2001.

A disease survey carried out in 1998, 1999, and 2000 in the states of Aguascalientes, San Luis Potosí, and Zacatecas revealed the dispersal of *Meloidogyne* spp in this region of Mexico. Pepper (*Capsicum annum* L.) Mirasol type plants showing general chlorosis, root rot, and galls were observed in central Zacatecas and western San Luis Potosí. Dry bean (*Phaseolus vulgaris* L.) plants (Landrace Flor de Mayo) collected in western San Luis Potosí and Aguascalientes also showed root galls. Roots of squash (*Cucurbita* spp) and sunflower (*Helianthus annuus* L.) plants that showed galled roots were found under dryland conditions in northern Zacatecas. Nursery peach (*Prunus persica* L.) plantlets with no foliar symptoms but showing severe root galling were detected in Zacatecas. Perineal patterns of *Meloidogyne* females obtained from those galled roots were coincident with those of *M. incognita* according to pictorial keys (1). This is the first report of *M. incognita* affecting these hosts in that region of the country. Alfalfa (*Medicago sativa*) plants collected in Aguascalientes showed galls caused by *Meloidogyne* spp; this is the first report of this nematode affecting alfalfa in the state. Volunteer onion (*Allium cepa* L., 'Grano Blanco') plants growing in a squash field in eastern Zacatecas had galled roots; a few *Meloidogyne* spp. females were obtained from small galls. This is the first report of the root-knot nematode affecting onion plants in north central México. Onion is known to be a host for several species of this nematode (2). Stunted, chlorotic squash plants had roots severely galled by *Meloidogyne* spp, but pepper crops growing in the same field in previous years showed general chlorosis, reduced size, and poor yield did not have root galls.

References: (1) Eisenback, J. D., et al. 1983. Guía para la identificación de las cuatro especies más comunes del nematodo agallador (*Meloidogyne* spp.) con una clave pictórica. International Meloidogyne Project, Raleigh, NC. (2) Schwartz, H. F., and Mohan, S. K. 1995. Compendium of onion and garlic diseases. American Phytopathological Society, St. Paul, MN.

First Report of *Phytophthora nicotianae* on *Limonium* in Europe. E. Ilieva, Plant Protection Inst., Kostinbröd 2230, Sofia, Bulgaria; W. A. Man in 't Veld, B. F. Wessels-Berk, and R. P. Baayen, Plant Protection Service, P.O. Box 9102, 6700 HC, Wageningen, Netherlands. Plant Dis. 85:445, 2001; published on line as D-2001-0213-02N, 2001. Accepted for publication 3 January 2001.

Limonium (statice or sea-lavendar, family *Plumbaginaceae*) is grown in the Netherlands as a perennial (*Limonium sinense*) or annual (*Limonium sinuatum*) crop. Plants have tufted leaves and numerous clustered flowers of different colors and are used for flower arrangements. In August 2000, we received diseased plants of *L. sinense* cv. Diamond and *L. sinuatum*. Disease symptoms consisted of leaf wilting followed by plant collapse. The base of the leaves showed progressive necrotic areas that later turned dark brown to black. The cortex of the stem and roots was water-soaked and dark brown to black. Longitudinal sections of stems and roots of diseased plants displayed discoloration of tissues. Rotted root tissue was brown with a characteristic black margin. Rotted vascular tissues and other stem parts were also dark brown. Pith parenchyma turned gray-brown and had a firm, wet rot. In plants with advanced disease symptoms, a cavity in the stem parenchyma was observed. Isolations were made from sections of symptomatic leaves, stems and roots of both *Limonium* species on cherry and water agar (WA), followed by incubation at 20°C. *Phytophthora* sp. was isolated consistently from the base of leaves, stems, and roots of diseased plants and identification of isolates was based on morphological characteristics and by isozyme analysis (3). Observations of colony morphology and growth at 35°C were made on V8 agar. Mating type was determined in dual cultures with mating type A2 (*P. nicotianae*, P 1923 [4]) and A1 (*P. nicotianae*, PD98/8/10402). Sporangial features were observed from liquid cultures of the isolates (autoclaved soil-extract or sterile distilled water). All isolates formed colonies consisting of loose, fluffy aerial mycelia. Sporangia and chlamydozoospores were present in all fungal isolates and all isolates were able to grow at 35°C. Few sporangia were produced on solid media (WA and V8 juice agar), but were abundant in liquid cultures. Sporangia were borne singly or in simple sympodial sporangiophores (3 to 4 sporangia), and were ovoid/spherical,

obturinate with rounded base and had prominent papillae (some had two papillae). Sporangia measured 40 to 64 × 24 to 56 µm, (average 50.4 × 38.4 µm) and had an average length:breath ratio of 1.3:1. Chlamydozoospores were terminal and intercalary and measured 18 to 44 µm (average 31.6 µm). Hyphal swellings with hyphal outgrowths were present. Isolates of the fungus were heterothallic and produced oogonia and oospores rapidly and abundantly on V8 agar at 22°C only with the A1 mating type of *P. nicotianae*. We concluded that all isolates from *Limonium* had the A2 compatibility type. Antheridia were amphigynous. Oogonia were spherical and ranged from 20 to 30 µm, (average 27.5 µm). Oospores ranged from 18 to 27 µm, (average 23.1 µm). The observed characteristics are similar to those described for *P. nicotianae*. Isozyme analysis, using the dimeric enzymes malic enzyme (EC 1.1.1.40) and malate dehydrogenase (EC 1.1.1.37), revealed the presence of the *Mdh*¹⁰⁰ allele and the *Mdh*-2¹⁰⁰ allele. Both alleles are characteristic for *P. nicotianae* (3). Based on morphological features and isozyme genotyping, isolates of *Phytophthora* from diseased *Limonium* plants could be assigned to *P. nicotianae* van Breda de haan (1). A report from Florida associated *Phytophthora* sp. with root rot of *Limonium* plants (2) but did not identify the species. According to the multi-decade records at the Netherlands Plant Protection Service (*unpublished data*) *Phytophthora* has never been observed on *Limonium* before. This is the first report of *P. nicotianae* associated with root rot and basal rot of *Limonium* plants in Europe.

References: (1) D. C. Erwin and O. K. Ribeiro. 1996. Phytophthora Diseases Worldwide. American Phytopathological Society, St. Paul, MN. (2) D. F. Farr et al. 1989. Fungi on Plants and Plant Products in the United States. American Phytopathological Society, St. Paul, MN. (3) W. A. Man in 't Veld et al. Phytopathology 88:922-929, 1998. (4) P. Oudemans and M. D. Coffey. Mycol. Res. 95:1025-1046, 1991.

Current Status and New Natural Hosts of Tomato yellow leaf curl virus (TYLCV) in Spain. C. Jordá, I. Font, and P. Martínez, Department Vegetal Production, Plant Pathology, Universidad Politécnica, Cno. Vera, 14, Valencia, Spain; M. Juárez and A. Ortega, Department Vegetal Production, Universidad Miguel Hernández, Orihuela, Alicante, Spain; and A. Lacasa, Centro Investigación Desarrollo Agrario, Murcia, Spain. Plant Dis. 85:445, 2001; published on-line as D-2001-0219-04N, 2001. Accepted for publication 11 December 2000.

Tomato yellow leaf curl virus (TYLCV) is a major constraint to tomato production in Spain. This virus was observed for the first time in several tomato fields in Murcia (Spain) in the autumn of 1992 and Canary Islands in 1999. Currently the virus is prevalent along the Mediterranean coast of Spain (provinces of Málaga, Granada, Almería, Murcia, Alicante, Valencia, and Barcelona) and in the Canary Islands. Two viral species have been identified in Spain, TYLCV-Sar in 1992 and TYLCV-Is in 1997. TYLCV-Is is more severe than TYLCV-Sar and produces the greatest economic losses. Curling of leaflets, yellowing, and growth reduction are more pronounced in plants infected with TYLCV-Is than in those infected with TYLCV-Sar. In order to study the presence and behavior of both viral species in the affected area, over 1,320 tomato plants were sampled. DNA was extracted from the samples and analyzed by polymerase chain reaction (PCR) amplification. The degenerate primer pair for Begomovirus detection (AV494/AC1048) (2) was used to amplify the core region of the capsid protein gene. The amplified fragments were later analysed by restriction fragment length polymorphism (RFLP) with *Hae*III enzyme to differentiate between TYLCV-Is and TYLCV-Sar species. The results showed that TYLCV-Sar (43.4%) and TYLCV-Is (56.6%) coexist in tomato crops and, in contrast with previous results (1), displacement of TYLCV-Sar for TYLCV-Is was observed. A search for the alternative hosts that may serve as virus reservoirs in areas where the virus is prevalent involved testing 210 samples of 95 species of weeds by PCR, with the same primers. The following species were found to be infected: *Conyza sumatrensis* (Retz.) E. Walker, *Convolvulus* sp., *Cuscuta* sp., *Chenopodium murale* L., *Datura stramonium* L., *Dittrichia viscosa* (L.) W. Greuter, *Malva parviflora* L., and *Solanum nigrum* L. This is the first reference of *C. sumatrensis*, *Convolvulus* sp., *Cuscuta* sp., and *Ch. murale* as natural hosts of TYLCV. These plants were symptomless.

References: (1) S. Sanchez-Campos et al. Phytopathology 89:1038, 1999. (2) S. D. Wyatt et al. Phytopathology 86:1288, 1996.

(Disease Notes continued on next page)

Disease Notes (continued)

First Report of Barley yellow striate mosaic virus Infecting Barley and Wheat in Lebanon. K. M. Makkouk, W. Ghulam, and S. G. Kumari, Virology Laboratory, Germplasm Program, International Center for Agricultural Research in the Dry Areas (ICARDA), P. O. Box 5466, Aleppo, Syria. *Plant Dis.* 85:446, 2001; published on-line as D-2001-0205-01N, 2001. Accepted for publication 25 January 2001.

Symptoms suggestive of virus infection in barley, bread wheat, and durum wheat were observed at high incidence in November 2000 in Terbol, Beqa'a Valley, Lebanon. The symptoms were mainly stunting, accompanied by leaf striping and yellowing. Symptomatic plant samples (27 barley, 37 bread wheat, and 81 durum wheat) were collected and tested for the presence of four different viruses by tissue-blot immunoassay (TBIA) (1) at the Virology Laboratory of ICARDA, Aleppo, Syria. Antisera used were for *Barley stripe mosaic virus* (BSMV, genus *Hordeivirus*) (2); *Barley yellow dwarf virus* (BYDV, genus *Luteovirus*, family *Luteoviridae*) (PAV serotype) (2); *Wheat streak mosaic virus* (WSMV, genus *Tritimovirus*, family *Potyviridae*) (3); and *Barley yellow striate mosaic virus* (BYSMV, genus *Cytorhabdovirus*, family *Rhabdoviridae*) provided by M. Conti, Instituto di Fitovirologia applicata, Turino, Italy. BYSMV was detected in 12 barley, 18 bread wheat, and 56 durum wheat samples; the corresponding numbers of barley, bread wheat, and durum wheat plants testing positive for BYDV-PAV were 4, 7, and 6, respectively. BSMV and WSMV were not detected in any of the samples tested. BYSMV was purified from infected wheat plants, and the purified preparation had a UV 260:280 ratio of 1.18, typical of Rhabdoviruses. In SDS-polyacrylamide gel electrophoresis, the purified virus preparation indicated the presence of 66, 47, and 15 kDa structural proteins, typical of the G, N and M proteins of Rhabdoviruses. In western blot, the 66 and 47 kDa protein bands reacted strongly with BYSMV antiserum. This is the first record of BYSMV infecting barley and wheat in Lebanon.

References: (1) K. M. Makkouk and A. Comeau. *Eur. J. Plant Pathol.* 100:71, 1994. (2) K. M. Makkouk and S. G. Kumari. *Rachis Newsl.* 12:24, 1993. (3) K. M. Makkouk and S. G. Kumari. *Rachis Newsl.* 16:74, 1997.

First Report of *Nectria haematococca* Stem Girdling of Greenhouse Peppers in Florida. E. Lamb, University of Florida, Institute of Food and Agricultural Sciences, Indian River Research and Education Center, Fort Pierce 34945; E. Roskopf, USDA-ARS, U.S. Horticultural Research Laboratory, Fort Pierce, FL 34945; and R. M. Sonoda, University of Florida, Institute of Food and Agricultural Sciences, Indian River Research and Education Center, Fort Pierce 34945. *Plant Dis.* 85:446, 2001; published on-line as D-2001-0213-04N, 2001. Accepted for publication 10 January 2001.

Nectria haematococca Berk. & Broome causing stem girdling of three cultivars of greenhouse pepper, *Capsicum annuum* (cvs. Kelvin, Cubico, and Grizzly), was found for the first time in a single greenhouse in south Florida in March 1999. Approximately 10% of the plants were affected at first report increasing to over 40% within 3 months. Black lesions occurred at nodes where the plant was pruned or where fruit had been harvested. No mycelium or perithecia were noted in association with the lesions. All tissue above a lesion appeared normal until the lesion girdled the stem, causing the tissue above the lesion to wilt and die. The plant appeared unaffected below the lesion. The pathogen was isolated on half-strength Difco potato-dextrose agar (½ DPDA). Reddish perithecia developed readily in culture. Two single spore isolates of the pathogen obtained from two naturally infected plants (cultivar Kelvin) were used to satisfy Koch's postulates. Five plants of Kelvin were inoculated with each isolate by inserting a 4-mm agar block of the pathogen grown for 5 days on ½ DPDA into the stem. Five plants of the same cultivar were similarly treated with fungus-free ½ DPDA. Plants were grown under greenhouse conditions after inoculation. In four plants, black lesions similar to those seen in the commercial greenhouse developed within 1 week. In one plant, the portions of the plants above the point of inoculation wilted after 5 days. The upper parts of the plants appeared healthy until lesions girdled the stems. The plants treated with fungus-free agar remained healthy. The fungus was re-isolated from the margins of lesions on the inoculated plants. The pathogen has been reported to

cause stem lesions and fruit rot of pepper in greenhouses in England (1) and Canada (2). Fruit symptoms were not observed in the Florida greenhouse. Stem symptoms were again reported from the same greenhouse in the following season.

References: (1) J. T. Fletcher. *Plant Pathol.* 43:225-222, 1994. (2) W. R. Jarvis. *Can. Plant Dis. Surv.* 74:131-134, 1994.

First Report of *Sclerotinia sclerotiorum* on *Calendula officinalis* in Italy. A. Garibaldi, A. Minuto, and M. L. Gullino, D.I.V.A.P.R.A. Patologia vegetale, Via Leonardo da Vinci 44, 10095 Grugliasco, Italy. *Plant Dis.* 85:446, 2001; published on-line as D-2001-0205-02N, 2001. Accepted for publication 19 January 2001.

Pot marigold (*Calendula officinalis*) has recently become popular as a potted ornamental plant in Italy. During the spring 1999, a sudden wilt of 120 day-old plants was observed in the Albenga region of Northern Italy, an area of intensive floriculture production. Initial symptoms included stem necrosis at the soil line and yellowing and tan discoloration of leaves. As stem necrosis progressed, infected plants wilted and died. Necrotic tissues resulted, covered with whitish mycelium that produced dark, spherical (2- to 6-mm diameter) sclerotia. *Sclerotinia sclerotiorum* was consistently recovered from symptomatic stem sections surface disinfested 1 min in 1% NaOCl and plated on potato dextrose agar (PDA), amended with 100 ppm streptomycin sulfate. Pathogenicity of three isolates was confirmed by inoculating 90-day-old pot marigold plants grown in containers. Inoculum that consisted of wheat kernels infested with mycelium and sclerotia was placed on the soil surface around the base of previously wounded or non-wounded plants. Non-inoculated plants served as controls. All plants were kept outdoors where temperatures ranged between 8 and 16°C, under 50% shade and were maintained moist. Inoculated plants developed symptoms of leaf yellowing, followed by wilt within 7 days, while control plants remained symptomless. Sclerotia developed on infected tissues and *S. sclerotiorum* was reisolated from inoculated plants. This is the first report of stem blight of *C. officinalis* caused by *S. sclerotiorum* in Europe. The disease was previously observed in the United States (1).

Reference: (1) D. F. Farr et al. 1989. *Fungi on Plants and Plant Products in the United States.* American Phytopathological Society, St. Paul, MN.

First Report of Artichoke Downy Mildew Caused by *Bremia lactucae* in Argentina. M. Carranza, S. Larran, and H. Alippi; CIDEFI, Facultad de Cs. Agrarias y Forestales, UNLP, 60 y 119, (1900) La Plata, Argentina and Comisión de Investigaciones Científicas (CIC). *Plant Dis.* 85:446, 2001; published on-line as D-2001-0219-02N, 2001. Accepted for publication 3 January 2001.

In 1999, downy mildew was detected on artichoke (*Cynara scolymus* L.) plants from La Plata, Buenos Aires Province. The disease was observed on various commercial varieties. Symptoms were angular interveinal chlorotic spots less than 3 cm in size. These infected areas, although not confluent, covered a wide surface and caused early death of the leaves. On the undersides of these lesions, white-grayish sporulation was abundant, consisting of sporangiophores with dichotomous branches, widened in their peaks with 2 to 7 terminal sterigmata. Sporangia were ellipsoidal, hyaline and 14 to 30 × 12 to 25 µm in size. Oospores were not observed in leaf tissues. The pathogen was identified as *Bremia lactucae* Regel (1). Pathogenicity was confirmed with the inoculation of healthy artichoke plants. They were incubated in a humidity chamber at 10 to 15°C, and after 16 days chlorotic spots and downy mildew colonies developed. The presence of *B. lactucae* was confirmed by macro- and microscopic observation and Koch's postulates were fulfilled. This is the first report of downy mildew on artichoke in Argentina. Because it is widespread in the most important artichoke-growing area in Argentina (2), downy mildew should be considered in the cultural and sanitary management of the crop.

References: (1) P. Corda. *Hypermedia Prot. Plantae*, INRA, 1995. (2) A. Ricceti et al. *Bol. Hortic.* 4:4, 1996.

First Report of *Colletotrichum acutatum* on *Kalmia*. S. R. H. Langrell and S. J. Irvine, Plant Health Section, Scottish Agricultural Science Agency, East Craigs, Edinburgh, EH12 8NJ, Scotland, United Kingdom. *Plant Dis.* 85:447, 2001; published on-line as D-2001-0219-03N, 2001. Accepted for publication 1 February 2001.

Colletotrichum acutatum J. H. Simmonds was isolated from diseased leaves of ornamental *Kalmia latifolia* L. (mountain laurel) cvs. Carousel and Peppermint on plants imported from the United States to Edinburgh, Scotland, in December 1999. Symptoms included sunken, desiccated, darkened necrotic areas, primarily at the leaf tip. Necrotic areas advanced toward the leaf base and were bordered by purple/red pigmentation. Isolations were made from salmon colored conidiomata that developed on abaxial leaf surfaces following incubation in a humidity box at 25°C for 7 to 10 days. White aerial mycelia, becoming gray to grayish beige, and producing salmon to orange colored conidial masses, formed on potato dextrose agar after 10 to 14 days. Conidia were hyaline, aseptate, fusiform to slightly irregular, and measured 13.4 to 13.8 × 4.3 to 4.9 µm. Both morphological and conidial characteristics were consistent with the description of *C. acutatum* (2). The identity of isolates was further verified by positive plate-trapped antigen ELISA of conidial preparations using a species-specific monoclonal antibody (1). Pathogenicity was assessed by inoculating the adaxial surface of healthy leaves of both cultivars of the imported plants with colonized agar disks and a range of spore suspensions (30, 300, and 3,000 spores delivered in 30 µl volumes) from test fungal isolates and a confirmed laboratory strain (three replicates per treatment). To ensure inoculum uptake, two 5 mm² areas of cuticle on either side of the mid-rib of each leaf were lightly scratched with a sterile hyperdermic needle prior to inoculation. Inoculated leaves were incubated in a humidity box at 25°C for up to 3 weeks. Symptom development was progressive but relatively slow on both cultivars. The relatively slow development on artificially infected leaf material may be partly attributable to residual fungicide treatment as prescribed by the Scottish Plant Health Service at the time of planting out. Symptoms produced on fruits (apple, banana, and strawberry), inoculated with both test and laboratory strains of the fungus, were identical. Symptoms did not occur on control leaves or fruits inoculated with sterile distilled water or uninoculated agar disks. Koch's postulates were confirmed by consistently reisolating isolates with morphological and immunological characteristics identical to the fungal isolate used to initially inoculate test material. Over the same period, additional symptoms, identical to those originally described at the time of interception, continued to develop on leaf tips of both *Kalmia* cultivars. Additional isolations from this material were characterized as *C. acutatum*. Identification of representative isolates was confirmed by CABI Bioscience, Egham, UK, where a reference culture (accession number IMI 384569) has been deposited. As advanced symptoms were observed immediately on arrival of this consignment in the UK, original infection is thought to have occurred prior to importation. This is the first report of *C. acutatum* infecting *K. latifolia*.

References: (1) I. Barker et al. 1994. Pages 179-182 in: *Assays for Plant Pathogenic Fungi: Identification, Detection and Quantification*. A. Schots, F. M. Dewey, and R. Oliver, eds. CABI International, Wallingford, UK. (2) B. J. Dyko and J. E. M. Mordue. *CMI Descriptions of Pathogenic Fungi and Bacteria*. No. 630, 1979.

First Report of an Aster Yellows Phytoplasma Associated with Cabbage in Southern Texas. I.-M. Lee and R. A. Dane, Molecular Plant Pathology Laboratory, USDA, ARS, Beltsville, MD 20705; and M. C. Black and Noel Troxclair, Texas A&M University Research and Extension Center, Uvalde 78802-1849. *Plant Dis.* 85:447, 2001; published on-line as D-2001-0202-03N, 2001. Accepted for publication 10 January 2001.

In early spring 2000 carrot crops in southwestern Texas were severely infected by an outbreak of phyllody associated with aster yellows phytoplasma. Cabbage crops that had been planted adjacent to these carrot fields began to display previously unobserved symptoms characteristic of phytoplasma infection. Symptoms included purple discoloration in leaf veins and at the outer edges of leaves on cabbage heads. Proliferation of sprouts also occurred at the base of the stem and

between leaf layers of some plants, and sprouts sometimes continued to proliferate on extended stems. About 5% of cabbage plants in the field exhibited these symptoms. Two symptomless and four symptomatic cabbage heads were collected in early April from one cabbage field. Veinal tissues were stripped from each sample and used for total nucleic acid extraction. To obtain specific and sufficient amount of PCR products for analysis, nested PCR was performed by using primer pairs (first with P1/P7 followed by R16F2n/R16R2) (1,2) universal for phytoplasma detection. A specific 16S rDNA fragment (about 1.2 kb) was strongly amplified from the four symptomatic but not from the two asymptomatic samples. The nested PCR products obtained from the four symptomatic samples were then analyzed by restriction fragment length polymorphism (RFLP) using the restriction enzymes *MseI*, *HhaI*, and *HpaII*, and the RFLP patterns were compared to the published patterns of known phytoplasmas (1). The resulting RFLP patterns were identical to those of a phytoplasma belonging to subgroup B of the aster yellows phytoplasma group (16SrI). These RFLP patterns were also evident in putative restriction sites observed in a 1.5 kbp nucleotide sequence of the 16S rDNA. This is the first report of aster yellows phytoplasma associated disease symptoms in cabbage in Texas. The occurrence of cabbage proliferation coincided with the presence of high populations of the insect vector, aster leafhopper.

References: (1) I.-M. Lee et al. *Int. J. Syst. Bacteriol.* 48:1153, 1998. (2) B. Schneider et al. 1995. *Molecular and Diagnostic Procedures in Mycoplasmaology*, Vol. 1. Academic Press, San Diego, CA.

Epidemic of Potato virus Y and Cucumber mosaic virus in Henan Province Tobacco. X. D. Li, Department of Plant Protection, Shandong Agricultural University, Tai'an, Shandong 271018, China; Y. Q. Li, Qingzhou Tobacco Institute of CNTC, Qingzhou, Shandong 262500, China; H. G. Wang, Department of Agronomy, Shandong Agricultural University, Tai'an, Shandong 271018, China. *Plant Dis.* 85:447, 2001; published on-line as D-2001-0219-01N, 2001. Accepted for publication 2 January 2001.

Flue-cured tobacco is an important crop in Henan Province, China. During the 2000 growing season, many tobacco plants showed various degrees of mottling, mosaic, vein clearing, or vein necrosis in most of the counties. Some plants even died at an early stage of growth. A survey was conducted in May-June in several tobacco-growing counties, and the incidence of symptomatic plants in individual fields ranged from 10 to 85%. The most widely planted tobacco varieties, NC89, K326, and K346, were highly susceptible. Symptomatic plants were collected from Jiaxian and Xiangcheng counties and samples were tested by enzyme-linked immunosorbent assay for Tobacco mosaic virus (TMV), Cucumber mosaic virus (CMV), Potato virus Y (PVY), and Potato virus X (PVX). Of 65 samples tested, 21 were positive for only PVY, 16 positive for only CMV, one each was positive for only TMV or PVX. Nineteen samples were doubly infected with various combinations of these viruses and six were infected with combinations of three viruses. The causal agent(s) in the remaining sample could not be determined. In total, CMV was detected in 40 samples, PVY in 38, PVX in 10, and TMV in 7 samples. TMV and CMV used to be the most important viruses and PVY occurred only rarely. But PVY has become prevalent in Henan and in neighboring Shandong province (2). CMV and TMV were reported to be the most prevalent viruses in Shanxi (1) and Fujian Provinces (3). Because resistant varieties are not available, and mixed infections are more common, the results presented here explain why huge damage is occurring in tobacco crops in recent years. Some varieties are partially resistant to TMV and CMV but the varieties commonly grown are highly susceptible to PVY. Therefore, breeding for resistance to viruses, especially to PVY, is urgent to control the occurrence of tobacco viral diseases.

References: (1) J. L. Cheng et al. *Acta Tabacaria Sin.* 4:43, 1998. (2) J. B. Wang et al. *Chinese Tobacco Sci.* 1:26, 1998. (3) L. H. Xie et al. *Acta Tabacaria Sin.* 2:25, 1994.

(Disease Notes continued on next page)

Disease Notes (continued)

First Report of a Member of Aster Yellows Phytoplasma Group and of Clover Proliferation Phytoplasma Group Associated with Onion in Texas. I.-M. Lee and R. A. Dane, Molecular Plant Pathology Laboratory, USDA, ARS, Beltsville, MD 20705; and M. C. Black, Texas A&M University Research and Extension Center, Uvalde 78802-1849. Plant Dis. 85:448, 2001; published on-line as D-2001-0202-02N, 2001. Accepted for publication 25 January 2001.

An unknown disease(s) emerged this spring (2000) in an onion field in southwestern Texas. Infected onion plants exhibited two symptom types, one with shoot proliferation, moderate stunting of plants, and light yellowish discoloration on leaves (A) and the other with only severe stunting of the plants (B). The bulbs of the infected plants collected from both symptom types were smaller than normal. When the aerial shoots were trimmed, the infected (but not asymptomatic or the severely stunted) bulbs produced multiple slender sprouts after storage in room temperature for about a month. These symptoms are characteristic of yellows diseases caused by phytoplasmas. Ten symptomatic (six with symptom type A and four with symptom type B) and ten symptomless onion plants were collected in early May from an onion field about 1 to 2 weeks prior to blooming. Total nucleic acid was extracted from 0.5 g of shoot tissues from each sample. Nested polymerase chain reaction (PCR) using universal primer pairs (P1/P7 followed by R16F2n/R16R2) previously designed based on 16S and 23S rRNA gene sequence (1,2) was employed for the detection of phytoplasma(s) present in the samples. Specific PCR products (all were about 1.2 kb) were heavily amplified from five samples with symptom type A and one with symptom type B. Three of the symptomatic plants showing symptom type B and five of the symptomless samples were scored as weak positives. Restriction fragment length polymorphism (RFLP) analyses of the PCR products obtained from all five symptomatic samples with symptom type A using restriction enzymes including *Mse*I, *Hha*I, and *Hpa*II revealed that the associated phytoplasmas detected belonged to aster yellows phytoplasma group (16SrI), subgroup A. RFLP analyses of PCR product from the sample with symptom type B indicated that the associated phytoplasma belonged to clover proliferation group (16SrVI), subgroup A (1). Since symptom type A resembles onion yellows reported elsewhere, we propose to adopt "onion yellows" to refer to the new onion disease occurring in Texas. However, correlation between a member of clover proliferation phytoplasma group and onion plants showing severe stunting could not be firmly established. A phytoplasma belonging to 16SrI-B is associated with onion yellows disease reported in Japan (1). This is the first report that onion yellows occurs in the United States.

References: (1) I.-M. Lee et al. Int. J. Syst. Bacteriol. 48:1153, 1998. (2) B. Schneider et al. 1995. Molecular and Diagnostic Procedures in Mycoplasma, Vol. I, Academic Press, San Diego, CA.

***Spathiphyllum* sp.: A New Natural Host of *Impatiens necrotic spot virus*.** A. Materazzi and E. Triolo, Dipartimento di Coltivazione e Difesa delle Specie Legnose, Università di Pisa, 56184 Pisa, Italy. Plant Dis. 85:448, 2001; published on-line as D-2001-0221-02N, 2001. Accepted for publication 17 January 2001.

In September 1999, several *Spathiphyllum* plants grown in a greenhouse in Tuscany (Italy) showed leaf symptoms in the form of concentric chlorotic ringspots, line patterns, and irregular chlorotic

blotches. These symptoms developed into localized necrosis. Crude sap of tissues showing symptoms was mechanically inoculated to young symptomless *Spathiphyllum* plants and to *Nicotiana benthamiana* and *N. clevelandii*. Samples drawn from symptomatic and symptomless tissues of naturally or artificially infected *Spathiphyllum* and *Nicotiana* plants were tested for the presence of *Alfalfa mosaic virus* (AMV), *Arabidopsis mosaic virus* (ArMV), *Cucumber mosaic virus* (CMV), *Dasheen mosaic virus* (DsMV), *Impatiens necrotic spot virus* (INSV), *Potato X virus* (PVX), *Potato Y virus* (PVY), *Tobacco mosaic virus* (TMV), and *Tomato spotted wilt virus* (TSWV) by double-antibody sandwich enzyme-linked immunosorbent assay carried out with commercial antisera. The symptomatic tissues obtained from *Spathiphyllum* and *Nicotiana* plants gave a positive reaction only for INSV. The symptomless samples obtained from various parts of the infected *Spathiphyllum* plants gave a negative reaction, even after 1 year from the appearance of localized necrosis, suggesting a non-systemic infection in this new host. This is the first report of infection of *Spathiphyllum* sp. by INSV.

First Report of a Root and Crown Rot Disease of Myrtle in California Caused by *Cylindrocladium pauciramosum*. S. T. Koike, University of California Cooperative Extension, Salinas 93901; P. W. Crous, Department of Plant Pathology, University of Stellenbosch, P. Bag X1, Matieland 7602, South Africa. Plant Dis. 85:448, 2001; published on-line as D-2001-0221-01N, 2001. Accepted for publication 1 February 2001.

Myrtle (*Myrtus communis*) is a woody, evergreen plant used in California as a landscape shrub or potted plant. In 2000, a new root and crown disease was found in commercial nursery myrtle being grown as potted plants. Roots were necrotic and crown tissue was brown. Affected plants became gray-green in color, withered, and died. A *Cylindrocladium* sp. was consistently isolated from roots, crowns, and lower stems of symptomatic plants. Isolates were characterized by having penicillate conidiophores terminating in obpyriform to broadly ellipsoidal vesicles. Conidia were hyaline, 1-septate, straight with rounded ends, (50-) 53 to 56 (-58) × (3.5-) 4 to 6 μm, placing it in the *Cylindrocladium candelabrum* Viégas species complex. Single-conidial isolates (STE-U 4012 to 4018) produced perithecia with viable progeny of *Calonectria pauciramosa* C.L. Schoch & Crous when mated on carnation leaf agar with tester strains of *Cylindrocladium pauciramosum* C.L. Schoch & Crous (2). Matings with tester strains of all other species in this complex proved unsuccessful. Only one mating type of *C. pauciramosum* has thus far been found in the United States. Pathogenicity of representative isolates was confirmed by applying 5 ml of a conidial suspension (1.0 × 10⁶ conidia/ml) to the crowns of potted, 5-month-old, rooted myrtle cuttings that were subsequently maintained in a greenhouse (23 to 25°C). After 4 weeks, plant crowns and roots developed symptoms similar to those observed in the nursery, and plants later wilted and died. *C. pauciramosum* was re-isolated from all plants. Control plants, which were treated with water, did not develop any symptoms. The tests were repeated and the results were similar. This is the first report of *C. pauciramosum* as a pathogen of myrtle in California. The disease has been reported on myrtle in Europe (1).

References: (1) G. Polizzi and P. W. Crous. Eur. J. Plant Pathol. 105:407, 1999. (2) C. L. Schoch et al. Mycologia 91:286, 1999.