First Report of *Colletotrichum destructivum* on Curly Dock. H. B. Lee and C.-J. Kim, Advanced Biomaterials Research Center, Korea Research Institute of Bioscience and Biotechnology, Yusong, Taejon 305-600. Plant Dis. 86:1271, 2002; published on-line as D-2002-0906-01N, 2002. Accepted for publication 14 August 2002.

Curly dock (*Rumex crispus* L.) is a perennial deciduous plant in the family Polygonaceae. It is widely distributed in grasslands and orchards and is an important weed that is traditionally used as a medicinal herb. During the summers of 2000 and 2001, a severe anthracnose disease was observed on leaves of mature curly dock in the foothills near the western coastal area of Muchangpo, Ungchon, the district of Chungnam in Korea. Initial symptoms usually appeared in June as a small number of slightly soaked spots on leaves. Typical symptoms, generally observed in late July and August following a long, rainy, hot period, consisted of a number of brown leaf spots that expanded and often twisted, resulting in discoloration of whole parts to blackish brown or slightly reddish brown and defoliation. A fungus, which was isolated from the leaf lesion, was identified as *Colletotrichum destructivum* O'Gara based on previous descriptions (1,3). The fungus was characterized by conidia, which were long, relatively narrow, and straight to slightly curved with abruptly tapered and obtuse ends, complex appressoria, and cultures with apricot- to salmon-colored sectors that lacked sclerotia. Conidial size ranged from 4.0 to 6.4 µm (average 4.8) × 10 to 23 µm (average 16.5). Setae were slender and straight but frequently flexuous, subulate, brown, and variable in length. *C. destructivum* has a teleomorph, *Glomerella glycines* (Hori) Lehman & Wolf, but the species is not well known, and the connection has not been studied in detail. The isolate has been deposited in the IMI Culture Collection as isolate IMI387103. The dimensions of conidia from the isolate matched those of *C. destructivum* N150 (GenBank Accession No. AF325064) isolated from *Nicotiana tabacum* (3). *C. destructivum* is distinguishable from *C. gloeosporioides*, whose spores are short and cylindrical with obtuse apices tapering slightly to a truncate base. Pathogenicity of the isolate was determined on 5-week-old leaves of curly dock. Leaves were inoculated with a conidial suspension of the fungus (approximately 1×10^6 conidia per ml), placed in a moist chamber for 3 days, and subsequently transferred to a growth chamber maintained at 25°C. Within 7 days after inoculation, symptoms appeared that were similar to those originally observed on leaflets. Uninoculated control (sprayed only with distilled water) leaves exposed to the same environmental conditions remained healthy. *C. destructivum* was consistently reisolated from infected leaves. *C. destructivum* has been reported as a pathogen on approximately 15 genera, including *Medicago sativa*, *Trifolium* spp., *Cuscuta* spp., and *N. tabacum* (1,3), and two fungal species, *C. erumpens* and *C. rumicis-crispi*, have been reported to cause anthracnose on *R. crispus*. To our knowledge, *R. crispus* represents a previously unreported host for *C. destructivum* causing anthracnose, although *C. gloeosporioides* has been reported as a pathogen of *R. crispus* in Korea (2).

References: (1) A. P. Baxter et al. S. Afr. Tydskr. Plantk. 2:259, 1983. (2) B. S. Kim et al. Korean J. Plant Pathol. 14:358, 1998. (3) S. Shen et al. Mycol. Res. 105:1340, 2001.

First Report of *Silybum marianum* as a Host of *Puccinia punctiformis*. D. K. Berner, L. K. Paxson, W. L. Bruckart, D. G. Luster, M. McMahon, and J. L. Michael, United States Department of Agriculture, Agricultural Research Service, Foreign Disease-Weed Science Research Unit, 1301 Ditto Avenue, Fort Detrick, MD 21702. Plant Dis. 86:1271, 2002; published on-line as D-2002-0910-01N, 2002. Accepted for publication 29 August 2002.

Silybum marianum (L.) Gaertn. (milk thistle) is a problematic invasive weed in the western United States. The rust fungus, *Puccinia punctiformis* (F. Strauss) Rohl., is found throughout the world as a pathogen of *Cirsium arvense* (L.) Scop. (Canadian thistle). Recently, plants of *S. marianum* grown from surface-disinfested seeds in our quarantine greenhouse were parasitized by a rust. Apparently, an isolate of *P. punctiformis* collected from *C. arvense* in Turkey that was present in the greenhouse had spread to adjacent *S. marianum* plants and caused infection without applying any artificial dew period. Ribosomal internal transcribed spacer region sequences from fungal spore DNA isolated from the two hosts were identical. Initial signs on *S. marianum* were abundant, fragrant spermogonia on large leaves. These signs occur on secondary shoots of *C. arvense* and are indicative of systemic fungal infection (1). As the fungus infection developed on *S. marianum*, uredinia and urediniospores were produced. Sori on older leaves also produced teliospores. Urediniospores from infected leaves were harvested and sprayed uniformly on eight 17-day-old plants of *S. marianum* grown in isolation from *P. punctiformis*. The spore suspension consisted of 4 mg urediniospores suspended in 40 ml distilled water. Inoculated plants were incubated for 18 h in a dew chamber at 20°C in the dark and transferred to a greenhouse (20 to 25°C, 30 to 50% relative humidity, and natural light). After 13 days, uredia with urediniospores developed on four of the plants. Using the same procedure, inoculations were repeated on plants of *S. marianum* and *S. eburneum* Coss. & Durieu (the only other species described in the genus) with urediniospores of a domestic isolate of the fungus from *C. arvense* in Maryland. Of 51 inoculated plants of *S. marianum*, 23 became infected and produced uredinia. None of the 12 inoculated plants of *S. eburneum* showed symptoms of infection. In nature, *C. arvense* and *S. marianum* occupy different ecological areas. *C. arvense* is found predominately in humid temperate habitats, while *S. marianum* is found in habitats with a dry Mediterranean climate. Life cycles of each host are also different. *C. arvense* is a perennial that emerges in spring and dies back in winter, while *S. marianum* is a winter annual that emerges in fall and dies in late spring. Because of the differences in life cycles combined with the different geographical distribution, *P. punctiformis* from *C. arvense* may rarely encounter susceptible *S. marianum* plants in the field. Since fungal spores can be produced routinely on artificially inoculated plants, there might be potential to use *P. punctiformis* for biological control of *S. marianum*. To our knowledge, this is the first report of *S. marianum* as a host for *P. punctiformis*.

Reference: (1) A. H. R. Buller. *Puccinia sauveolens* and its sexual process. Page 345 in: Researches on Fungi. Vol VII. The Sexual Process in the Uredinales, Toronto, Canada, 1950.

Natural Occurrence of *Groundnut ringspot virus* on Soybean in South Africa. G. Pietersen, ARC-Plant Protection Research Institute, Private Bag X134, Pretoria, South Africa; and J. Morris, Central Science Laboratory, Sand Hutton, York, Y041 1IZ, U.K. Plant Dis. 86:1271, 2002; published on-line as D-2002-0905-03N, 2002. Accepted for publication 25 August 2002.

Mechanically transmissible viruses were isolated from two soybean (*Glycine max* Merr.) plants from Rustenburg, Northwest Province and Greytown, KwaZulu-Natal, South Africa, respectively (2). Viruses were isolated by two serial local-lesion transmissions on *Chenopodium quinoa*. Ringspot symptoms on *Nicotiana benthamiana* suggested the presence of tospoviruses. This was supported by the detection of typical tospovirus-like particles in ultrathin sections of infected plants. Serological analysis of samples using various tospovirus antisera in a number of enzyme-linked immunosorbent assay formats suggested the two isolates had epitopes in common with *Tomato spotted wilt virus* (TSWV), *Groundnut ringspot virus* (GRSV), and *Tomato chlorotic spot virus* (TCSV). Nucleotide sequences were determined using reverse transcription-polymerase chain reaction (RT-PCR) for 857 bp of the nucleoprotein gene of the two isolates (GenBank Accession Nos. AF487516 and AF 487517). These revealed a 99% nucleotide identity with each other. Sequence comparison with cognate regions of TSWV (GenBank Accession No. D00645), GRSV-SA-05 (GenBank Accession No. S54327), and TCSV (GenBank Accession No. S54325) revealed that both isolates share 97% nucleotide sequence identity with GRSV (SA-05) from peanut also originally from South Africa (1). Both isolates are therefore considered GRSV. To our knowledge, this is the first report of the natural occurrence of GRSV on soybean worldwide.

References: (1) A. C. de Avila et al. J. Gen. Virol. 74:153, 1993. (2) G. Pietersen et al. Afr. Plant Prot. 4:65, 1998.

(*Disease Notes* continued on next page)

Identification of the Fungal Endophyte *Epichloe festucae* in the Fine Fescue *Festuca ampla.* I. Zabalgogeazcoa and B. García Criado, IRNA-CSIC, Cordel de Merinas 40-52, 37008 Salamanca, Spain; and S. Bony, UMR INRA-ENVL 188, Métabolisme et Toxicologie Comparés des Xénobiotiques, Ecole Vétérinaire de Lyon, B.P. 83, Marcy l'Etoile, 68280, France. Plant Dis. 86:1272, 2002; published on-line as D-2002-0902-01N, 2002. Accepted for publication 25 July 2002.

Festuca ampla is native to the Iberian Peninsula (4). Endophytic mycelium was observed by microscopy (2) in stem pith samples of two of eight asymptomatic plants of *F. ampla* collected in one population from a natural grassland in Salamanca, Spain. The fungus could be isolated (2) only from these two plants, and conidiophores and reniform conidia typical of *Epichloe* species (3) were observed in pure cultures. The two infected plants maintained in pots outside, developed ectostromata in some reproductive stems (choke disease) the year after the field sampling. Six seeds collected from an infected plant were germinated, and all six seedlings were found to be infected based on microscopy (2), implying seed transmission of the endophyte. These observations suggest that this is a pleiotropic symbiont, having both mutualistic and pathogenic states in its host. An ergovaline concentration of 120 ng/g dry weight was detected in a sample of leaves and leaf sheaths of an infected plant. All of the above characteristics are typical of the genus *Epichloe*, and in particular of the fine fescue endophyte *E. festucae* (1,3). To determine the species, internal transcribed spacer and 5.8-rDNA sequences as well as a partial sequence of the β-tubulin gene were obtained. These two sequences (EMBL Accession Nos. AJ488497 and AJ488498) showed 100% sequence homology to the corresponding sequences in *E. festucae*. To our knowledge, this is the first report of this endophyte species in the grass *F. ampla*.

References: (1) L. P. Bush et al. Plant Physiol. 114:1, 1997. (2) E. M. Clark et al. J. Microbiol. Methods 1:149, 1983. (3) A. Leuchtmann et al. Mycologia 86:802, 1994. (4) I. Markgraff-Dannenberg. Festuca. Pages 125-153 in: Flora Europaea, Vol 5. Cambridge University Press, Cambridge, 1980.

First Report of Frogeye Leaf Spot (*Cercospora sojina*) in Wisconsin. Alemu Mengistu, USDA, ARS, Crop Genetics and Production Unit, Stoneville, MS 38776; and N. C. Kurtzweil and C. R. Grau, Department of Plant Pathology, University of Wisconsin, Madison, 53706. Plant Dis. 86:1272, 2002; published on-line as D-2002-0912-01N, 2002. Accepted for publication 29 August 2002.

Frogeye leaf spot, caused by *Cercospora sojina*, is an economically important foliar disease of soybean (*Glycine max* (L.) Merr.) in areas where growing conditions are warm and humid. During a survey conducted in 2000 and 2001 in soybean fields in Wisconsin, reddish brown, circular to angular spots varying in diameter from 1 to 5 mm were observed on soybean leaves in four fields in Dane and Iowa counties, and in five and six fields in Lafayette and Green counties, respectively. Soybean plants were in growth stages between R3 and R5 during sampling. Disease incidence ranged from 30 to 100% with 5 to 10% of leaf area covered with leaf spot in 2000. In 2001, trace levels of the disease were detected in Dane County, but no symptomatic plants were present in the other counties. Symptomatic leaves were collected from all locations in 2000 and Dane county in 2001. Ten leaves were randomly picked from all samples for each year, placed in a 100 × 15 mm petri dish dampened with Whatman No.1 filter paper, and incubated overnight at 24°C. Fungal sporulation developed after 24 h. Fifteen spores were removed from the 10 leaves, placed on acidified potato dextrose agar (APDA), and incubated in the dark at 24°C. Cultures with dark pigmentation and associated conidia and conidiophores were observed after 3 weeks. The conidiophore, spore type, and leaf symptoms correspond to the description of *C. sojina* (1). Conidiophores were light-to-dark brown, one to four septate, and fasciculate. The conidiophores were also geniculate and measured 52 to 120 x 4 to 6 µm. Conidia were 0 to 10 septate, hyaline, elongate to fusiform, and measured 40 to 60 x 6 to 8 µm. Cultures were maintained on APDA, and spores for inoculations were produced on this medium. Spores from the 2000 cultures were harvested, bulked together, and used for pathogenicity tests. Pathogenicity tests were conducted in a growth chamber using a known susceptible soybean cultivar, Blackhawk. Ten-cm-diameter pots each containing 4 plants was used. Twenty plants were inoculated and 20 served as noninoculated controls. Ten-day-old plants were inoculated with a spore suspension of 3×10^5 spores/ml by spraying inoculum over the entire leaf surfaces with a spray atomizer. Control plants were sprayed similarly with sterile distilled water. Plants were incubated in an enclosed, transparent fiberglass box with a humidifier that provided 95 to 100% humidity. Lighting in the growth chamber was adjusted to 18-h light and 6-h dark during the inoculation period. Plants were removed from the box after 48 h and placed in a growth chamber with a 12-h photoperiod. The light output in the growth chamber was 300 $\mu mol \cdot m^{-2} \cdot s^{-1}$ and the temperature was maintained at 24 ± 3°C. The experiment was repeated once. Typical field symptoms appeared on each of the inoculated plant 8 days after inoculation, while the controls expressed no leaf symptoms. *C. sojina* was reisolated from all symptomatic plants. To our knowledge, this is the first report of *C. sojina* from soybean in Wisconsin.

Reference: (1) D. V. Phillips. Frogeye leaf spot. Page 20 in: Compendium of Soybean Diseases. 4th ed. G. L. Hartman, J. B. Sinclair, and J. C. Rupe, eds. American Phytopathological Society, St. Paul, MN, 1999.

First Report of Web Blight Caused by *Rhizoctonia solani* on *Catharanthus roseus* in Louisiana. G. E. Holcomb, Department of Plant Pathology and Crop Physiology, Louisiana State University Agricultural Center, Baton Rouge 70803; and D. E. Carling, University of Alaska, Palmer Research Center, Palmer 99645. Plant Dis. 86:1272, 2002; published on-line as D-2002-0916-02N, 2002. Accepted for publication 8 September 2002.

Web (aerial) blight was observed in field plots of *Catharanthus roseus* (L.) G. Don (Madagascar periwinkle) during three consecutive summers at the Burden Research Center in Baton Rouge. Leaf spots formed first, followed by a general blighting of leaves and stems that resulted in circular areas of dead plants in the plots. Dead leaves were matted together but remained attached to plants. Mycelia, and occasionally small, brown sclerotia (1 to 3 mm) were observed on blighted foliage. During the first year, only prostrate-growing cultivars belonging to the Mediterranean series of *C. roseus* were infected, but in 2001 and 2002 upright-growing cultivars as well as those with prostrate growth habit became infected. The disease occurred in July and August during periods of hot, humid, and rainy weather. Among 52 cultivars in the 2001 trial, only 'Tropicana Pink', 'Tropicana Rose' and 'Stardust Orchid' were disease free. A *Rhizoctonia* sp. was consistently isolated from diseased plants and further characterized as *R. solani* Kühn AG-1 based on its multinucleate cells and hyphal anastomosis with several AG-1 tester isolates. On potato dextrose agar, colonies displayed morphologies with characteristics of AG-1 IA and AG-1 IB, therefore, identification to AG subgroup was not made. Mature colonies ranged from light tan to brown and produced sclerotia, individually or in clumps, at the edge of the culture dish. Pathogenicity tests were performed by placing agar blocks, taken from the margins of 7-day-old cultures, on stems of eight healthy Madagascar periwinkle plants (15 to 20 cm tall). Inoculated and noninoculated control plants were held in a dew chamber at 26°C for 3 days and then moved to a greenhouse. Leaves on all inoculated plants developed water-soaked spots that turned dark brown or black prior to death, whereas noninoculated plants remained healthy. *R. solani* was reisolated from inoculated plants and its cultural characteristics were similar to those of the original isolate. Web blight occurs in Louisiana on Madagascar periwinkle used as landscape bedding plants, but has not been observed on container-grown plants. Web blight caused by *R. solani* AG-1 was previously reported on Madagascar periwinkle from Alabama (1). *R. solani* AG-1 has been reported previously as causing web blight in Louisiana on rosemary (2), dianthus (4), and verbena (3). To our knowledge, this is the first report of web blight on Madagascar periwinkle (*C. roseus*) in Louisiana.

References: (1) A. K. Hagan and J. M. Mullen. Plant Dis. 77:1169, 1993. (2) G. E. Holcomb. Plant Dis. 76:859, 1992. (3) G. E. Holcomb and D. E. Carling. Plant Dis. 84:492, 2000. (4) G. E. Holcomb and D. E. Carling. Plant Dis. 84:1344, 2000.

First Report of Bacterial Soft Rot of White Flowered Calla Lily Caused by *Erwinia chrysanthemi* in Taiwan. Y.-A. Lee and K.-P. Chen, Department of Life Science, Fu Jen Catholic University, Hsin Chuang 24205, Taipei, Taiwan, Republic of China; and Y.-C. Chang, Department of Plant Pathology, National Taiwan University, Taipei, Taiwan, Republic of China. Plant Dis. 86:1273, 2002; published on-line as D-2002-0905-02N, 2002. Accepted for publication 16 August 2002.

In 2002, soft rot symptoms on white flowered calla lily (*Zantedeschia aethiopica*) were found in some nurseries in the Yang Ming Shan area, Taipei, Taiwan. The disease was characterized by foul smelling rot and collapse of flower stems. Isolations from diseased flower stems consistently yielded bacterial colonies that were translucent, white, and glistening on nutrient agar. Ten representative isolates were chosen for further characterization. All isolates were gram-negative rods, facultatively anaerobic, sensitive to erythromycin (25 µg/ml), negative for oxidase and arginine dihydrolase, and positive for catalase, phosphatase, tryptophanase (indole production), and lecithinase. They fermented glucose and reduced nitrates to nitrites. The maximum temperature for growth was 37°C. The isolates hydrolyzed gelatin and esculin, produced acids from utilizing D(+)–glucose, melibiose, amygdalin, L(+)–arabinose, D-mannitol, and sucrose, but not from trehalose, lactose, D-sorbitol, or maltose. They degraded pectate and rotted potato, carrot, sweet pepper, and onion slices. Bacterial suspensions (10^8 CFU/ml) were injected in stems of white flowered calla lily to fulfill Koch's postulates. Control plants were inoculated with sterile distilled water. Inoculated plants were kept in a growth chamber at 30°C. Symptoms developed 1 to 2 days in all four inoculated plants and appeared to be identical to those observed on diseased material in nurseries. The four control plants did not rot. The bacterium was readily reisolated from diseased plants, confirmed to be the inoculated pathogen, and identified as *Erwinia chrysanthemi*. *E. carotovora* subsp. *carotovora* has been reported to cause soft rot of other calla lilies, such as *Zantedeschia* sp. cvs. Black Magic and Pink Persuasion and *Z. elliottiana* in Taiwan (1). However, to our knowledge, this is the first report of soft rot caused by *E. chrysanthemi* on white flowered calla lily in Taiwan.

Reference: (1) S. T. Hsu and K. C. Tzeng. Pages 9-18 in: Proc. Int. Conf. Plant Path. Bact., 5th. J. C. Lozano, ed. CIAT, Cali, Colombia, 1981.

Occurrence of Potato Powdery Scab Caused by *Spongospora subterranea* f. sp. *subterranea* in Costa Rica. M. Montero-Astúa and V. Vásquez, Centro de Investigación en Biología Celular y Molecular (CIBCM), Universidad de Costa Rica (UCR); and C. Rivera, CIBCM and Facultad de Microbiología, UCR. Plant Dis. 86:1273, 2002; published on-line as D-2002-0826-01N, 2002. Accepted for publication 12 August 2002.

Powdery scab of potatoes, caused by *Spongospora subterranea* (Wallr.) Lagerheim f. sp. *subterranea* Tomlinson, is important worldwide due to its effect on tuber quality and transmission of *Potato mop-top virus*. Although powdery scab-like lesions have been observed on potato in Costa Rica (1), the presence of the pathogen has not been confirmed. During a survey in 2001, powdery scab-on was observed from a field and a greenhouse in the high elevation zone of the main potato-producing area of Costa Rica. Commercial potatoes with scab-like lesions were also obtained at a farmers' market. Scraping the lesions, and observing spore balls or cystosori with a honey-comb-like structure under light microscopy confirmed the identity of *S. subterranea*. The identity of the pathogen was also confirmed by enzyme-linked immunosorbent assay using monoclonal antibodies specific for *S. subterranea* (BioReba Ag, Reinach, Switzerland). Pathogenicity of *S. subterranea* was confirmed by a bioassay on tomato plants grown in nutrient solution culture (2). Tomato cv. Supermarmande plants were grown from seed in pots filled with quartz and watered with nutrient solution. Three weeks after planting, the roots were trimmed to 60 mm, and the plants were transferred to the nutrient solution for additional growth. After growing for 1 week in the nutrient solution, tomato seedlings were inoculated by replacing the nutrient solution with nutrient solution containing cystosori (20 mg/liter, wt/vol) that were scraped from the scab lesions. Zoosporangia of *S. subterranea* were observed in root hairs and epidermal cells of the seedlings 2 weeks after inoculation. To our knowledge, this is the first report that confirms the presence of *S. subterranea* on potato in Costa Rica.

References: (1) R. Amador. Invest. Agri. Costa Rica. 1(1):16, 1987. (2) U. Merz. Bull. OEPP 19:585, 1989.

First Report of *Botryosphaeria rhodina* Causing Shoot Blight of Pistachio in California. T. J. Michailides, D. P. Morgan, and D. Felts, University of California Davis, Kearney Agricultural Center, Parlier 93648; and J. Phillimore, Pest Control Advisor, Bakersfield, CA 93308. Plant Dis. 86:1273, 2002; published on-line as D-2002-0906-02N, 2002. Accepted for publication 21 August 2002.

In the summers of 2000 and 2001, shoot blight was observed in pistachios (*Pistacia vera* L.) grown in Kern County, California. Black, necrotic lesions developed at the base of shoots originating from contaminated or partially infected buds. Infection moved upward resulting in a progressive wilting and blighting of leaves. Leaf blades on infected shoots withered, and petioles became necrotic. Symptoms have been considered characteristic of infection by *Botryosphaeria dothidea* (Moug.:Fr.) Ces. & de Not., but this pathogen causes panicle and shoot blight of pistachio (1). However, there were no symptoms of any fruit panicle infections on trees we observed. Isolations on acidified potato dextrose agar from the base of blighted shoots in both years revealed a fast-growing fungus producing pycnidia which was identified as the anamorph *Lasiodiplodia theobromae* (Pat.) Griffon & Maubl. of *B. rhodina* Berk. & Curt. Arx. Identification of the pathogen was based on characteristic dark brown, oval pycnidiospores with striations on the surface of the spore along the long axis. Pathogenicity tests were performed on 12 Kerman pistachio trees grown at Kearney Agricultural Center, in Parlier, CA, using three isolates recovered from pistachios grown in two locations. Six to 16 current season shoots of pistachio trees (1 to 2 shoots per tree) were wounded with a 5-mm-diameter cork borer, and a mycelial plug of 5-day-old cultures of *B. rhodina* was inserted in each wound. Shoots were wrapped with Parafilm to prevent desiccation of inoculum. Six other shoots (one per tree) were inoculated similarly with mycelial agar plugs of a pistachio isolate of *B. dothidea* and served as positive controls, while six similar shoots were inoculated with only agar plugs and served as negative controls. Wilting of lower leaves in the majority of inoculated shoots started within 4 days for *B. rhodina* and 7 days for *B. dothidea*. Depending on the isolate of *B. rhodina*, 1 to 5 shoots and 50 to 80% of leaves were blighted within 7 days after inoculation. All inoculated shoots were left on the trees until 3 to 4 months after inoculation, pruned and assessed again. For inoculations done in September 2001, 33 to 71% of shoots were blighted, and the rest had cankers ranging from 22.5 to 28 mm long and 13.5 to 23.5 mm wide. A majority (67 to 100%) of shoots had pycnidia of the pathogen present. For inoculations done in October 2001, none of the shoots was blighted, but cankers ranged from 5 to 55.4 mm long and 6 to 22 mm wide and 33.3 to 100% developed pycnidia. *B. rhodina* was isolated from all inoculated shoots but not from negative controls or those inoculated with *B. dothidea*. Inoculations of shoots with *B. dothidea* produced similar symptoms as those of *B. rhodina*. Shoots that served as negative controls did not develop symptoms. Because panicle and shoot blight of pistachio caused by *B. dothidea* has developed to epidemic levels in commercial pistachio orchards and is of concern to the pistachio industry in California, it would be of interest to monitor how much shoot blight caused by *B. rhodina* would eventually develop over the years in commercial pistachio orchards. A survey was initiated in 2002 to determine how widespread *B. rhodina* is in California pistachios. To our knowledge, this is the first report worldwide of *B. rhodina* causing shoot blight of pistachio.

Reference: (1) T. Michailides. Panicle and shoot blight. Page 68 in: Compendium of Nut Crop Diseases in Temperate Zones. B. L. Teviotdale, T. J. Michailides, and J. W. Pscheidt, eds. American Phytopathological Society, St. Paul, MN 2002.

(*Disease Notes* continued on next page)

First Report of *Phytophthora ramorum* on Coast Redwood in California. P. E. Maloney and D. M. Rizzo, Department of Plant Pathology, One Shields Ave., University of California, Davis 95616; S. T. Koike, University of California Cooperative Extension, 1432 Abbott Street, Salinas; and T. Y. Harnik and M. Garbelotto, Department of Environmental Science, Policy and Management, Ecosystem Science Division, 151 Hilgard Hall, University of California, Berkeley 94720; Plant Dis. 86:1274, 2002; published on-line as D-2002-0906-03N, 2002. Accepted for publication 26 August 2002.

Phytophthora ramorum S. Werres & A.W.A.M. de Cock was isolated from discolored leaves and cankers on small branches (<0.5 cm in diameter) on 27 coast redwood (*Sequoia sempervirens*) saplings (2 to17 cm in diameter) at two locations in California (Jack London State Park, Sonoma County and Henry Cowell State Park, Santa Cruz County). Symptoms were observed on branches throughout the crowns of affected trees. Isolates were identified as *P. ramorum* by their abundant chlamydospores and caducous, semi-papillate sporangia (2) and internal transcribed spacer (ITS) rDNA sequences identical to those of *P. ramorum* from *Quercus* spp., *Lithocarpus densiflorus,* and *Rhododendron* (1,2). *P. ramorum* was also detected in dying basal sprouts on mature redwood trees from an additional five locations in coastal California by polymerase chain reaction (PCR) amplification of the ITS region using DNA extracted from symptomatic tissue and *P. ramorum*-specific PCR primers. To test for pathogenicity, foliage inoculations were conducted on redwood seedlings in two trials by misting 30 leaves per trial (five leaves per seedling plus controls) with sterile distilled water and then pinning inoculum plugs to the upper surface of leaves. Inoculation resulted in lesions of 1 to 20 mm on individual leaves, and *P. ramorum* was recovered from 43% of inoculated leaves. Symptoms were not restricted to inoculated leaves because 15 inoculations of individual leaves led to discoloration of two or more adjacent leaves. On one inoculation, 60 mm of the adjacent stem was killed. Stems of redwood seedling (approximately 1 cm in diameter) were wound inoculated (1) in two trials consisting of 10 inoculated seedlings per trial plus 10 controls. After 6 weeks, lesion lengths in the cambium caused by *P. ramorum* averaged 13.7 mm (range 4 to 21 mm). *P. ramorum* was recovered from 100% of inoculated stems. Entire branches near the inoculation point became chlorotic even though no direct connection was evident between the lesion and the branches. No chlorosis was observed among the control inoculations. Mean lesion lengths of inoculated stems were significantly greater in both trials than those of control inoculations (mean 6.2 mm) at $P < 0.05$ based on analysis of variance (ANOVA). Redwood saplings (2.5 to 4.5 cm in diameter) were also wound inoculated in a separate trial. No phloem or cambial discoloration was observed after 7 weeks, but necrotic lesions in the xylem had a mean length of 39 mm (range 12 to 73 mm). In addition, narrow streaks, 1 to 2 mm in diameter, were also noted in the xylem extending from the necrotic areas upward to 90 cm. *P. ramorum* was recovered from 70% of inoculated stems in this trial. Mean lesion lengths of *P. ramorum* were significantly greater in all trials than those of control inoculations (mean 20 mm) at $P < 0.05$ based on ANOVA. While *P. ramorum* causes a lethal canker on *Quercus* spp. and *L. densiflorus* (1), we have not observed unusual mortality or disease symptoms on overstory redwoods in natural forests. The impact of infection by *P. ramorum* on understory redwoods is also unclear. However, the pathogen appears to be able to kill sprouts.

References: (1) D. M. Rizzo et al. Plant Dis. 86:205, 2002. (2) S. Werres et al. Mycol. Res. 105:1155, 2001.

First Report of *Phytophthora ramorum* on Douglas-Fir in California. J. M. Davidson, Pacific Southwest Research Station, USDA Forest Service, P.O. Box 245, Berkeley, CA 94701; M. Garbelotto, Department of Environmental Science, Policy and Management, Ecosystem Science Division, 151 Hilgard Hall, University of California, Berkeley 94720; S. T. Koike, University of California Cooperative Extension, 1432 Abbott Street, Salinas; and D. M. Rizzo, Department of Plant Pathology, One Shields Ave., University of California, Davis 95616. Plant Dis. 86:1274, 2002; published on-line as D-2002-0906-04N, 2002. Accepted for publication 26 August 2002.

Phytophthora ramorum S. Werres & A.W.A.M. de Cock was isolated from three Douglas-fir (*Pseudotsuga menziesii*) saplings in a mixed-evergreen forest in Sonoma County, California. Symptoms on these saplings included cankers on small branches (0.5 to 1 cm in diameter) resulting in wilting of new shoots, dieback of branches, and loss of leaves as much as 15 cm from the twig tip. Symptoms were observed on most saplings growing in the same area. On several smaller saplings (<1 m tall), *P. ramorum* infection resulted in the death of the leader and the top several whorls of branches. Isolates were identified as *P. ramorum* by their abundant chlamydospores and caducous, semi-papillate sporangia (2) and internal transcribed spacer rDNA sequences identical to those of isolates of *P. ramorum* from *Quercus* spp., *Lithocarpus densiflorus,* and *Rhododendron* (1,2). To test for pathogenicity, foliage inoculations were conducted on seedlings in two trials by misting 30 leaves per trial (five leaves per seedling plus controls) with sterile distilled water and pinning inoculum plugs, taken from the margin of *P. ramorum* cultures, to the upper surface of leaves. Inoculation resulted in lesions ranging between 1 and 12 mm long, and *P. ramorum* was recovered from 47% of inoculated leaves. Symptoms were not restricted to inoculated leaves, and in 26 single-leaf inoculations, lesions 17 to 85 mm long developed on branches (five mm in diameter) adjacent to the inoculated leaf. Isolation success from branch lesions was 50%, despite the fact that such lesions were apparently disjunct from the small 1-mm lesions developing on inoculated leaves. Stems of Douglas-fir seedlings (approximately 1 cm in diameter) were wound inoculated (1) in two trials consisting of 10 inoculated seedlings per trial plus 10 controls. After 6 weeks, lesion lengths in the cambium averaged 38 mm (range 12 to 62 mm), and three seedlings were completely girdled. *P. ramorum* was recovered from 75% of inoculated stems. Mean lesion lengths on seedlings inoculated with *P. ramorum* were significantly greater ($P < 0.05$) in both trials than those of control inoculations (mean 9 mm) based on analysis of variance. We have not observed unusual mortality or disease symptoms on overstory Douglas-fir trees in natural forests. The importance of *P. ramorum* branch tip dieback for growth and reproduction of Douglas-fir is unknown. Douglas-fir is present in many forests in California and Oregon already infested by *P. ramorum*, yet we have found infection of plants at only one location. At this site, symptomatic Douglas-fir saplings were surrounded by bay laurel (*Umbellularia californica*) trees with extremely high levels of *P. ramorum* infection. *P. ramorum* is known to sporulate prolifically on bay laurel leaves. More studies are necessary to determine if the incidence of *P. ramorum* in Douglas-fir extends to other locations or if it is limited to this one locale.

References: (1) D. M. Rizzo et al. Plant Disease 86:205, 2002. (2) S. Werres et al. Mycol. Res. 105:1155, 2001.

First Report of White Fir Dwarf Mistletoe on Mountain Hemlock. R. Mathiasen, School of Forestry, Northern Arizona University, Flagstaff 86011. Plant Dis. 86:1274, 2002; published on-line as D-2002-0916-01N, 2002. Accepted for publication 6 September 2002.

White fir dwarf mistletoe (*Arceuthobium abietinum* Engelm. ex Munz f. sp. *concoloris* Hawksw. & Wiens) is a serious and common pathogen of white fir (*Abies concolor* (Gord. & Glend.) Hildebr.), grand fir (*A. grandis* (Dougl. ex D. Don) Lindl.), and Low's fir (*A. lowiana* (Gord.) A. Murr.) in the western United States (1). In August 2002, this dwarf mistletoe was observed parasitizing mountain hemlock (*Tsuga mertensiana* (Bong.) Carr.) growing among severely infected grand fir near the trailhead to Cabot Lake in the Mount Jefferson Wilderness Area, Oregon at 44°34′27″N, 121°43′43″W, elevation 1,340 m. Only 2 of 27 mountain hemlocks observed in this area were infected. One tree had four infections, and one tree had two infections. Several fully developed male plants were found on one of the infected branches of mountain hemlock and were morphologically similar to those growing on the nearby grand fir. Other dwarf mistletoes that commonly parasitize mountain hemlock (*Arceuthobium tsugense* subsp. *mertensianae* and *Arceuthobium laricis*) were not observed in the area. In addition, white fir dwarf mistletoe can be distinguished from these mistletoes by its larger, yellowish shoots (1). Specimens of the mistletoe from mountain hemlock have been deposited in the Deaver Herbarium, Northern Arizona University, Flagstaff. To my knowledge, this is the first report of white fir dwarf mistletoe on mountain hemlock (1).

Reference: (1) F. Hawksworth, and D. Wiens. Dwarf mistletoes: biology, pathology, and systematics. USDA Agric. Handb. 709, 1996

First Report of Bacterial Wilt of Common Bean Caused *by Curtobacterium flaccumfaciens* in Western Canada. T. F. Hsieh, H. C. Huang, R. S. Erickson, L. J. Yanke, and H.-H. Mündel, Agriculture and Agri-Food Canada Research Centre, Lethbridge, AB, T1J 4B1 Canada. LRC Contribution No. 38702073. Plant Dis. 86:1275, 2002; published on-line as D-2002-0918-01N, 2002. Accepted for publication 4 September 2002.

Bacterial wilt of common bean (*Phaseolus vulgaris* L.) caused by *Curtobacterium flaccumfaciens* pv. *flaccumfaciens* (Hedges) Collins & Jones (4) was found in 1947 in Ontario, Canada (3), but not in western Canada. Infected seeds exhibit yellow, orange, or purple discoloration (4). Examination of 36.7 kg of cull beans of crops grown in southern Alberta in 2001 obtained from a processing plant revealed 5.9% yellow and 0.014% orange seeds, each with wrinkled seed coats. Bacteria were isolated on potato dextrose agar. Three strains were identified using conventional tests (2), carbohydrate oxidation (GP Microplates, Biolog Inc., Hayward, CA), and cellular fatty acids (CFA) (MIDI, Inc., Newark, DE). Strains were gram-positive, motile, aerobic rods with yellow (YSB-1, YSB-2) or orange (OSB-3) colonies. Growth occurred at 27 and 37°C. The strains were positive for citrate utilization, catalase, hydrolysis of hippurate, and indoxyl acetate, and negative for urease, gelatin liquification, and oxidase. CFA profiles were ≈48% 15:0 anteiso, 37% 17:0 anteiso, 8% 16:0 iso, 3% 15:0 iso, and 3% 16:0; with17:1 anteiso A sometimes present at <2%. Acid production was weak from carbohydrates, but all oxidized many carbohydrates in the microplates. These results match *C. flaccumfaciens* pv. *flaccumfaciens* (2) in MIDI and Biolog databases. Strains were tested for pathogenicity using seed and pod inoculations. Seeds of great northern ('US1140') and navy ('AC Skipper') beans were soaked in bacterial suspension (1 to 3 × 10^8 CFU/ml) for 1 h, sown in Cornell Peatlite Mix (1) in Root Trainers (Spencer-Lemaire Industries, Edmonton, AB, Canada), incubated at 28°C (16-h day) and 22°C (8-h night), and examined for seedling wilt after 10 days. Seeds soaked in sterile distilled water served as controls. Testing was repeated once with 3 replicates per treatment and 10 seeds per replicate. Experiments were conducted using a complete randomization design. For pod inoculation, a suspension (0.1 ml) of each strain was injected into the midrib at the basal end of each young pod of 'AC Skipper'. Pods inoculated with sterile distilled water, 0.1 ml per pod, were used as controls. After 21 days, pods were harvested and examined. Testing was repeated once with three plants per treatment and five pods per plant. Bacteria were reisolated from hypocotyls of wilted seedlings and diseased pods. Results of seed inoculations showed all strains were pathogenic to both cultivars. Wilt incidence was 38, 35, and 57% for strains YSB-1, YSB-2, and OSB-3, respectively, on 'US1140' and 44, 40, and 63% respectively, on 'AC Skipper'. Results of pod inoculations showed 63% (YSB-1) and 55% (YSB-2) of seeds had wrinkled, yellow seed coats, and 72% (OSB-3) of seeds had wrinkled, orange seed coats. Control seedlings and seeds remained healthy. *C. flaccumfaciens* pv. *flaccumfaciens* was reisolated from wilted seedlings and seeds showing yellow or orange discoloration, but not from the controls. To our knowledge, this is the first report of bacterial wilt of bean caused by yellow and orange strains of *C. flaccumfaciens* pv. *flaccumfaciens* in western Canada.

References: (1) J. W. Boodley and R. Sheldrake Jr. N.Y. State Coll. Agric. Life Sci. Inform. Bull. 43, 1977. (2) K. Komagata et al. Page 1313 in: Bergey's Manual of Systematic Bacteriology, Vol. 2, Williams and Wilkens, Baltimore, MD, 1986. (3) Z. A. Patrick, Can. J. Bot. 32:705, 1954. (4) A. W. Saettler. Bacterial wilt. Page 31 in: Compendium of Bean Diseases. R. Hall, ed. American Phytopathology Society, St. Paul, MN, 1994.

First Report of *Sclerotium rolfsii* on Star-cluster (*Pentas lanceolata*) in Taiwan. C. H. Fu, Division of Forest Protection, Taiwan Forest Research Institute, Taiwan R.O.C.; H. J. Hsieh, Department of Plant Pathology, National Taiwan University, Taipei, Taiwan; and J. C. Yao, Taoyuan District Agricultural Improvement Station, Crop Environment Section, Taiwan R.O.C. Plant Dis. 86:1275, 2002; published on-line as D-2002-0905-01N, 2002. Accepted for publication 15 August 2002.

Star-cluster (*Pentas lanceolata* (Forssk.) Deflers) has recently become popular as a bedding plant in Taiwan. During the summer of 2000, a sudden wilt of 60-day-old plants was observed in a nursery in Tainan City (southern Taiwan). 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 were covered with white mycelium that differentiated into reddish brown, spherical (1 to 2 mm in diameter) sclerotia. *Sclerotium rolfsii* was consistently recovered from the surface of symptomatic stem sections that were disinfected for 1 min in 0.5% NaOCl and plated on potato dextrose agar amended with 100 ppm streptomycin sulfate. Pathogenicity of three isolates of *S. rolfsii* was confirmed by inoculating 90-day-old plants of *P. lanceolata* that were grown in pots. Three plants each were inoculated with a 5-mm plug of agar with mycelium or two sclerotia of the pathogen. Inoculum was placed on the soil surface against the stem of each plant. Three noninoculated plants served as controls. All plants were kept in a growth chamber at 20 to 30°C with relative humidity >85%. The pathogenicity test was repeated. Inoculated plants developed symptoms within 7 days, while control plants remained symptomless. Sclerotia developed on infected tissues and *S. rolfsii* was reisolated from symptomatic tissues. Although this disease has been observed on many species of plants (1), to our knowledge, this is the first report of southern blight of *P. lanceolata* caused by *S. rolfsii* in Taiwan.

Reference: (1) Tsai, Y. P., ed. List of Plant Diseases in Taiwan. The Plant Protection Society of the Republic of China and The Phytopathological Society of the Republic of China. 1991.

First Report of Littleleaf Disease Caused by *Phytophthora cinnamomi* on *Pinus occidentalis* in the Dominican Republic. T. Jung and G. Dobler, Bavarian State Institute of Forestry (LWF), Section of Forest Ecology and Protection, Am Hochanger 11, D-85354 Freising, Germany. Plant Dis. 86:1275, 2002; published on-line as D-2002-0917-01N, 2002. Accepted for publication 3 September 2002.

Pinus occidentalis Sw. is an endemic species of the Caribbean island of Hispaniola (Dominican Republic and Haiti). It shows an extreme ecological plasticity and grows on a wide range of soil types from 0 to 3,175 m in elevation with annual mean temperatures ranging from 6 to 25°C and annual precipitation of 800 to 2,300 mm. *P. occidentalis* is a major component of forests above 800 m in elevation and forms pure climax forests above 2,000 m (4). For more than 10 years, stands of *P. occidentalis* in the Sierra (Cordillera Central) growing on a wide range of site conditions have suffered from a serious widespread disease. Symptoms include yellowing and dwarfing of needles, a progressive defoliation and dieback of the crown, and finally, death of weakened trees often caused by attacks by secondary bark beetles. Mature stands are mainly affected, but the disease is also present in plantations and natural regeneration that is older than 10 years. Disease spread is rapid, and occurs mainly along roads and from diseased trees downslope following the path of water runoff. Initially, *Leptographium serpens* was isolated from necrotic roots and was thought to be the causal agent (1). However, the symptoms of the disease more closely resemble those of littleleaf disease of *P. echinata* and *P. taeda* in the southeastern United States, which is caused by the aggressive fine-root pathogen *Phytophthora cinnamomi* Rands (3). Moreover, spread and dynamics of the disease are similar to the diebacks of *Chamaecyparis lawsoniana* in Oregon and *Eucalyptus* spp. in western Australia, which are caused by the introduced soilborne pathogens *Phytophthora lateralis* and *Phytophthora cinnamomi*, respectively. Soil samples containing the rhizosphere and fine roots of diseased *P. occidentalis* trees were collected in February 2002 at five sites near Celestina and Los Montones (Dominican Republic) and transported to the Bavarian State Institute of Forestry. The pathogen was baited from the soil by floating 3- to 7-day-old leaves of *Quercus robur* seedlings over flooded soil and placing the leaves on selective PARPNH agar (2). *Phytophthora cinnamomi* was isolated from the soil of all five sites. Crossing with A1 and A2 tester strains of *Phytophthora cinnamomi* confirmed that all isolates belong to the A2 mating type. In cross sections of necrotic fine roots, characteristic structures of *Phytophthora cinnamomi* such as nonseptate hyphae and chlamydospores could be observed. Our results indicate that the disease of *P. occidentalis* is caused by the introduced pathogen *Phytophthora cinnamomi*. Because of the ecological and economical importance of *P. occidentalis*, the disease poses a major threat to forestry in the Dominican Republic. Future research should include the mapping of the disease, pathogenicity tests on *P. occidentalis* and alternative pine species, in particular *P. caribaea*, screening for resistance in the field, and testing of systemic fungicides such as potassium phosphonate, which is known to be effective against *Phytophthora cinnamomi*.

References: (1) G. Dobler. Manejo y Tablas de Rendimiento de *Pinus occidentalis*. Plan Sierra, San José de las Matas, Dominican Republic, 1999. (2) T. Jung et al. Plant Pathol. 49:706, 2000. (3) S. W. Oak and F. H. Tainter. How to identify and control littleleaf disease. Protection Rep. R8-PR12, USDA Forest Service Southern Region, Atlanta, Georgia, 1988. (4) L. Sprich. Allg. Forst. Jagdztg. 168:67, 1997.