## **Disease Notes**

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**First Report of** *Xylella fastidiosa* **Infecting Coffee in Costa Rica.** C. M. Rodríguez and J. J. Obando, Instituto del Café de Costa Rica; and W. Villalobos, L. Moreira, and C. Rivera, Centro de Investigación en Biología Celular y Molecular, Universidad de Costa Rica. Plant Dis. 85:1027, 2001; published on-line as D-2001-0723-02N, 2001. Accepted for publication 20 April 2001.

In 1995, severe symptoms were observed on 'Caturra' and 'Catuaí' coffee (Coffea arabica L.) varieties in farms in the southern part of the Central Valley in Costa Rica. Symptoms were reduced leaf size, malformation of leaves, curling of leaf edges, shortening of internodes, and severe leaf chlorotic mosaic, which sometimes became necrotic. Abortion of flowers and young beans was also observed, with a reduction in yield. Plants also showed irregular growth with an atypical curling appearance that gave rise to the Spanish name "crespera." Ten and three healthy plants were inoculated by grafting in the greenhouse, using infected and healthy budwoods, respectively. Approximately 6 months after inoculation, 3 of 10 grafted plants with infected budwoods showed symptoms of leaf chlorosis, curling, and malformation of leaves and bunched new flushes. Samples of 39 symptomatic plants collected from the field and samples of 3 healthy plants maintained in the greenhouse were tested by enzyme-linked immunosorbent assay (ELISA). All (100%) analyzed symptomatic samples were positive for X. fastidiosa, and all healthy controls were negative. The symptoms observed in Costa Rica are different from those described for coffee leaf scorch in Brazil (1,2), but the climatological conditions and soil type present in Costa Rica are also very different from the areas where X. fastidiosa occurs in Brazil. Leafhoppers were collected randomly in one of the most affected regions. Graphocephala permagna and Erythrogonia sonora were the most frequent insect species found associated with coffee. In ELISA, 34.5% (10 of 29) and 23.8% (5 of 21) of the collected specimens belonging to G. permagna and E. sonora, respectively, tested positive for X. fastidiosa. These positive ELISAs do not necessarily mean that the insect is a vector. The results presented here extend the known geographic distribution of X. fastidiosa. To our knowledge, this is the first report of X. fastidiosa in coffee in Costa Rica.

*References*: (1) M. J. G. Beretta et al. Plant Dis. 80:821, 1996. (2) de Lima et al. Plant Dis. 82:94, 1998.

**First Report of** *Potato spindle tuber viroid* **in Tomato in New Zealand.** D. R. Elliott, B. J. R. Alexander, T. E. Smales, Z. Tang, and G. R. G. Clover, NPPRL, MAF, P.O. Box 2095, Auckland, New Zealand. Plant Dis. 85:1027, 2001; published on-line as D-2001-0719-03N, 2001. Accepted for publication 3 July 2001.

During May 2000, symptoms resembling those of Potato spindle tuber viroid (PSTVd) infection were observed in glasshouse tomatoes (cv. Daniella) growing on one site in Tuakau, South Auckland, New Zealand. Symptoms appeared 2 to 3 months after planting, were confined to plant tops, and included leaf interveinal chlorosis, epinasty, and brittleness. Affected plants comprised ≈10% of the crop and were located near access points. PSTVd was identified in symptomatic plants by the Dutch Plant Protection Service and confirmed by mechanical transmission and grafting to tomato cv. Rutgers and reverse transcription polymerase chain reaction (2). The sequenced genome of this isolate (Accession AF369530) was 358 nt in length and had the closest homology to a Dutch isolate (Accession X17268). Electron microscopy did not reveal the presence of any viruses in affected plants and specific tests for other tomato pathogens were negative. A survey of 50 tomato glasshouse facilities throughout New Zealand revealed three further infected sites, two located close to the original site and one in Nelson, some 480 km distant. However, a survey of field-grown potato crops within 1.5 km of the original outbreak site did not reveal the presence of the viroid. PSTVd is seed transmitted and was probably introduced in glasshouses by use of infected seed. Glasshouse tomatoes are an important crop in New Zealand and annual production is currently 40,000 tonnes. The yield

of affected plants may be decreased by up to 80% if suitable controls are not implemented (1).

References: (1) S. Kryczynski et al. Phytopath. Polonica 22:85, 1995. (2) A. M. Shamloul et al. Can. J. Plant Pathol. 19:89, 1997.

*Melon chlorotic leaf curl virus*, a New Begomovirus Associated with *Bemisia tabaci* Infestations in Guatemala. J. K. Brown, A. M. Idris, and D. Rogan, Department of Plant Sciences, University of Arizona, Tucson 85721; M. H. Hussein, Genetics Department, Cairo University, Egypt; and M. Palmieri, Virology Laboratory, Research Institute, Universidad Del Valle, Guatemala. Plant Dis. 85:1027, 2001; published on-line as D-2001-0705-02N, 2001. Accepted for publication 15 March 2001.

In 2000, geminivirus-like symptoms were widespread in muskmelon (Cucumis melo L.) fields (70 to 80% incidence) in Zacapa Valley, Guatemala. Muskmelon fields were infested with the whitefly Bemisia tabaci (Genn.), and plants exhibited patchy foliar chlorosis, leaf curling, and reduced fruit set, which is reminiscent of symptoms caused by certain whitefly-transmitted geminiviruses. Quarantine restrictions prevented experimental transmission experiments from being carried out with the whitefly vector or biolistic inoculation. Leaves collected from six symptomatic plants were assessed for the presence of begomovirus DNA by polymerase chain reaction (PCR) with the use of degenerate primers that amplify the core region of the coat protein (CP) gene of most begomoviruses (1). PCR products of the expected size (approximately 576 bp) were obtained from all three melon samples. The core CP amplicons were cloned, and their nucleotide sequences were compared. Nucleotide sequences of core CP fragments shared 99.7% identity, suggesting the presence of a single begomovirus in all assayed symptomatic melon plants. Two additional pairs of degenerate primers were used to obtain contiguous viral fragments containing the CP gene, the common region of the A component (CR-A; approximately 2,100 bp), and a fragment containing the CR of the B component (CR-B; approximately 1,100 bp), respectively (2). At least three amplicons obtained with each primer pair were cloned and their nucleotide sequence was determined. Virus-specific PCR primers were then designed within the CP open reading frame and used to obtain fragments that overlapped with the 2,100-bp fragment to yield an apparent full-length A component of 2,662 nucleotides (accession no. AF325497). CR-A and CR-B (accession no. AF325498) sequences (161 nucleotides) shared 98.1% identity and contained an identical directly repeated, replicationassociated protein (REP) binding site: GGTGT CCT GGTGT. Nucleotide sequence alignment, with CLUSTAL W, of the melon virus A-component with that of other well-studied begomoviruses revealed that its closest relatives were members of the Squash leaf curl virus (SLCV) group. The melon virus from Guatemala shared its greatest sequence identity, 83.1%, with SLCV extended (SLCV-E) (accession no. M38183), indicating that it is a new, previously unidentified begomovirus species, herein referred to as Melon chlorotic leaf curl virus (MCLCV). The next closest relatives of MCLCV were SLCV restricted (SLCV-R; 78.6%) (S. G. Lazarowitz, unpublished) Cucurbit leaf curl virus-Arizona (CuLCV-AZ; accession no. AF256199; 74.1%) (3), Cabbage leaf curl virus (CaLCV; 72.0%), Bean calico mosaic virus (BCMoV; 71.7%), and Texas pepper virus-Tamaulipas (71.4%). Additionally, the theoretical REP binding element, GGTGT, is 100% identical among MCLCV and BCMoV, CaLCV, CuLCV-AZ, SLCV-E, and SLCV-R. On the basis of shared nucleotide sequence identities with other begomoviruses described to date and the presence of B. tabaci in melon fields, it is likely that MCLCV also is whitefly-transmitted. Collectively, CP and CR sequences suggest that MCLCV is a new species of the SLCV lineage that contains other bipartite begomoviruses indigenous to Central America, Mexico, and the U.S. Sunbelt states.

*References*: (1) S. D. Wyatt and J. K. Brown. Phytopathology 86:1288, 1996. (2) A. M. Idris and J. K Brown. Phytopathology 88:648, 1998. (3) J. K. Brown et al. Plant Dis. 84:809, 2000.

(Disease Notes continued on next page)

## Disease Notes (continued)

First Report of Fusarium Stem and Root Rot of Greenhouse Cucumber Caused by *Fusarium oxysporum* f. sp. radiciscucumerinum in Ontario. R. F. Cerkauskas and J. Brown, Agriculture & Agri-Food Canada; G. Ferguson, Ontario Ministry of Agriculture, Food & Rural Affairs, Greenhouse & Processing Crops Research Centre, Harrow, Ontario, Canada NOR 1GO. Plant Dis. 85:1028, 2001; published on-line as D-2001-0716-01N, 2001. Accepted for publication 11 June 2001.

Fusarium stem and root rot on greenhouse long English cucumber (Cucumis sativus L.) cvs. Bodega and Gardon was observed at four commercial greenhouses in Leamington, Ontario, Canada. Losses of 25 to 35%, representing 2.5 ha, were noted. The greenhouse cucumber industry in Ontario comprises 119 ha, with sales in 1999 of \$78 million (Canadian). Some foliar chlorosis developed slowly on the lower foliage of affected plants. Basal stem tissue developed a yellow buff discoloration with superficial rot, followed by advanced stages of stem disintegration, which were accompanied by the production of white buff fungus mycelium and orange spore masses externally, and yellowish or reddish brown discoloration of vascular tissue that extended for 5 to 6 cm. Yellowish brown to brown external discoloration extended throughout the affected roots. In contrast, the main symptoms on cucumber infected by F. oxysporum f. sp. cucurbitaceae are wilt, yellowing, and vascular discoloration. The shape of macroconidia, the presence of microconidia on short lateral phialides, and the occurrence of chlamydospores and sporodochia on acidified potato dextrose agar (aPDA) were used to identify the isolates as F. oxysporum. To confirm pathogenicity and formae specialis designation, the roots of a range of 19-day-old host plants of the family Cucurbitaceae and some of the family Solanaceae were clipped and inoculated with the use of a root-dip method (1), with 5  $\times 10^5$  spores per ml from 6-day-old aPDA cultures of three isolates and a water check with six plants per isolate. Plants were subsequently grown in a greenhouse soil mix (3:2 Fox sandy loam-peat moss, vol/vol) at 22°C day/19°C night, under environmental conditions similar to those reported elsewhere (1). A 0 (healthy) to 5 (dead) scale was used to rate plants after 30 days. In preliminary studies, cucumber cv. Corona, inoculated with various isolates and grown at 26 to 28°C for 30 days, remained asymptomatic, similar to the results of Vakalounakis 1996 (2) at 30°C and Punja and Parker 2000 (1) at 32°C. Muskmelon (Cucumis melo L. cvs. Early Dawn and Summet), watermelon (Citrullus vulgaris Schrad. cv. Yellow Doll), and cucumber cvs. Tasty Green, Odessa, Mustang, and Orient Express were the most susceptible and had ratings of 3.7, 3.0, 2.3, 3.8, 3.3, 1.1, and 1.6, respectively, with stem and root symptoms similar to those we observed in naturally infected plants and reported previously in other work (1). Cucumber cvs. Calypso, Slicemaster, Flamingo, and Marketmore 76 were less susceptible, with ratings of 0.6, 1.0, 0.1, and 1.0, respectively. No symptoms were observed on tomato (Lycopersicon esculentum Mill. cv. Trust), pepper (Capsicum annuum L. cv. Cubico), or squash (Cucurbita pepo L. cv. Taybelle) in our trials, which is in agreement with other work (1). Symptom development on a range of hosts and the cultural and morphological characteristics of the imperfect state of the fungus in vivo and in vitro confirm the identity of the fungus. To our knowledge, this is the first report of F. oxysporum f. sp. radicis*cucumerinum* in Ontario

*References*: (1) Z. K. Punja and M. Parker. Can. J. Plant Pathol. 22:349, 2000. (2) D. J. Vakalounakis. Plant Dis. 80:313, 1996.

Introduction of the Exotic *Tomato yellow leaf curl virus*–Israel in Tomato to Puerto Rico. J. Bird, Department of Plant Protection, University of Puerto Rico, Rio Piedras 00928; A. M. Idris, D. Rogan, and J. K. Brown. Department of Plant Sciences, University of Arizona, Tucson 85721. Plant Dis. 85:1028, 2001; published on-line as D-2001-0705-01N, 2001. Accepted for publication 22 June 2001.

Thirty-five-day-old tomato plants (cultivar Florasette) exhibited yellow leaf curling, stunting, and extremely reduced fruit set in spring 2001, in Guanica, Puerto Rico (PR). Twenty percent disease incidence was observed in this field and, 8 weeks later, 75% of the plants showed symptoms. These symptoms were distinct from those caused by other tomato-infecting begomoviruses reported previously from PR, namely *Merremia mosaic virus, Tomato mottle virus* (ToMoV), and *Potato yellow mosaic virus* (1). A colony of the B biotype of *Bemisia tabaci* (Genn.) was used to transmit the suspect virus from symptomatic plants collected in the field and established in the greenhouse in Rio Piedras, PR. The suspect virus was transmitted readily to tomato cultivar Roma (10 of 10

plants), and symptoms were like those observed in the field. Symptoms also were reminiscent of those described for several Old World begomoviruses, referred to as Tomato yellow leaf curl virus (TYLCV). Total nucleic acids were isolated from three symptomatic field samples and three greenhouse-inoculated tomato plants showing typical disease symptoms. Extracts were analyzed by polymerase chain reaction (PCR) with primers AV2466 and AC1145 to amplify a begomoviral fragment (approximately 1.1 bp) that contains a portion of the intergenic region and the viral coat protein gene (CP) (2). Amplicons were cloned, and their nucleotide sequences were determined. A comparison of CP with other well-studied begomoviral nucleotide sequences revealed that the CP sequences for field isolates 1 to 6 shared 99.7 to 100% identity with each other and 99.9 to 100% identity with TYLCV from Israel (TYLCV-IL; accession no. X76319) as well as TYLCV-IL isolates discovered in the Dominican Republic (DO; accession no. AF024715) and, subsequently, in Florida. TYLCV-specific PCR primers (forward) 5'-GAATTCCGCCTTTAA-TTTG-3' and (reverse) 5'-GAATTCCCACTA TCTTTCTC-3' were used to amplify the complete viral genome form a PR field isolate. An expected-sized amplicon of approximately 2.8 kb was obtained, and the nucleotide sequence of two cloned amplicons was determined. Genome organization revealed a predicted precoat open reading frame of 351 bp, which is characteristic of other Old World begomoviruses, including TYLCV-IL. Nucleotide comparisons indicated that the PR isolate shared 99% nucleotide sequence identity with TYLCV-IL (first reported from Israel) and introduced TYLCV-IL isolates in DO and Florida, thereby confirming the introduction of TYLCV-IL into PR. TYLCV-IL was first identified several years ago in the Western Hemisphere, and the virus has been reported in five offshore locations and three continental U.S. states since its initial introduction into the DO in the early 1990s. Considering the extreme virulence of TYLCV-IL compared with most New World tomato-infecting begomoviruses, this introduction, which likely occurred from a nearby Caribbean country or Florida, has the potential to destroy the fresh-market tomato industry in PR, which supplies tomatoes to the continental United States during the winter months. There is compelling evidence for the routine movement of tomato seedlings from the continental United States to this location in PR throughout the last 10 years, including the previous introduction of ToMoV (1). These incidences and others indicate the need for those in infected areas to take precautions to avoid further spread of this highly damaging virus in and adjacent to the Caribbean region.

References: (1) A. M. Idris et al. Phytopathology 88:S42, 1998. (2) A. M. Idris and J. K. Brown, Phytopathology 88:648, 1998.

**First Report of Infection of Kiwifruit by** *Pestalotiopsis* **sp. in Turkey.** A. Karakaya, Ankara University, School of Agriculture, Department of Plant Protection, Diskapi, 06110, Ankara, Turkey. Plant Dis. 85:1028, 2001; published on-line as D-2001-0718-01N, 2001. Accepted for publication 5 July 2001.

Circular to irregular brown leaf spots, 0.2 to 1.5 cm in diameter, were commonly observed on kiwifruit (Actinidia deliciosa) cv. Hayward plants in the Artvin-Arhavi region of northeastern Turkey. Leaf spots sometimes covered large portions of infected leaves, giving them a blighted appearance. Fruit symptoms consisted of brown, sunken, shriveled areas that were 0.5 to 3 cm in diameter. A fungus, later identified as a Pestalotiopsis sp. (1), was consistently isolated from diseased tissues. Pathogenicity tests were performed on 2-year-old kiwifruit plants and mature fruits at 18/22°C (day/night). A spore suspension  $(1 \times 10^6$  conidia per ml) was sprayed on leaves of 2-year-old kiwifruit plants. Agar pieces, 3 mm in diameter, from 10-day-old cultures also were applied to the leaves. Controls were treated with water and agar alone. Plants were covered with plastic bags for 3 days to ensure high humidity. After 2 weeks, disease symptoms were observed on inoculated leaves. Pestalotiopsis sp. was consistently isolated from these regions. Agar pieces from 10-day-old cultures were placed in small wounds made on the surfaces of mature, surface-disinfested fruits. Controls were treated with agar pieces alone. Softening of tissues next to the wound was observed 1 week after inoculation. Pestalotiopsis sp. was isolated from these areas. No symptoms were observed on noninoculated leaves or fruits. It was concluded that this disease is caused by Pestalotiopsis sp. This is the first report of its occurrence in Turkey.

*Reference*: (1) T. R. Nag Raj. Coelomycetous anamorphs with appendage bearing conidia. Mycologue Publications, Ontario, Canada, 1993.

**First Report of** *Bean pod mottle virus* in Soybean in Ohio. A. E. Dorrance, D. T. Gordon, and A. F. Schmitthenner, Department of Plant Pathology, The Ohio State University, Wooster, Ohio and C. R. Grau, Department of Plant Pathology, University of Wisconsin, Madison; Plant Dis. 85:1029, 2001; published on-line as D-2001-0702-01N, 2001. Accepted for publication 24 June 2001.

Soybean has been increasing in importance and acreage over wheat and corn for the past decade in Ohio and is now planted on 4.5 million acres. Previous surveys in Ohio of viruses infecting soybean failed to identify Bean pod mottle virus (BPMV) and soybean virus diseases have rarely caused economic losses (1). During 1999, producers in Ohio noticed virus-like symptoms in soybeans in a few isolated locations. Soybeans with green stems, undersized and "turned up pods" were collected from Union, Wood and Wyandot Counties during October 1999 and soybeans with crinkled, mottled leaves were collected in Henry, Licking and Sandusky during August 2000. Five to six plants were collected from a single field from each county each year. In 1999, samples were sent to the University of Wisconsin-Madison, where one symptomatic leaflet/sample was ground in 3 ml of chilled phosphate buffered saline (pH 7.2). Leaf sap was placed in 1.5ml centrifuge tubes and stored at 4°C for 24 h. Sap was assayed for the presence of BPMV using an alkaline phosphatase-labeled doubleantibody sandwich enzyme-linked immunosorbent assay (DAS ELISA) for BPMV (AgDia Inc., Elkhart, IN). All samples tested were positive for BPMV. Samples collected in 1999 were also maintained at The Ohio State University in Harosoy soybean and in 2000 assayed serologically along with samples collected in 2000 for BPMV and Soybean mosaic virus (SMV) by ELISA and for Tobacco ringspot virus (TRSV) and Bean yellow mosaic virus (BYMV) by a host-range symptom assay; SMV, BYMV and TRSV had been identified from soybean in previous Ohio surveys. Soybean leaf samples were assayed using F(ab')<sub>2</sub>-Protein A ELISA with antiserum prepared in 1968 to a southern U.S. isolate of BPMV and to an Ohio isolate of Soybean mosaic virus (SMV) prepared in 1967, both stored at -20°C. Diseased and non-symptomatic soybean leaf samples were ground in 4 ml 0.025M Tris pH 8.0, 0.015M NaCl and 0.05% Tween 20. Extracts were tested for BPMV and SMV by ELISA following a protocol described elsewhere (2). All of the samples collected during 1999 and maintained in the greenhouse tested positive for both BPMV and SMV while all of those samples collected during 2000 tested positive for BPMV and negative for SMV. Host-range symptom assays were conducted with leaf extracts prepared by grinding 1 g tissue:10 ml potassium phosphate buffer, pH 7.0. Extracts were inoculated by leaf rub method to Harosoy soybean, Phaseolus vulgaris cvs. Red Kidney and Bountiful, cowpea, and cucumber. The host-range symptom assays of both the 1999 and 2000 samples were negative for TRSV and BYMV; cowpea failed to express local lesions and cucumber systemic mosaic characteristic of TRSV infection and the two Phaseolus cultivars the yellow mosaic characteristic of BYMV infection. These results indicate that both BPMV and SMV were present in the samples in 1999 but only BPMV in 2000. The distribution of BPMV within Ohio and economic impact of this virus have yet to be determined. This is the first report of BPMV in Ohio.

*References*: (1) A. F. Schmitthenner and D. T. Gordon. Phytopathology 59:1048, 1969. (2) R. Louie et al. Plant Dis. 84:1133-1139, 2000.

**First Report of Ceratocystis Wilt of** *Acacia mearnsii* **in Uganda.** J. Roux and M. J. Wingfield, Tree Pathology Co-operative Program (TPCP), Forestry and Agricultural Biotechnology Institute (FABI), University of Pretoria, Pretoria 0002, South Africa; and D. Mujuni Byabashaija, Forestry Research Institute, P.O. Box 1752, Kampala, Uganda. Plant Dis. 85:1029, 2001; published on-line as D-2001-0622-01N, 2001. Accepted for publication 8 June 2001.

*Ceratocystis albofundus*, the cause of Ceratocystis wilt of *Acacia mearnsii*, is known only from South Africa. The only known hosts of this fungus are *A. mearnsii*, *Acacia decurrens*, and two species of *Protea* (1). This pathogen causes stem cankers, xylem discoloration, wilt, and the death of susceptible *A. mearnsii* trees in South Africa, leading to considerable losses to the forestry industry (1). During a recent survey of

forest plantation diseases in Uganda, A. mearnsii trees with "streaked" discoloration of the xylem, typical of Ceratocystis infection, were found in southwestern Uganda. These trees had been damaged mechanically by the harvesting of side branches and/or stems for firewood and construction. Xylem discoloration was spreading through the trees from these wounds. Trees showed typical stem cankers and gummosis, which is associated with C. albofundus infection, as well as foliage wilting. Isolations from infected trees yielded a fungus that was similar morphologically to C. albofundus, with typical hat-shaped ascospores and light-colored perithecial bases (2). Sequencing of the internal transcribed spacer region of the ribosomal RNA operon of Ugandan isolates (CMW5329, CMW5964, GenBank accession no. AF388947) confirmed their identification, grouping them with C. albofundus and separating them from all other Ceratocystis species. This is the first report of C. albofundus from a country other than South Africa. C. albofundus is an important pathogen, and strategies to reduce losses need to be established in Uganda because the aggressiveness of C. albofundus to A. mearnsii has been shown in inoculation experiments (1).

References: (1) Morris et al. Plant Pathol. 42:814, 1993. (2) Wingfield et al. Syst. Appl. Microbiol. 19:191, 1996.

Association of a Monopartite Begomovirus with Yellow Mosaic Disease of Pumpkin (*Cucurbita maxima*) in India. R. Singh, S. K. Raj, and G. Chandra, Plant Virology Division, National Botanical Research Institute, Lucknow-226 001, India. Plant Dis. 85:1029, 2001; published on-line as D-2001-0612-01N, 2001. Accepted for publication 21 May 2001.

Pumpkin (Cucurbita maxima) is an economically important vegetable crop of the family Cucurbitaceae. Yellow mosaic in C. maxima was observed in and around Lucknow, India, during the winter season of 1999-2000. High populations of whiteflies (Bemisia tabaci) were associated with diseased plants. Whiteflies were allowed to feed on diseased C. maxima plants and placed on healthy C. maxima seedlings. Symptoms of yellow mosaic disease were successfully reproduced in the seedlings. These results suggested a possible involvement of a geminivirus with the disease. Total DNA was extracted from infected and uninfected C. maxima seedlings by the CTAB method. DNA samples were used as templates in polymerase chain reaction (PCR) using degenerate primers A (5'-TAATATTACCKGWKGVCCSC-3') and B (5'-TGGACYTTRCAW-GGBCCT TCACA-3'), which have been designed to amplify a region of about 500 to 600 nt from DNA-A of a majority of begomoviruses (1). The PCR products obtained were analyzed on a 1.5% agarose gel. The major amplicon obtained from the DNA of infected plants was about 550 bp as expected from these primers. However, no such amplicon was observed in DNA samples from uninfected plants. The amplicon obtained from PCR was cloned in a suitable cloning vector. Three separate clones were sequenced and the corrected sequence data (GenBank Accession No. AF381977) was compared with the corresponding sequences of other known geminiviruses. The highest identity (88%) was with a strain of monopartite Tomato leaf curl virus (TLCV) from southern India (GenBank Accession No. U38239). The first 34 nucleotides showed significant identity (91% and 97%, respectively) with Althea rosea enation mosaic virus (GenBank Accession No. AF014881) and TLCV strain D2 reported from Australia (GenBank Accession No. AF084006), both of which are monopartite begomoviruses. These results establish the provisional identity of the pathogen causing yellow mosaic disease on pumpkin in India as a closely related strain of the monopartite TLCV. The yellow leaf disease of muskmelon in Thailand and watermelon leaf curl disease in Pakistan also were recently reported to be caused by an Indian strain of TLCV (2,3). However, to our knowledge this is the first report of detection of a monopartite geminvirus causing yellow mosaic disease on pumpkin in India.

*References:* (1) D. Deng et al. Ann. Appl. Biol. 125:327, 1994. (2) S. H. Khan et al. Plant Dis. 84: 102, 2000. (3) K. Sarmretwanich, Plant. Dis. 84:707, 2000.

(Disease Notes continued on next page)

## Disease Notes (continued)

First Report of Thiabendazole-Resistant Isolates of *Fusarium* sambucinum Infecting Stored Potatoes in Nova Scotia, Canada. R. D. Peters, I. K. Macdonald, and K. A. MacIsaac, Agriculture and Agri-Food Canada, Crops and Livestock Research Centre, P.O. Box 1210, Charlottetown, PE, Canada C1A 7M8; and S. Woodworth, Cavendish Agri Services, P.O. Box 247, Kentville, NS, Canada B4N 3W4. Plant Dis. 85:1030, 2001; published on-line as D-2001-0719-04N, 2001. Accepted for publication 19 April 2001.

Fusarium dry rot is a significant postharvest disease of potato (Solanum tuberosum L.) and is often controlled by applying thiabendazole to tubers prior to storage. However, thiabendazole-resistant isolates of Fusarium spp. have been reported from Europe (2), the United States (1), and Canada (1,4). To address concerns, samples of potato tubers showing symptoms of dry rot caused by Fusarium spp. were collected from three storage bays in a commercial storage facility in Nova Scotia, Canada, in February 2001. All tubers had been treated with thiabendazole after harvest and prior to storage. Tubers were cut longitudinally, and small tissue samples  $(10 \times 5 \times 3 \text{ mm})$  were taken from the margins of internal necrotic regions with a sterile scalpel, surfacesterilized in 0.6% sodium hypochlorite for 15 s, rinsed twice in sterile distilled water (SDW), and blotted dry on sterile filter paper. Tissue pieces were plated on 0.5-strength potato dextrose agar (PDA) amended with tetracycline (0.05 g/liter) and streptomycin sulfate (0.1 g/liter). Petri dishes were incubated in the dark at 22°C for 4 to 7 days. After incubation, hyphal tips from the margins of actively growing isolates were removed with a sterile probe and plated on 0.5-strength PDA to generate pure cultures. Of 35 potato tubers examined, 10 (29%) yielded Fusarium isolates for further study. All 10 isolates were identified as F. sambucinum Fuckel according to Nelson et al. (3). Agar plugs (5 mm diameter) taken from the margins of 7- to 10-day-old cultures of F. sambucinum isolates were transferred to petri dishes containing 0.5strength PDA amended with thiabendazole at 0, 1, 5, 10, 20, 50, or 100 mg/liter. Thiabendazole was prepared as a stock solution in SDW and added to molten agar after autoclaving. Cultures were grown in the dark for 7 days at 22°C, after which mycelial growth diameter was measured using digital calipers. Two measurements, along orthogonal diameters, were taken from each of three replicate plates for a total of six measurements per thiabendazole concentration. Means were calculated, and the diameter of the inoculation plug was subtracted from each mean. Calculated EC<sub>50</sub> values (thiabendazole concentration inhibiting pathogen growth by 50%) were obtained by regression of the log of the chemical concentration against the corresponding probit of percent fungal inhibition. All isolates of F. sambucinum were resistant to thiabendazole, with  $EC_{50}$  values ranging from 7 to 82 mg/liter. Six isolates had  $EC_{50}$ values between 40 and 82 mg/liter. Control isolates of F. sambucinum, F. avenaceum, F. solani, and F. oxysporum were sensitive to thiabendazole, with EC<sub>50</sub> values of <1 mg/liter. Although isolates of F. sambucinum resistant to thiabendazole have been recovered from eastern Canada (1,4), this is the first report of thiabendazole resistance in F. sambucinum isolates from tubers in commercial storage in the Annapolis Valley of Nova Scotia, Canada, a production region that concentrates on growing processing potatoes for the potato chip industry and is several hundred kilometers from other potato-growing regions of Prince Edward Island and New Brunswick.

*References*: (1) A. E. Desjardins. Am. Potato J. 72:145, 1995. (2) G. A. Hide et al. Plant Pathol. 41:745, 1992. (3) P. E. Nelson et al. 1983. Fusarium *Species: An Illustrated Manual for Identification*. Pennsylvania State University Press, University Park, PA. (4) H. W. Platt. Phytoprotection 78:1, 1997.

**First Report of** *Meloidogyne partityla* **on Pecan in New Mexico.** S. H. Thomas and J. M. Fuchs, Department of Entomology, Plant Pathology, and Weed Science, New Mexico State University, Las Cruces 88003; and Z. A. Handoo, USDA-ARS Nematology Laboratory, Beltsville, MD 20705. Plant Dis. 85:1030, 2001; published on-line as D-2001-0719-06N, 2001. Accepted for publication 6 July 2001.

For several years, decline was observed in mature pecan (*Carya illinoensis* (F.A. Wangenheim) K. Koch) trees in an orchard in Dona Ana County, New Mexico despite normal fertilization and irrigation practices. Affected trees were growing in sandy soil in two widely separated irrigation terraces and exhibited chlorosis of foliage and substantial dieback of branches in the upper canopy. Examination of feeder roots

revealed the presence of numerous small galls and egg masses, with rootknot nematode females often visibly protruding from root tissue. Attempts to culture the nematode on tomato (Lycopersicon esculentum Mill. 'Rutgers') were unsuccessful. Females and egg masses were collected from fresh pecan roots and sent to the USDA Nematology Laboratory in Beltsville, MD, in October 2000, where specimens were identified as Meloidogyne partityla Kleynhans (1) based on morphological examination. This is the first report of M. partityla from New Mexico, and the second report of this nematode outside South Africa. Starr et al. (2) first reported M. partityla from pecan in the United States in 1996, after recovering the nematode from five orchards in Texas. In their study, the host range of M. partityla was limited to members of the Juglandaceae, which may explain the inability of the New Mexico population to reproduce on tomato. Additional information is needed regarding distribution of this nematode within pecan-growing regions throughout North America.

*References*: (1) K. P. N. Kleynhans. Phytophylactica 18:103, 1986. (2) J. L. Starr et al. J. Nematol. 28:565, 1996.

**Cuban Isolate of** *Bean golden yellow mosaic virus* is a Member of the Mesoamerican BGYMV Group. A. L. Echemendía, P. L. Ramos, R. Peral, A. Fuentes, G. González, J. Sanpedro, and F. Morales. First and fifth authors, Instituto de Investigaciones de Sanidad Vegetal, Cuba; sixth author, Laboratorio Provincial de Sanidad Vegetal de Holguín; seventh author, Centro Internacional de Agricultura Tropical, Colomá; second, third, and fourth authors, Centro de Ingeniería Genética y Biotecnología, P.O. Box 6162. La Habana. CP 10600. Cuba. E-mail: pedro.ramos@cigb.edu.cu. Plant Dis. 85:1030, 2001; published on-line as D-2001-0712-01N, 2001. Accepted for publication 4 June 2001.

In Cuba, the emergence of bean golden mosaic was associated with high populations of Bemisia tabaci in common bean (Phaseolus vulgaris L.) plantings in the 1970s (1). During the last two decades, the disease has caused significant economic losses, forcing some growers to abandon bean production. In Holguín, one of the main bean producing provinces of the country, about 2,000 ha of beans were abandoned in 1991 due to the high incidence of this whitefly-transmitted virus. At that time, yield losses associated with this disease reached 90 to 100% in farmer's fields. In spite of various control measures, the disease affected 33, 28, and 6.5% of the total area planted in Cuba to common bean in 1990, 1992, and 1996, respectively. For this investigation, common bean leaves showing systemic yellowing symptoms were collected in fields located in the provinces of Havana, Matanzas, and Holguín during 1998-1999. Sap and total DNA leaf extracts were used to inoculate healthy bean plants by manual and biolistic procedures, respectively. Characteristic yellowing symptoms were more efficiently reproduced using a particle gun device than by manual inoculation (18/20 plants and 5/20 plants, respectively, for a Holguín virus isolate). DNA extracts were further analyzed by polymerase chain reaction using two degenerate primer sets: PAL1v1978-PAR1c715 and PAL1c1960-PAR1v722 (2). Fragments of approximately 1.4 and 1.2 kb were amplified and cloned. Restriction fragment length polymorphism analysis of the cloned 1.4-kb fragments was performed with BgIII, HincII, SalI, EcoRI, PstI, and XbaI, indicating that selected isolates from the three Cuban provinces shared identical restriction patterns. The nucleotide sequence obtained from two clones of a virus isolate from Holguín, was compared to sequences available for other begomoviruses using BLAST. The Cuban isolate shared up to 94% nt sequence identity with various strains of Bean golden yellow mosaic virus (BGYMV) in the first 250 nt of the rep gene. For the common region (CR), scores were 93% for BGYMV-GA (Guatemala), 92% for BGYMV-MX (southern Mexico) and BGYMV-PR (Puerto Rico), and 91% for BGYMV-DR (Dominican Republic). The iterative sequence ATGGAG was identified in the CR of the Cuban BGYMV isolate, as reported for other BGYMV isolates. Finally, the Cuban begomovirus, hereafter referred to as BGYMV-CU, shared nt and aa sequence identities of 94 and 100%, respectively, with the coat protein gene of BGYMV-MX. We conclude that the begomovirus isolated from mosaic-affected common bean plants in the province of Holguín is a member of the Mesoamerican BGYMV group (3).

*References*: (1) N. Blanco and C. Bencomo. Cienc. Agric. 2:39, 1978. (2) M. R. Rojas et al. Plant Dis. 77:340, 1993. (3) Morales and Anderson, Arch. Virol. 146:415, 2001.

First Report of *Phomopsis longicolla* from Velvetleaf Causing Stem Lesions on Inoculated Soybean and Velvetleaf Plants. S. Li and C. A. Bradley, Department of Crop Sciences; G. L. Hartman, USDA, ARS, and Department of Crop Sciences; and W. L. Pedersen, Department of Crop Sciences, University of Illinois, 1101 W. Peabody Dr., Urbana 61801. Plant Dis. 85:1031, 2001; published on-line as D-2001-0723-01N, 2001. Accepted for publication 11 July 2001.

Reddish brown lesions were observed on the lower stem and upper root area of velvetleaf (Abutilon theophrasti Medik.) plants growing in an Illinois soybean field in June 2000. The lesions were similar in appearance to those caused by Rhizoctonia root rot of soybean. Stems and roots with lesions were cut into ≈5-mm pieces, surface-disinfested, and placed on 2% water agar at pH 4.5. The cultural morphology of the two isolates fit the description of *Phomopsis longicolla* Hobbs (1). Colonies on potato dextrose agar (PDA) were floccose, dense, and white. The undersides of the cultures were colorless. Stromata were large, black, and spreading. The pattern of stromata in one isolate was effuse, and most of the stromata were immersed or semiimmersed in the medium, whereas the stromata from the other isolate were massive and prominent. Neither isolate turned green on PDA. Alpha conidia were hyaline, ellipsoidal to fusiform, and guttulate. DAPI (4',6diamidino-2-phenylindole)-stained alpha conidia were uninucleate. Beta conidia and perithecia did not occur on either PDA or oat flakes on water agar from 1 to 10 weeks at 25°C under a 12-h photoperiod. The DNA sequences of the mitochondrial small subunit rRNA genes of the two isolates were identical and shared 100% sequence identity with two P. longicolla soybean isolates that we had identified previously. Pathogenicity tests were conducted in a greenhouse by cutting the stems of 3-week-old soybean and velvetleaf plants at the second internode. Mycelial plugs (4 mm in diameter) from the margin of 1week-old cultures of the two isolates from velvetleaf and one from soybean were individually placed mycelial side down directly on the top of cut stems of 10 to 15 plants per isolate. Controls included noninoculated plants with and without PDA plugs. Plants were kept in a mist chamber in the dark at 25°C for 4 days and were then transferred to a greenhouse with a 16-h photoperiod at  $24 \pm 3^{\circ}$ C. Stem lesions were measured 7 days after inoculation. The experiment was repeated once. Mean stem lesion lengths caused by the velvetleaf and soybean isolates were 23 and 20 mm, respectively, on soybean stems, while negative controls produced no lesions. Mean stem lesion lengths caused by the velvetleaf and soybean isolates were 23.5 and 12 mm, respectively, on velvetleaf stems. P. longicolla was reisolated from the stem lesions of five randomly collected plants. This is the first report of P. longicolla being isolated from velvetleaf and causing stem lesions on inoculated soybean and velvetleaf plants.

Reference: (1) T. W. Hobbs et al. Mycologia 77: 535, 1985.

**First Report of Tan Spot on Wheat in Pakistan.** S. Ali and L. J. Francl, Department of Plant Pathology, North Dakota State University, Fargo 58105; S. Iram and I. Ahmad, Crop Diseases Research Institute, Islamabad, Pakistan. Plant Dis. 85:1031, 2001; published on-line as D-2001-0719-01N, 2001. Accepted for publication 6 July 2001.

Tan spot is caused by Pyrenophora tritici-repentis and is an economically important foliar disease of wheat worldwide. The fungus produces two types of symptoms, necrosis and chlorosis, on susceptible wheat cultivars. Isolates have been grouped into five races based on their ability to induce necrosis (nec+) and chlorosis (chl+) on appropriate wheat differentials (1,2). During March 2000, foliar diseases of wheat were surveyed in major wheat-growing areas of the Punjab Province of Pakistan. Tan spot was observed at 13 locations in the province. Diseased leaf samples were collected from all 13 locations to satisfy Koch's postulates. Isolations were made by placing 2-cm-long diseased leaf pieces in petri dishes with three layers of dampened Whatman No. 1 filter paper. The leaf pieces were incubated under an alternating cycle of 24 h of light at 21°C and 24 h of dark at 16°C. A fungus that produced erect, single dark yellow-brown conidiophores with single light yellow-brown conidia was recovered from all the samples and identified as Drechslera tritici-repentis, the

anamorph of *Pyrenophora tritici-repentis*. Single spores were transferred on V8 potato dextrose agar for further study. Seven single-spore isolates recovered from the samples were imported through APHIS and tested for pathogenicity and race structure in a growth chamber at North Dakota State University. Two-week-old seedlings of wheat differentials were inoculated individually with a spore suspension of each isolate, and consistent results were found in four replicated experiments. Four of seven isolates were identified as race 1 (nec+chl+), whereas, three isolates did not correspond to any of the currently identified five races. For all these isolates, one of the differentials (Katepwa), which exhibits necrosis, chlorosis, or neither symptom to the first report of the occurrence of tan spot of wheat in Pakistan. The widespread presence of tan spot in the region is a potential threat to wheat production.

*References*: (1) De Wolf et al. Can. J. Plant Pathol. 20:349, 1998. (2). Lamari et al. Can. J. Plant Pathol. 17:312, 1995.

Detection of Cucurbit Yellow Vine Disease in Squash and Pumpkin in Massachusetts. R. L. Wick and J. Lerner, University of Massachusetts, Department of Plant Pathology, Amherst 01103; S. D. Pair, USDA-ARS, Lane, OK 74555; J. Fletcher, Department of Entomology and Plant Pathology, U. Melcher, Department of Biochemistry and Molecular Biology, Oklahoma State University, Stillwater 74078; F. Mitchell, Texas Agricultural Experiment Station, Stephenville 76401; and B. D. Bruton, USDA-ARS, Lane, OK 74555. Plant Dis. 85:1031, 2001; published on-line as D-2001-0717-01N, 2001. Accepted for publication 27 June 2001.

Commercial plantings of summer squash in Charlemont, Franklin County, MA, were decimated in 1999 by 100% incidence of a yellowing disease resembling cucurbit yellow vine disease (CYVD) (1). Both plantings were established in the same field during the third week of May, one with transplants and the second by direct-seeding. Each planting consisted of four 30-m rows each of yellow zucchini (Cucurbita pepo cv. Gold Rush), summer squash (C. pepo cv. Seneca Prolific), and zucchini (C. pepo cv. Condor). Crops were produced organically and pyrethrum was used to control a high infestation of squash bugs, Anasa tristis (De Geer) (Heteroptera:Coreidae), a putative vector of CYVD (3). Just prior to fruit set, during the first two weeks of June, plants began showing symptoms of foliar chlorosis, plant stunting, or both. All of the plants in the field eventually wilted and collapsed. Cross-sections of the below-ground stem and primary root revealed a honey-brown phloem discoloration and healthy appearing xylem, symptoms characteristic of CYVD. Plants yielded marketable fruit for only about 1 week. When plant samples were tested by polymerase chain reaction (PCR) with CYVD bacterium specific primers (2), a band of the expected size for the CYVD bacterium, identified as Serratia marcescens based on 16s rDNA and groE sequence analyses (4), was amplified in every case. Since all plant samples collected were symptomatic and PCR positive for S. marcescens, asymptomatic greenhouse plants were run simultaneously as a control. All control plants tested negative. A third planting, similar to the two disease-affected plantings and containing the same three squash cultivars from the same seed lot, was established at about the same time approximately 3 km away. No symptoms of CYVD occurred at this site, further evidence that the pathogen is not seedborne (1). Furthermore, squash bugs were not observed in this field. In 2000, the disease was observed in a planting of 'Atlantic Giant' pumpkin in Erving, Franklin County, MA, and confirmed by PCR. Until now, CYVD has been reported only in the states of Oklahoma, Texas, and Tennessee. Confirmation of the disease in Massachusetts significantly increases the known geographical range of CYVD to include the New England area.

*References*: (1) B. D. Bruton et al. Plant Dis. 82:512-520, 1998. (2) U. Melcher et al. Phytopathology 89:S95, 1999. (3) S. D. Pair et al. Pages 145-148 in: Proc. 19th Ann. Hort. Conf., Okla. State Univ. (4) J. Rascoe et al. Phytopathology 90:S63, 2000.

(Disease Notes continued on next page)

## Disease Notes (continued)

**First Report of Aflatoxin in Dried Yam Chips in Benin.** S. Bassa and C. Mestres, CIRAD/FSA-UNB, 01 BP 5667, Cotonou, Bénin; D. Champiat, CIRAD, 34398 Montpellier Cedex 5, France; and K. Hell, P. Vernier, and K. Cardwell, IITA, 08 BP 932, Cotonou, Bénin. Plant Dis. 85:1032, 2001; published on-line as D-2001-0723-03N, 2001. Accepted for publication 13 July 2001.

Dried yam (Dioscorea spp.) chips are widely consumed in Bénin but are often attacked by molds. Invasion of food by Aspergillus flavus may lead to aflatoxin contamination. We report here the result of a survey on the sanitary quality of dried yam chips in Bénin. During July and August 2000, 50 dried yam chips samples were collected from different points in the marketing chain; 10 samples were collected from each of 5 stages: producers, wholesalers, retailers, dried yam-based food sellers and consumers. Aflatoxin content was assayed by the bio-luminescence method (1) after methanol/water extraction. Aflatoxins were detected in all dried yam chip samples, with levels ranging from 2.2 to 200 ppb and a mean value of 14 ppb. An aflatoxin concentration higher than the European Union's maximum residue limit (MRL) of 4 ppb was found in 98% of the samples (N = 50), while 6% had an aflatoxin concentration higher than the World Health Organization's MRL of 20 ppb. Molds were analyzed from two samples, each with aflatoxin levels around 5 ppb, on colony unit medium specific for A. flavus (2). Aspergillus spp. were detected in the inner part of dried yam chips of both samples, with a mean level of 9,000 CFU/g. Fusarium colonies were also present but were not identified to species.

*References*: (1) D. Champiat and J. Larpent. Bio-chimie-luminescence: Principes et Applications. Masson, Paris, France. 1993. (2) P. J. Cotty. Mycopathologia 125:157, 1994.

**First Report of** *Botrytis cinerea* **on Kenaf in South Africa.** W. J. Swart and M. T. Tesfaendrias, Department of Plant Pathology, University of the Free State, P.O. Box 339, Bloemfontein 9300, South Africa; and J. Terblanche, ARC Institute for Industrial Crops, Private Bag X2075, Rustenburg 0300, South Africa. Plant Dis. 85:1032, 2001; published on-line as D-2001-0719-05N, 2001. Accepted for publication 12 July 2001.

Kenaf (Hibiscus cannabinus L.) (Malvaceae) is a source of highquality cellulose fibers and is being investigated in South Africa with a view to commercial production. In April 2001, 20 to 30% of 5-month-old kenaf plants grown from seed in experimental plots near Rustenburg, Northwest Province, South Africa, were affected by gray mold caused by Botrytis cinerea Pers.: Fr. Infected plants displayed brown necrotic areas that girdled the stem, resulting in wilting and lodging in at least 50% of observed cases. Symptoms included extensive growth of mycelia and gray conidia on stem lesions. Microscopic examination revealed hyaline, one-celled conidia and conidiophores conforming to the description of B. cinerea. Plating of diseased stem tissue on malt extract agar (MEA) consistently yielded B. cinerea. Koch's postulates were satisfied by applying toothpick tips (5 mm) colonized by B. cinerea on MEA to the stems of 10 120-day-old greenhouse-grown plants of each of five kenaf cultivars. A colonized toothpick tip was placed on the stem of each of five plants per cultivar at a point ≈15 cm above soil level. Another five plants of each cultivar were wounded once using a sharp dissecting needle, and a colonized toothpick tip was placed on top of each wound. Corresponding control treatments consisted of five additional plants per cultivar, each wounded and mock-inoculated with sterile toothpick tips.

Inoculation points were wrapped in Parafilm. The experiment was conducted twice. Developing lesions were measured after 7 days. Mean lesion lengths for the two treatments, nonwounded and wounded, on the five cultivars were, respectively: 32.4 and 35.2 mm for Everglades 41; 14.9 and 53.8 mm for Cuba 108; 39.5 and 55.8 mm for El Salvador; 19.0 and 44.3 mm for SF459; and 12.4 and 43.9 mm for Tainung 2. The Newman-Keuls multiple comparison test revealed no significant difference (P < 0.05) in means among cultivars for the wounded treatment. For the nonwounded treatment, Everglades 41 and El Salvador were significantly more susceptible (P < 0.05) than the three remaining cultivars. No lesions developed on control treatments. The fungus was reisolated on MEA from all artificially inoculated plants. The pathogen is reported to cause serious losses in yield and fiber quality of kenaf in Spain (1). This is the first report of B. cinerea on kenaf in South Africa, and its potential impact on kenaf production in this country should be taken seriously.

Reference: (1) A. De Cal and P. Melgarejo. Plant Dis. 76:539, 1992.

First Record of *Pea enation mosaic virus* Naturally Infecting Chickpea and Grasspea Crops in Syria. K. M. Makkouk and S. G. Kumari, Virology Laboratory, Germplasm Program, ICARDA, P.O. Box 5466, Aleppo, Syria; and D.-E. Lesemann, Federal Biological Research Centre for Agriculture and Forestry, Institute for Plant Virology, Microbiology and Biosafety, Messeweg 11-12, D-38104 Braunschweig, Germany. Plant Dis. 85:1032, 2001; published on-line as D-2001-0719-02N, 2001. Accepted for publication 10 July 2001.

Virus-like symptoms not commonly encountered on most chickpea (Cicer arietinum L.) and grasspea (Lathyrus sativus L.) genotypes were noticed at the ICARDA farm near Aleppo, Syria, during April and May 2001. Primary symptoms included stunting, accompanied by leaf mottling and yellowing. The causal agent was transmitted by the pea aphid (Acyrthosiphon pisum Harris) in a persistent manner. Efficiency of transmission was 100% when aphids acquired the virus from grasspea and then inoculated lentil, whereas transmission efficiency was 21% when aphids acquired the virus from chickpea and then inoculated lentil. Samples of symptomatic chickpea and grasspea reacted strongly with the antiserum prepared against a Dutch isolate (E154) of Pea enation mosaic virus (PEMV), provided by L. Bos (Wageningen, the Netherlands) (1), using tissue blot immunoassay (2). Negatively stained preparations from chickpea and grasspea revealed typical PEMV-like isometric particles ≈30 nm in diameter. With immunoelectron microscopy, these particles were effectively trapped and strongly decorated with PEMV antibodies (immunoglobulin G diluted 1:10) provided by M. Musil (Bratislava, formerly Czechoslovakia) (4). The virus capsid protein was 22 kDa based on sodium dodecyl sulfate-polyacrylamide gel electrophoresis, typical of the PEMV coat protein, and reacted strongly with PEMV antiserum (E154) in western blots. This is the first report of PEMV naturally infecting chickpea and grasspea in Syria and, to our knowledge, the first report in West Asia. PEMV reached epidemic levels on lentil in Syria for the first time in 1994 (3). Field symptoms observed in May 2001 suggest that PEMV may also seriously affect lentil, chickpea, and grasspea crops in Syria.

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