

**Infection of Radish in Idaho by Beet Transmitted Virescence Agent.** M. E. Shaw, D. A. Golino, USDA-ARS, and B. C. Kirkpatrick, Department of Plant Pathology, University of California, Davis. *Plant Dis.* 74:252, 1990. Accepted for publication 5 December 1989.

In 1988, approximately 10% of radishes (*Raphanus sativus* L.) grown in seed fields near Moscow, Idaho, had symptoms of virescence, phyllody, and reduced seed set. Pleomorphic, membrane-bound mycoplasma-like organisms (MLOs) were observed in the phloem sieve elements of infected plants. Healthy *Circulifer tenellus* (Baker) transmitted a virescence agent from the radish plants to periwinkle (*Catharanthus roseus* (L.) G. Don), and transmission to daikon (*R. sativus* cv. Longipinnatus) caused a premature induction of flowering. Vector specificity, symptomatology, and detection of MLOs in the phloem of infected plants all suggest that the disease is caused by the beet leafhopper transmitted virescence agent (BLTVA) (1). Characteristic symptoms on inoculated wild tobacco (*Nicotiana rustica* L.), plantain (*Plantago* L. sp.), and celery (*Apium graveolens* L.) support this diagnosis. In addition, infected plants were positive by ELISA using a polyclonal antiserum to the BLTVA (2).

*References:* (1) D. A. Golino et al. *Phytopathology* 77:954, 1987. (2) D. A. Golino et al. *Phytopathology* 79:1139, 1989.

**First Report of *Magnaporthe poae*, Cause of Summer Patch on Annual Bluegrass, in Ohio.** J. C. Stier and W. W. Shane, Department of Plant Pathology, The Ohio State University, Columbus 43210. *Plant Dis.* 74:252, 1990. Accepted for publication 4 December 1989.

*Magnaporthe poae* Landschoot & Jackson (2), the cause of summer patch (1), was isolated from symptomatic annual bluegrass (*Poa annua* L.) in golf course putting greens at Cincinnati and Columbus during the summer of 1988. Small (<15 cm) chlorotic patches typical of the early symptoms of summer patch consisted of plants with decayed crowns and discolored roots covered with dark ectotrophic hyphae. The teleomorph, required for accurate identification, was produced in vitro (acidified potato-dextrose agar) only when paired with the compatible mating type (2). Perithecia, asci with refractive apical rings, and ascospore morphology and measurements were similar to those reported by Landschoot and Jackson (2). Pathogenicity of Ohio isolates was verified with Koch's postulates in greenhouse studies. Kentucky bluegrass (*P. pratensis* L. 'S-21') and *P. annua* were inoculated with millet seed infested with *M. poae* at the time of planting in sterilized sand. This is the first published report of *M. poae* in Ohio.

*References:* (1) P. J. Landschoot and N. Jackson. *Phytopathology* 77:119, 1987. (2) P. J. Landschoot and N. Jackson. *Mycol. Res.* 93:59, 1989.

**New Bacterial Disease of *Lobelia erinus* Cultivar *Richardii* Caused by *Xanthomonas campestris*.** G. Poschenrieder, Bayerische Landesanstalt für Bodenkultur und Pflanzenbau, Voettingerstr. 38, D-8050 Freising; E. Lohweg and W. W. P. Gerlach, Staatliche Versuchsanstalt für Gartenbau Weihenstephan, Institut für Botanik und Pflanzenschutz, D-8050 Freising 12, West Germany; and J. D. Janse, Plantenziektenkundige Dienst, Geertjesweg 15, NL-6700 HC Wageningen, Netherlands. *Plant Dis.* 74:252, 1990. Accepted for publication 5 December 1989.

A leaf blight and dieback of *Lobelia erinus* L. 'Richardii' was noticed in 1988 in a commercial nursery near Munich, West Germany (1). The disease later appeared in other glasshouse units in Germany and in 1989 was found in a greenhouse in the Netherlands containing plants obtained from Great Britain. Losses within individual houses ranged from 30 to 100%. Symptoms began as a water-soaking, followed by chlorosis of leaf bases and adjacent areas on the stems. These areas became necrotic, leading to death of acropetal portions of the stem. Water-soaking and chlorosis initiated at leaf tips were followed by necrosis that developed interveinally. Occasionally, droplets of bacterial ooze were seen. Gradually the plants died, especially those

that were watered overhead. Isolations from diseased tissue as well as symptomless plants consistently yielded only a yellow bacterium. Based on results of standard physiological and biochemical tests, as well as fatty acid analysis, the bacterium was identified as a new pathovar of *Xanthomonas campestris* (Pammel) Dowson. Spray inoculations of the causal bacterium ( $1 \times 10^8$  cfu/ml) onto healthy seedlings reproduced the symptoms seen on naturally infected plants. The suspect pathogen was reisolated from the inoculated plants, which died within 4 wk. The bacterium was pathogenic to artificially inoculated *L. fulgens* L. 'Queen Victoria' plants, but the only symptom was severe stunting. This is the first report of a disease in *Lobelia* caused by a pathovar of *X. campestris*.

*Reference:* (1) G. Poschenrieder et al. *Gb + Gw* 88:2204, 1988.

**First Report of Tan Spot Caused by *Drechslera tritici-repentis* on Winter Wheat in Arkansas.** M. C. Hirrell, Southeast Research and Extension Center, Monticello, AR 71655; J. P. Spradley, Cooperative Extension Service, Little Rock, AR 72203; J. K. Mitchell, Department of Plant Pathology, University of Arkansas, Fayetteville 72701; and E. W. Wilson, Cooperative Extension Service, Morrilton, AR 72110. *Plant Dis.* 74:252, 1990. Accepted for publication 21 November 1989.

*Drechslera tritici-repentis* (Died.) Shoem., causal agent of tan spot of wheat (*Triticum aestivum* L.), was isolated from diseased soft red winter wheat plants (cv. Florida 302) in Conway County, Arkansas, in March 1989. Diagnosis was confirmed by the production of conidia 45–200  $\mu$ m long with a conical basal cell on agar media and by completion of Koch's postulates. The 81-ha field had a history of reduced tillage and summer fallow since 1986. At soft dough (Feekes' growth stage 11.1–11.2), disease incidence (based on plants) was greater than 95% and disease severity of flag leaves ranged from 1 to 10% of leaf area affected. The yield was 1,210 kg/ha, a decrease of approximately 65% for typical yields of Florida 302 in 1989. Wheat straw from the previous year was examined for the teleomorph (*Pyrenophora tritici-repentis* (Died.) Drechs.), and pseudothecia were observed to be empty. This is the first report of this disease in the Mississippi Delta area.

**Occurrence of Three Races of *Xanthomonas campestris* pv. *vesicatoria* on Pepper and Tomato in Taiwan.** G. L. Hartman and C. H. Yang, Asian Vegetable Research and Development Center, Shanhua, Tainan, Taiwan, 74199, Republic of China. *Plant Dis.* 74:252, 1990. Accepted for publication 10 October 1989.

Three races of *Xanthomonas campestris* pv. *vesicatoria* (Dooidge) Dye have been reported to cause bacterial spot of pepper (*Capsicum annuum* L.) based on strains collected from various locations (1,2). No report has been published on the races that occur in Taiwan, even though bacterial spot is prevalent in most fields and causes losses in quality and quantity. Diseased leaves and fruits were collected from 40 fields of pepper and 15 fields of tomato (*Lycopersicon esculentum* Mill.) from 16 distinct areas of production between 1988 and 1989. Bacteria were isolated on PDA, and single colonies were selected, increased, and stored on slants of 523 medium. Cell suspensions ( $5 \times 10^8$  cfu/ml) from 24-hr-old cultures grown on 523 medium were infiltrated into the three youngest fully expanded leaves of 1-mo-old pepper plants. A differential set of pepper lines with *Bs1*, *Bs2*, and *Bs3* genes and Early Calwonder (no known genes for resistance) were used according to Hibberd et al (2). Hypersensitive reaction was recorded within 48 hr after inoculation. Of the strains from pepper, 70, 10, and 20% were races 1, 2, and 3, respectively. In contrast, of the strains from tomato, 13, 7, and 80% were races, 1, 2, and 3, respectively. The *Bs1*, *Bs2*, and *Bs3* genes do not exist in commercial pepper cultivars in Taiwan. Thus, races 1–3 are natural components of the population and have not arisen because of selection pressure.

*References:* (1) A. A. Cook and R. F. Stall. *Plant Dis.* 66:388, 1982. (2) A. M. Hibberd et al. *Plant Dis.* 71:1075, 1987.