

M. L. Lacy  
Michigan State University, East Lansing

Richard D. Berger  
University of Florida, Gainesville

Robert L. Gilbertson and Elizabeth L. Little  
University of California, Davis

# Current Challenges in Controlling Diseases of Celery

Celery (*Apium graveolens* L. var. *dulce* (Mill.) Pers.) originated in the Mediterranean region. Although celery was originally cultivated for medicinal purposes, it was later developed into a food and flavoring crop. Celery is the third most important salad crop in the United States and is popular in most European countries (38). It is produced year-round in California, Florida, and Texas and during late spring, summer, and early fall in Michigan, Wisconsin, New York, and Ohio. Celery provides significant amounts of vitamins A and C, calcium, and sodium, and contains very little carbohydrate or fat, making it popular as a diet food.

Celery is attacked by a number of important diseases that affect yield and quality. The most important in the United States are Septoria leaf (late) blight caused by *Septoria apiicola* Speg., Cercospora leaf (early) blight caused by *Cercospora apii* Fresen., northern bacterial blight caused by *Pseudomonas syringae* pv. *apii*, and the soilborne disease Fusarium yellows caused by *Fusarium oxysporum* f. sp. *apii* (R.R. Nelson & Sherb.) W.C. Snyder & H.N. Hans. race 2 (40). Septoria leaf blight is more important in northern states; Cercospora leaf blight is more important in southern states (especially Florida); bacterial blight is important in all celery-growing states; and Fusarium yellows is a limiting factor in celery production in all states except Florida. Recent events have drawn attention to these four diseases and have led to new and important information

on all of them. We discuss the current state of knowledge on these four diseases below.

## Septoria Leaf (Late) Blight

Septoria leaf blight, caused by *S. apiicola*, is an important foliar disease of celery in the United States and in the world (40). Symptoms include irregularly shaped spots of necrotic tissue on leaves, in which are embedded spore-containing, flask-shaped structures called pycnidia (Fig. 1). The presence of pycnidia in leaf spots is an important characteristic used to distinguish Septoria leaf blight from Cercospora leaf blight. These pycnidia appear to the unaided eye similar in size and color to grains of ground black pepper. *Septoria* spp. produce long, multicellular conidia (spores) within pycnidia in leaf or petiole tissues or in seed coats (13,40). Each pycnidium is capable of releasing 1,500 to 5,400 conidia (31). Conidia within the pycnidia are surrounded by a mucilaginous matrix composed of proteins and sugars that swells when the mucilage absorbs free water or is exposed to relative humidities of 90% or higher. The conidia and mucilage are forced out in tendrils through a pore in the pycnidium (17); the mucilage dissolves in water on the leaf surface; and spores germinate and infect (40).

Leaf spots expand with time and may coalesce and eventually cause leaf death. The disease can begin in seedlings infected from diseased seeds or from infested debris in soil. Conidia are spread within or between plants by splash droplets from rainfall or overhead irrigation and by workers and machinery moving through the crop when plants are wet (12). High amounts of precipitation promote disease development in celery (3,41). If this disease is not controlled, losses can exceed 70% (27). Growers have historically applied fungicides for control at regular 5- to 10-day intervals (25).

In 1994, Lacy (26) reported that conidia of *S. apiicola* collected from dried infected celery leaves did not germinate until 7 to 12 h after placement on agar plates or on celery leaves. Time of initiation of germination was dependent on temperature and germination medium. This is a relatively long time for germination to begin compared to many other fungi. Germination of conidia was 20% at 21°C and 10% at 25°C on water agar (which contains some nutrients) 8 h after placement on water agar plates. Germination of conidia did not begin in distilled water or on celery leaves until 12 h after placement on agar plates or leaves. Germination was complete (95% or higher) on water agar and reached 78 to 80% on leaves at 21 or 25°C by 36 h after



Fig. 1. Lesions caused by *Septoria apiicola*, the causal agent of Septoria leaf (late) blight of celery.

Dr. Lacy's address is: Department of Botany and Plant Pathology, Michigan State University, East Lansing, MI 48824.  
E-mail: lacym1@pilot.msu.edu

Publication no. D-1996-0627-07F  
© 1996 The American Phytopathological Society

placement on water agar or leaves. Lesions formed on celery leaves in significant numbers (one or more lesions per leaflet) only after 24 h of continuous or interrupted (12 h wet–12 h dry–12 h wet) dew within 15 days after inoculation at 21°C. Lesions formed as early as 8 days after inoculation following wet periods of 36 to 48 h and reached a high of 14 lesions per leaflet after 21 days at 21°C. A maximum of only three lesions per leaflet formed on celery leaves exposed to 36 to 48 h of dew at 25°C after 21 days.

Results of germination studies were used to develop and evaluate weather-based spray schedules for *Septoria* leaf blight control. With a wetness period of 12 h or more as a conservative threshold value for a risk of infection, chlorothalonil sprays were applied to inoculated field plots weekly or after 12 h or more of wetness (provided that no sprays had been applied within the previous 7 days) (26). In 3 years of trials, two fewer sprays annually were applied using the 12-h leaf wetness threshold than were applied on a weekly schedule without any sacrifice in efficacy of disease control (Table 1).

Leaf wetness sensors are available on field weather instruments such as Enviro-caster (Neogen Food Tech, 620 Leshler Place, Lansing, MI 48912) and Field Monitor (Sensor Instruments Co., 41 Terrill Park Drive, Concord, NH 03301), among others. These instruments record wetness periods and store them in memory for later retrieval. Leaf wetness can also be measured and recorded on a paper chart with the deWit leaf wetness recorder (Valley Stream Farms, Orono, Ontario, Canada).

### Cercospora Leaf (Early) Blight

*Cercospora* leaf blight, caused by *C. apii*, is a very destructive disease of celery in warm, humid climates. Prior to 1975, it was not unusual for celery growers in Florida to experience 50% or more losses in yield to *Cercospora* leaf blight during the fall and spring seasons (4,42). Crop losses occur when blighted stalks (petioles) or leaves have to be removed or trimmed at harvest (42). Under extreme disease conditions, only the celery hearts may remain after trimming. Additionally, the application of fungicides to control *Cercospora* leaf blight is a major component of the total cost of growing a crop (40).

*Cercospora* leaf blight first appears as small, yellow spots visible on both sides of the leaf, which rapidly enlarge to lesions up to 10 mm in diameter containing dead tissue of a tan to gray color (Fig. 2). All aboveground tissues of celery plants can be infected by the early blight fungus, which sporulates profusely on leaf lesions during favorable weather conditions. The long (200 to 400 µm), narrow (3 to 5 µm), needlelike conidia are readily detached and

moved by wind for several kilometers. Because the conidia are so easily windborne, gradients of disease between areas are seldom seen. A wider distance between plants in the row and between rows consequently has little effect on slowing the epidemic (42). Infections take place on the younger leaves as they are formed from the hearts of plants and move into the upper canopy. After an incubation period of about 10 days, brown circular lesions appear. By this time, new leaves have formed, and leaves with lesions have been forced to the periphery of the plant. Conidia are almost always present on diseased older leaves in the humid understorey of the crop canopy.

To avoid crop loss to epidemics of early blight, celery growers use an intense program of preventive sprays of efficacious fungicides (40). During warm, rainy peri-

ods, a typical program in Florida would be one to three applications per week (20 to 40 sprays during the 3-month life of the crop in the field). The applications are made with ground rigs, fixed-wing aircraft, or helicopters.

In celery-producing areas of Florida, the crop season is 13 months long, with staggered plantings beginning with the first seedbeds in May and ending with the harvest of the last crop in June of the following year. It is during the winter period of November to March that forecasting of early blight can be used to reduce the number of applications of fungicides. After 1975, Florida growers were able to begin their celery crops with transplants that were disease-free, or nearly so, by growing their seedlings under shade houses rather than under cumbersome A-frames covered with tobacco cloth. Timing sprays with

**Table 1.** Disease severities and yields in celery plots sprayed according to leaf wetness periods or weekly with chlorothalonil

Timing of spray	Number of sprays <sup>a</sup>	% Disease <sup>b</sup>	Yield (kg/ha) <sup>c</sup>
Timed <sup>d</sup> 1991	5	0.1	58,581
Weekly 1991	7	0.1	68,868
Nonsprayed 1991	0	45.0 <sup>e</sup>	17,864 <sup>e</sup>
Timed 1992	5	0.1	51,867
Weekly 1992	7	0.0	50,204
Nonsprayed 1992	0	52.5 <sup>e</sup>	17,494 <sup>e</sup>
Timed 1993	5	0.0	61,600
Weekly 1993	7	0.0	59,444
Nonsprayed 1993	0	52.5 <sup>e</sup>	17,864 <sup>e</sup>

<sup>a</sup> Plots were sprayed with chlorothalonil as Bravo 720 6FL at 1,753 ml/ha (1.5 pints/acre).

<sup>b</sup> Percent leaf area containing *Septoria* leaf blight lesions was visually estimated.

<sup>c</sup> Weight of celery trimmed for packing.

<sup>d</sup> Timed plots were sprayed after leaf wetness periods exceeded 12 h, provided that no fungicide had been applied during the previous 7 days; weekly plots were sprayed weekly.

<sup>e</sup> Significantly different from sprayed treatments (Student-Newman-Keuhl test,  $P = 0.05$ ) in a given year. Sprayed treatments were not significantly different from each other in a given year.



**Fig. 2.** Lesions caused by *Cercospora apii*, the causal agent of *Cercospora* leaf (early) blight of celery.

forecasts works best if transplants are initially free from *Cercospora* leaf blight.

Mature conidia of *C. apii* are formed on conidiophores only during periods of high relative humidity (95% or higher) of 10 h or more duration at temperatures of 15 to 30°C (blight-favorable hours) (4). The conidia on lesions in the crop overstory are detached during the morning (0800 to 1000 hours) as temperatures rise and relative humidity decreases, accompanied by an increase in wind speed. Light rain may splash conidia to vulnerable tissues in the canopy, and heavy rain may wash conidia from the lesions and carry them to the ground. The optimum conditions for infection are the same as those for spore formation. Thus, the typical infection process encompasses conidial formation at night, dissemination the next morning, then spore germination and infection through stomata the following night. After a prolonged period of cold, dry weather, two successive nights of 10 or more blight-favorable hours are necessary to reinitiate the epidemic. Environmental conditions favorable for sporulation and dispersal were verified (4) by the use of volumetric spore samplers operated in commercial fields of celery in Florida over a period of more than 1,000 trap-days (days in the field × number of traps). The conidia of *C. apii* are readily identifiable on trapping surfaces because of their lack of color, length, needlelike shape, sharply defined base with attachment scar, and acute apex. With early blight present in celery fields, significant numbers of conidia were always found on trapping surfaces when conditions were favorable (10 or more hours of RH 95% or higher at temperatures of 15 to 30°C). When these conditions were not met, significant numbers of conidia were caught

only when the crop upwind of the samplers was disturbed by mechanical operations (primarily cultivation or harvesting) (4).

For implementation of the forecasting system, hygrothermograph charts are examined daily at about 0800 hours to determine the likelihood of the presence of inoculum for that day. If 10 or more blight-favorable hours have occurred and if no fungicide has been applied during the previous 3 days, then a spray is advised. Some growers use 2-day intervals between sprays during periods of blight-favorable weather after midseason because large areas of new, unprotected leaf tissue are rapidly produced (5). Large growers of celery in south Florida generally choose to contract weather monitoring and scouting duties to crop consultants. When farm managers conduct their own forecasting, they are frequently distracted from their monitoring duties by the problems of labor, equipment, and crop culture in their daily supervisory routine.

### Northern Bacterial Blight

*P. s. pv. apii* was first described in 1921 as the causal agent of a foliar disease of celery in New York State (22). Disease symptoms on celery are characterized initially by small, water-soaked lesions (rarely larger than 5 mm) that are roughly circular to angular and are confined to leaves. Lesions later become necrotic (Fig. 3) and can coalesce and cause leaf blight and extensive leaf tissue death under conducive conditions. This disease is referred to as northern bacterial blight to differentiate it from southern bacterial blight, a similar disease caused by *P. cichorii* (43), which is found predominantly in Florida. Northern bacterial blight has been a moderately important disease in the major cel-

ery-growing regions of the United States and Canada, and causes losses mostly due to additional trimming at harvest (40). Prior to 1989, northern bacterial blight had not been reported in California, although it had been reported in other states (40). It has since become an important disease in California.

Celery production in California is concentrated in the cool coastal counties, such as Monterey, San Benito, Santa Barbara, and Ventura, and fields are established with greenhouse-produced transplants. These transplants are grown in closely spaced plug trays in the greenhouse (Fig. 4) for 8 to 10 weeks before being planted in the field. Growers of celery transplants in California historically had few significant disease problems, except for an occasional outbreak of *Septoria* leaf blight. However, in 1989, a leaf blight disease that resembled northern bacterial blight was reported from a few celery transplant houses and production fields (23). By 1991, the disease was found in all celery-growing regions of California and was most severe on greenhouse-produced transplants (32). Disease incidence reached nearly 100% in some greenhouses (24,32), resulting in a brown, blighted appearance in plantings. The vigor and value of blighted transplants was reduced, and these transplants were occasionally rejected by growers. The disease was carried into the field on infected transplants, but there the disease was generally confined to older leaves. However, in fields with overhead irrigation, the disease often progressed onto younger foliage, which resulted in additional trimming at harvest.

Isolations made from blighted leaves collected from transplant greenhouses and from the field consistently yielded a white, fluorescent bacterium that was identified as *Pseudomonas syringae* based on the LOPAT determinative tests (Levan positive, Oxidase, Potato rot, and Arginine dihydrolase negative, and Tobacco hypersensitivity positive) (30). When tested for pathogenicity on celery, representative strains caused bacterial blight symptoms that were indistinguishable from symptoms induced by the ATCC type strain of *P. s. pv. apii*. The disease was identified as northern bacterial blight based on these results.



Fig. 3. Lesions caused by *Pseudomonas syringae* pv. *apii*, the causal agent of northern bacterial blight.



Fig. 4. Plug trays producing celery transplants in a California greenhouse.

Susceptible celery cultivars and cultural practices conducive to northern bacterial blight development had been used in celery transplant greenhouses before 1989, but the disease had not been observed. Northern bacterial blight was therefore probably introduced recently into California, as opposed to being a minor endemic problem that suddenly increased in severity. A study was initiated to determine the inoculum sources of the blight epidemic and to identify management strategies for control. Epiphytic populations of *P. s. pv. apii* and disease development were monitored for 3 years during the transplant-growing season (January to July) in three greenhouse operations in the Salinas Valley. Epiphytic *P. s. pv. apii* was not recovered from leaves until April in each of the 3 years with a leaf wash and dilution plating assay. Approximately 7 to 10 days after epiphytic populations were detected on symptomless leaves, disease symptoms were observed on transplants.

A variety of environmental factors and cultural practices were associated with the buildup of *P. s. pv. apii* populations and the development of northern bacterial blight in celery transplant greenhouses. High-pressure overhead irrigation caused wounding and water congestion in leaves, which favored infiltration of epiphytic *P. s. pv. apii* into leaves. Development of blight was most severe on rapidly growing transplants with succulent leaves. Heavy applications of nitrogen fertilizer also were associated with extremely lush and succulent foliage and persistent outbreaks of northern bacterial blight. Longer duration of leaf wetness periods was the only environmental factor associated with outbreaks of blight. These periods corresponded with the increasing water demands of transplants 6 weeks of age or older. Favorable temperature (10 to 25°C) and relative humidity (60 to 100%) conditions inside the greenhouses provided optimal conditions for increases in *P. s. pv. apii* populations on leaves and disease development. Secondary spread of *P. s. pv. apii* in greenhouses was favored by a number of factors. Mowing of transplants is a common practice that produces robust and uniform plants. This practice can rapidly spread *P. s. pv. apii* within and between greenhouses, and provide wounds for entry of bacteria into plants.

The sudden appearance of northern bacterial blight in California may have resulted from the introduction of *P. s. pv. apii* associated with celery seed. Thus, a celery seed assay was developed (33) in which the celery seed (20 g = 40,000 to 50,000 seeds) was washed in potassium phosphate buffer for 4 h at 4°C and the diluted seed wash solution was plated onto a semiselective medium (KBBC) (36). Some commercial lots of celery seed were contaminated with bacteria at levels ranging from 10 to 91 CFU/g of seed. Thus, *P.*

*s. pv. apii* can be seedborne, and seed is a potential inoculum source for northern bacterial blight.

The ability of *P. s. pv. apii* to infest celery seeds was examined in a field plot of celery plants. Young plants were inoculated with a cell suspension of the bacterium. The plants developed only a few small, inconspicuous lesions on leaves and flower umbels, but flower umbels had high epiphytic populations of *P. s. pv. apii* ( $10^4$  to  $10^6$  CFU/g fresh weight) (33). The seeds harvested from these plants were heavily contaminated with the bacterium ( $2 \times 10^5$  CFU/g of seed). Celery seedlings produced with this infested seed had high epiphytic populations of *P. s. pv. apii* ( $10^4$  to  $10^5$  CFU/g fresh weight); therefore, the source of this epiphytic population probably was infested seeds.

Various seed treatments were evaluated for the capacity to reduce bacterial contamination of the experimentally infested seed lot. Hot water treatment (50°C for 25 min) reduced contamination with *P. s. pv. apii* by 99.9% (using the seed wash assay) and did not significantly reduce seed germination.

A number of management practices for northern bacterial blight were developed for celery transplant growers. To reduce initial inoculum, celery seed should be hot water-treated (50°C for 25 min). Planting trays and greenhouses should be disinfested between celery-transplant seasons to eliminate additional carryover inoculum. Transplants should be irrigated at pressures where wounding and water congestion will not occur on leaves and during times where subsequent periods of leaf wetness will be shortest. Ventilation (i.e., by opening side vents or curtains or using fans) can be used to reduce humidity and duration of leaf wetness in greenhouses. Heavy use of fertilizers, particularly those high in nitrogen, should be minimized. Growers can reduce secondary spread of *P. s. pv. apii* by mowing transplants when leaves are dry, disinfecting mowers when they are moved to different greenhouses, and removing clipped leaf material. Movement of workers between celery houses should be restricted, especially when northern bacterial blight is present and plants are wet. In some cases, disinfection of workers' hands,



Fig. 5. Field symptoms of a severe case of Fusarium yellows of celery, caused by *Fusarium oxysporum* f. sp. *apii* race 2.

and even changes of clothing, may be necessary.

Beginning in the 1993 season and continuing into the 1994 and 1995 seasons, some or all of the above strategies were used in the three transplant operations in the Salinas Valley. These growers experienced a significant reduction in incidence and severity of northern bacterial blight, and major outbreaks have not occurred in their greenhouses since these strategies were employed.

## Fusarium Yellows

Fusarium yellows was first found in a field near Kalamazoo, Michigan, in 1914 (7). The disease caused yellowing, stunting, and withering of petioles and leaves (Fig. 5), and a red to brown discoloration in the water-conducting tissues of the crown, roots, and petioles (Fig. 6). The disease spread and grew more serious with time, and by 1931 Fusarium yellows was widespread in North America. Resistant varieties began to appear about 1939, and in 1952 an immune variety (Tall Utah 52-70), developed from a single plant found in screening trials, was introduced (19). Within a few years, most of the celery acreage in the United States was planted to this variety and its descendants, and the disease essentially disappeared. However, in 1978, Hart and Endo (19) reported that the disease had reappeared in California in about 1959 (its first occurrence since 1937) and was widespread by 1978. The causal agent was identified as a new strain of the Fusarium yellows fungus (*F. o. f. sp. apii*). The original strain attacked Golden Detroit and Fordhook varieties but not Tall Utah 52-70; the new strain attacked all three



Fig. 6. Vascular discoloration in root, crown, and basal petiole of susceptible celery caused by *Fusarium oxysporum* f. sp. *apii* race 2.

varieties (39). The original strain of *Fusarium* was given the designation race 1, while the new strain was designated race 2 (39). Race 1 caused disease mainly in the older, self-blanching varieties; whereas the newer green varieties derived from Tall Utah 52-70 and its descendants were highly resistant.

By 1987, *Fusarium* yellows was prevalent in California (39), Michigan (8), New York (2), and Texas (35). By 1991, it was also present in Canada (6,15). Florida is the only major celery-producing area in which *Fusarium* yellows is not prevalent. Summer flooding of fields in Florida, which results in anaerobic fermentation at high temperatures, keeps populations of plant parasitic nematodes in check (16). It is possible that populations of the *Fusarium* yellows fungus are also affected in Florida. Muck soils in Michigan where celery is grown are routinely flooded in winter, but this has never been observed to result in a reduction in disease level (M. L. Lacy, unpublished).

*Fusarium* yellows can be spread readily by movement of infested soil or infected transplant seedlings (40). The fungus survives for long periods in soil as resistant overwintering chlamydospores, even in the absence of a susceptible celery crop. The persistence of these chlamydospores makes short-term crop rotation ineffective once fungus populations have built up to the point where serious disease occurs. Attempts to control the disease in production fields with fungicides have been unsuccessful (2).

*Fusarium* yellows incidence increased in seedbeds, which resulted in the rapid spread of the disease by means of infected transplant seedlings. However, the disease could be controlled in seedbeds with methyl bromide fumigation or with steam pasteurization (1). More recently, many growers in the United States have abandoned seedling production in greenhouse benches filled with soil, and switched to plastic plug trays and clean artificial planting media to prevent seedling infection in the transplant production phase of celery culture.

Celery researchers quickly began looking for sources of resistance to race 2 of the *Fusarium* yellows fungus. In 1984, Orton et al. (37) released a breeding line developed at the University of California from celeriac (*Apium graveolens* var. *rapaceum*) × celery crosses designated UC-1. While UC-1 had superior resistance, its horticultural type was not acceptable. However, it was used successfully as a breeding line by seed companies. Deacon and Tall Utah 52-70 HK were the most resistant commercially available varieties in Michigan in 1986 (10). In 1992, Toth and Lacy (46) reported that newer varieties (Picador, Matador, A863, and Starlet) had increased levels of resistance to *Fusarium* yellows in Michigan. By this time, soil

infestation levels had increased sufficiently in many fields that Deacon and Tall Utah 52-70 HK were not producing acceptable crops. Since then, the resistant cultivars Picador, Peto 285, Promise, and Rocket have been introduced and are being widely grown in Michigan (28) as well as in other locations. In California, additional cultivars such as VTR 1330, VTR 1758, PSR 10490, and Conquistador have received favorable reviews in field trials (18).

In 1988, a novel method of introducing resistance into existing celery varieties that did not involve traditional breeding techniques was reported (20). Individual plantlets were induced to grow from undifferentiated celery cells grown in a sterilized liquid nutrient broth. When these plants were screened for their reaction to *Fusarium* yellows, a wide range of resistance levels was found. Some plants reacted in a more susceptible and others in a more resistant way than the parent material. This type of reaction is termed somaclonal variation. By selecting the most resistant individuals, a breeding line called UC-T3 was released (21).

Toth and Lacy (45) also used this unconventional breeding method to produce variants of Tall Utah 52-70 HK that were highly resistant compared to the moderately resistant parent line. With a program of field screening and recurrent selection, and a vernalization technique that shortens the normal seed generation time from 2 years to 1 year, five superior lines were produced and field-tested with selected growers. One selection, called MSU SHK-5, was released to commercial seed producers to be used directly in seed production or as a breeding line (29). This new line was produced in about 6 years, a shorter time than is generally required to produce a new variety using conventional breeding methods. Selections from the cultivar Florida 683, which is more horticulturally desirable but also more susceptible than Tall Utah 52-70 HK, have been used in a similar program. Release of a resistant line of Florida 683 is projected to be about 2 to 3 years in the future.

Modifications in cultural practices also promise to improve *Fusarium* yellows management. Soil populations of the *Fusarium* yellows fungus were suppressed with amendments of onion or peppermint crop residues in the greenhouse (9). Toth (44) further showed that, in a field previously monocropped to celery, populations of *Fusarium* in soil had dropped to levels undetectable by means of soil dilutions after 5 years in various vegetable crops other than celery. In contrast, soil populations of *Fusarium* were 80 propagules per gram (ppg) in the spring of 1986 (a level high enough to cause severe damage in susceptible cultivars) in an adjacent field that was planted to celery continuously for at least 20 years. After a crop of onions in summer 1986, populations were at 94 ppg

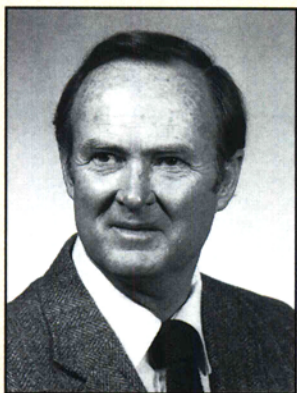
at season's end, but had dropped to 26 ppg by spring of 1987. When celery followed this onion crop in 1987, populations of the fungus had increased to 160 ppg by the end of the growing season. Michigan celery growers have been encouraged to rotate celery with onions as part of their disease management strategy. Almost all Michigan growers now practice this rotation. Where rotation is used along with resistant varieties, good crops of fresh market celery are being produced in *Fusarium*-infested soil.

## Conclusions and Outlook

**Septoria leaf blight.** Because of increasing concerns about chemical inputs into the environment and the cost-price squeeze placed on growers by the high cost of fungicide applications, it has become important to have more efficient, lower cost production without sacrificing good disease control. Spray predictors allow growers to time sprays for optimum benefit and to skip unneeded sprays during periods of nonconductive weather without compromising disease control effectiveness. The *Septoria* leaf blight predictor saves about two sprays per year compared to a weekly schedule (Table 1). We should also try to increase levels of resistance to blights in celery using either traditional breeding methods such as interspecific crosses or nontraditional methods such as somaclonal variation, discussed earlier in this paper. Even partial resistance to a disease can have an additive effect in increasing the effectiveness of fungicidal disease control (14).

**Cercospora leaf blight.** Resistance to early blight exists in certain celery cultivars (5,40), but even the most resistant cultivars available require an intense fungicide program in Florida during periods of blight-favorable weather. Celery growers in Florida and Texas save costs of fungicidal applications during the cooler and drier winter months with disease forecasting and spray timing. However, even during this time, occasional sprays of fungicide are necessary to reduce the occurrence of *Rhizoctonia* stalk rot and other diseases. During the time when weather is very favorable for sporulation of and infection by *C. apii*, epidemics of *Cercospora* leaf blight can be suppressed with frequent sprays of reduced (0.25×) rates of efficacious fungicides.

**Northern bacterial blight.** Ultimately, the most effective control of northern bacterial blight will involve the production and use of celery seed free of *P. s. pv. apii* contamination. The California Crop Improvement Association has recently established a set of guidelines for future production of certified celery seed that includes inspection of seed fields for northern bacterial blight. A test to rapidly diagnose blight was developed based on the use of the polymerase chain reaction (PCR) (34). Random amplified polymor-



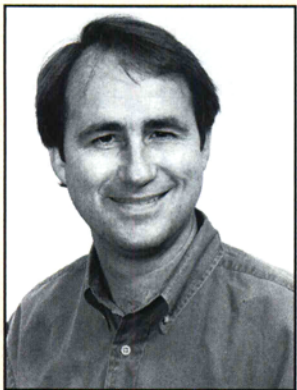
**Melvyn L. Lacy**

Dr. Lacy is professor emeritus of plant pathology at Michigan State University. He holds a B.S. in agriculture (1959) and an M.S. in plant pathology (1961) from the University of Wyoming, and a Ph.D. in plant pathology from Oregon State University. His Ph.D. research work was done on *Verticillium* wilt of mints under the guidance of C. E. Horner, USDA. Dr. Lacy began his work on celery and other vegetable crops at Michigan State University on 1 January 1965 and retired on 1 January 1996. He has interests in integrated control of both soilborne and airborne fungal diseases. Dr. Lacy's assignment was research and teaching until 1983, when he accepted a 50% extension responsibility. He has a strong interest in problem-solving and has worked closely with growers and county agents in the vegetable industry, especially since 1983. Dr. Lacy was named a fellow of APS in 1993, received the Distinguished Service Award of the north central division of APS in 1989, and was twice named outstanding extension specialist in Michigan. He resides in East Lansing and maintains an office in his department.



**Richard D. Berger**

Dr. Berger is a professor in the Department of Plant Pathology at the University of Florida, Gainesville. He received his Ph.D. from the University of Wisconsin, Madison. He was an extension plant pathologist at Pennsylvania State University before joining the faculty at the University of Florida. His research interests are quantitative and comparative epidemiology, modeling, and crop loss. Dr. Berger received the APS/Campbell Award in 1974 for the most outstanding paper on vegetable diseases published in *Phytopathology* in the preceding 2 years; the subject was epidemiology of *Cercospora* early blight of celery. He was named APS Fellow in 1985.



**Robert L. Gilbertson**

Dr. Gilbertson is an associate professor of plant pathology in the Department of Plant Pathology at the University of California, Davis. He received a B.S. in wildlife biology (1978) and an M.S. in plant pathology (1981) from the University of Massachusetts, Amherst. He received a Ph.D. in botany and plant pathology from Colorado State University in 1985. He took a postdoctoral position in 1985 in the Department of Plant Pathology at the University of Wisconsin, Madison, and worked on common bacterial blight of bean in the laboratory of D. J. Hagedorn. In 1987, he joined the staff of the Department of Plant Pathology at U.W. Madison as an assistant scientist and worked on bean golden mosaic geminivirus in the laboratory of D. P. Maxwell. He assumed a faculty position in the Plant Pathology Department at U.C. Davis in 1990. His research interests include the ecology, detection, and management of seedborne pathogens, and the genetics, taxonomy, and management of whitefly-transmitted geminiviruses. His teaching responsibilities include introductory plant pathology and portions of plant bacteriology and plant virology.



**Elizabeth L. Little**

Dr. Little is a postdoctoral research scientist in the Department of Plant Pathology at the University of California, Davis. She received her B.S. degree in plant pathology from Cornell University before working for several years in the horticultural industry in New York State. Under the guidance of R. Gilbertson, she received her Ph.D. in plant pathology from U.C. Davis in 1995. Her thesis was on the epidemiology of celery bacterial blight in California and molecular characterization of *Pseudomonas syringae* pv. *apii* and related pathovars. In 1995, she joined the lab of B. Kirkpatrick at U.C. Davis as a postdoctoral researcher. Her current research focuses on epidemiological aspects of bacterial canker of stone fruits and characterization of *P. s. pv. syringae* strains in California.

phic DNA (RAPD) analysis of *P. s. pv. apii* strains and other pathovars of *P. syringae* was used to identify a unique 800-bp fragment of *P. s. pv. apii* DNA. The sequence of this fragment was used to design PCR primers that directed the amplification of a 700-bp fragment from total genomic DNA of all *P. s. pv. apii* strains tested, as well as from extracts of boiled colonies of *P. s. pv. apii*. The bacterium was consistently detected from DNA extracts prepared from celery leaf lesions using the PCR and the *P. s. pv. apii* primers. The PCR test can provide a positive diagnosis of blight in less than 1 day, compared to the weeks or longer that are required for a positive diagnosis based on isolations and pathogenicity tests. Detection of *P. s. pv. apii* from contaminated celery seed by the PCR method was also evaluated, but the method developed was not reliable and failed to provide increased sensitivity compared to the seed wash assay. The use of cultural practices, hot water seed treatment, certification of celery seed, and rapid diagnosis will be important tools in future management of bacterial blight.

**Fusarium yellows.** Celery production has declined in some northern states (New York, Ohio, and Wisconsin) because of the inroads made by *Fusarium* yellows, but Michigan is still ranked third nationally, behind California and Florida (11). Celery

yields in Michigan remained relatively constant over a 5-year period from 1990 to 1995 at 47,000 to 51,500 kg/ha (420 to 460 CWT per acre), in spite of increasing incidence of *Fusarium* yellows. Most Michigan growers have abandoned monoculture of celery and have entered into a rotation program with onions, another crop commonly grown in the Midwest. This rotation, along with the use of resistant cultivars, should enable growers to produce celery competitively in *Fusarium*-infested soils for the foreseeable future. Celery growers in other parts of the United States may profit from this experience.

#### Literature Cited

1. Awuah, R. T., and Lorbeer, J. W. 1991. Methyl bromide and steam treatment of an organic soil for control of *Fusarium* yellows of celery. *Plant Dis.* 75:123-125.
2. Awuah, R. T., Lorbeer, J. W., and Ellerbrock, L. A. 1986. Occurrence of *Fusarium* yellows of celery caused by *Fusarium oxysporum* f. sp. *apii* race 2 in New York and its control. *Plant Dis.* 70:1154-1158.
3. Berger, R. D. 1970. Epiphytology of celery late blight. *Proc. Fla. State Hortic. Soc.* 83:208-212.
4. Berger, R. D. 1973. Early blight of celery: Analysis of disease spread in Florida. *Phytopathology* 63:1161-1165.
5. Berger, R. D. 1973. Infection rates of *Cercospora apii* in mixed populations of susceptible and tolerant celery. *Phytopathology* 63:535-537.
6. Cerkauskas, R. F., and McDonald, M. R. 1989. Race 2 of *Fusarium oxysporum* f. sp. *apii* new to Ontario. *Plant Dis.* 73:859.
7. Coons, G. H. 1915. The Michigan plant disease survey for 1914. Celery Diseases. *Mich. Acad. Sci. Rep.* 17:126-128.
8. Elmer, W. H., and Lacy, M. L. 1982. The reappearance of *Fusarium* yellows of celery in Michigan. (Abstr.) *Phytopathology* 72:1135.
9. Elmer, W. H., and Lacy, M. L. 1987. Effects of crop residues and colonization of plant tissues on propagule survival and soil populations of *Fusarium oxysporum* f. sp. *apii* race 2. *Phytopathology* 77:381-387.
10. Elmer, W. H., Lacy, M. L., and Honma, S. 1986. Evaluations of celery germ plasm for resistance to *Fusarium oxysporum* f. sp. *apii* race 2 in Michigan. *Plant Dis.* 70:416-419.
11. Fedewa, D. J. 1994. Michigan Agricultural Statistics, 1994. Mich. Dep. Agric., Lansing, MI.
12. Fitt, B. D. L., McCartney, H. A., Walklate, P. J. 1989. The role of rain in dispersal of pathogen inoculum. *Annu. Rev. Phytopathol.* 27:241-270.
13. Fournet, J. 1969. Propriétés et rôle du cirrhe du *Septoria nodorum* Berk. *Ann. Phytopathol.* 1:87-94.
14. Fry, W. E. 1978. Quantification of general resistance and fungicide effects for integrated control of potato late blight. *Phytopathology* 68:1650-1655.
15. Gaye, M. M., Ormrod, D. J., Seywerd, F. M., and Odermatt, W. J. 1991. Occurrence of *Fusarium* yellows of celery in southwestern British Columbia and evaluation of cultivars for disease tolerance. *Can. J. Plant Pathol.* 13:88-92.
16. Good, J. M. 1987. The effect of flooding on nematode populations. Pages 35-39 in: *Agri-*

- cultural Flooding of Organic Soils. G. H. Snyder, ed. Univ. Fla. Tech. Bull. 870.
17. Gough, F. J., and Lee, T. S. 1985. Moisture effects on the discharge and survival of conidia of *Septoria tritici*. *Phytopathology* 75:180-182.
  18. Greathead, A. S. 1993. Celery Fusarium yellows - control through the use of resistant varieties. Pages 47-62 in: *Calif. Res. Advisory Bd. 1992-93 Annu. Rep.*
  19. Hart, L. P., and Endo, R. M. 1978. The reappearance of Fusarium yellows of celery in California. *Plant Dis. Rep.* 62:138-142.
  20. Heath-Pagliuso, S., Pullman, J., and Rappaport, L. 1988. Somaclonal variation in celery: Screening for resistance to *Fusarium oxysporum* f. sp. *apii*. *Theor. Appl. Genet.* 75:446-451.
  21. Heath-Pagliuso, S., Pullman, J., and Rappaport, L. 1989. 'UC-T3 somaclone': celery germplasm resistant to *Fusarium oxysporum* f. sp. *apii* race 2. *HortScience* 24:711-712.
  22. Jagger, I. C. 1921. Bacterial leafspot of celery. *J. Agric. Res.* 21:185-188.
  23. Koike, S. T., and Bishop, A. L. 1990. Bacterial leaf spot of celery caused by *Pseudomonas syringae* pv. *apii* in California. (Abstr.) *Phytopathology* 80:890.
  24. Koike, S. T., Little, E. L., Bishop, A. L., and Gilbertson, R. L. 1994. New celery disease appears in California. *Calif. Agric.* 48:32-34.
  25. Lacy, M. L. 1973. Control of *Septoria* leafspot of celery with systemic and nonsystemic fungicides. *Plant Dis. Rep.* 57:425-428.
  26. Lacy, M. L. 1994. Influence of wetness periods on infection of celery by *Septoria apiicola* and on timing of sprays for control. *Plant Dis.* 78:975-979.
  27. Lacy, M. L., and Cortright, B. D. 1992. Chemical control of *Septoria* late blight of celery, 1991. *Fungic. Nematicide Tests* 47:88.
  28. Lacy, M. L., Grumet, R., and Cortright, B. D. 1995. Summary of progress report on 1994 celery disease research. Page 82 in: *Proc. Great Lakes Veg. Growers Conv.*, Jan. 17-19, 1995.
  29. Lacy, M. L., Grumet, R., Toth, K. F., Krebs, S. L., and Cortright, B. D. 1996. MSU-SHK5: A somaclonally-derived Fusarium yellows resistant celery line. *HortScience* 31:289-290.
  30. Lelliot, R. A., Billing, E., and Hayward, A. C. 1966. A determinative scheme for the fluorescent plant pathogenic pseudomonads. *J. Appl. Bacteriol.* 29:470-489.
  31. Lin, K. H. 1939. The number of spores in a pycnidium of *Septoria apii*. *Phytopathology* 29:646-647.
  32. Little, E. L., Koike, S. T., and Gilbertson, R. L. 1991. Celery bacterial blight: A new and increasingly important disease in California. *Proc. Int. Working Grp. Pseudomonas syringae* pathovars, 4th, Florence, Italy.
  33. Little, E. L., Koike, S. T., and Gilbertson, R. L. 1992. Association of *Pseudomonas syringae* pv. *apii* with celery seed. (Abstr.) *Phytopathology* 82:1072.
  34. Little, E. L., Koike, S. T., and Gilbertson, R. L. 1993. Molecular approaches for the detection of *Pseudomonas syringae* pv. *apii*. (Abstr.) Page 30 in: *Proc. Int. Congr. Plant Pathol.*, 6th, Montreal, Canada.
  35. Martyn, R. D. 1987. Fusarium yellows of celery in Texas. *Plant Dis.* 71:651.
  36. Mohan, S. K., and Schaad, N. W. 1987. An improved agar plating assay for detecting *Pseudomonas syringae* pv. *phaseolicola* in contaminated bean seed. *Phytopathology* 77:1390-1395.
  37. Orton, T. J., Hulbert, S. H., Durgan, M. E., and Quiros, C. F. 1984. UC-1, Fusarium yellows-resistant celery breeding line. *HortScience* 19:594.
  38. Ryder, E. J. 1979. Celery. Pages 95-126 in: *Leafy Salad Vegetables*. AVI Publishing, Westport, CT.
  39. Schneider, R. W., and Norelli, J. L. 1981. A new race of *Fusarium oxysporum* f. sp. *apii*. (Abstr.) *Phytopathology* 71:108.
  40. Sherf, A. F., and MacNab, A. A. 1986. Celery. Pages 157-201 in: *Vegetable Diseases and their Control*. John Wiley & Sons, New York.
  41. Sheridan, J. E. 1968. Conditions for infection of celery by *Septoria apiicola*. *Plant Dis. Rep.* 52:142-145.
  42. Strandberg, J. O., and White, J. M. 1978. *Cercospora apii* damage on celery — effects of plant spacing and growth on raised beds. *Phytopathology* 68:223-226.
  43. Thayer, P. L., and Wehlburg, C. 1965. *Pseudomonas cichorii*, the cause of bacterial blight of celery in the Everglades. *Phytopathology* 55:554-557.
  44. Toth, K. F. 1989. Biology and control of *Fusarium oxysporum* f. sp. *apii* race 2. Ph.D. thesis. Michigan State University, East Lansing. p. 154.
  45. Toth, K. F., and Lacy, M. L. 1991. Increasing resistance in celery to *Fusarium oxysporum* f. sp. *apii* race 2 with somaclonal variation. *Plant Dis.* 75:1034-1037.
  46. Toth, K. F., and Lacy, M. L. 1992. Field evaluation of celery germ plasm for resistance to *Fusarium oxysporum* f. sp. *apii* race 2. *Mich. State Univ. Agric. Exp. Stn. Res. Rep.* 523.