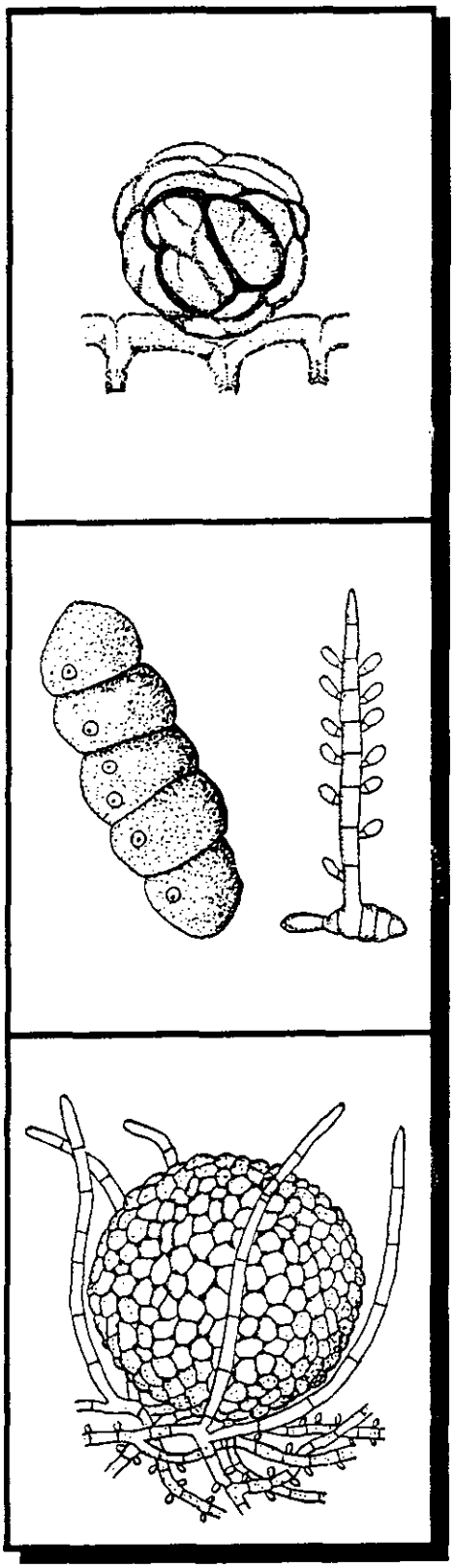

March, 1992
Volume XIII, Number 1

PLANT DIAGNOSTICS QUARTERLY

Features

Plant Disease Diagnostic Sheets

On the cover: A close look at one of the common, but ignored, fungi:
Top = young cleistothecium of *Meliola*, a sooty mold, on a leaf surface.
Middle = mature ascospore (left) and germinating ascospore (right) of *Meliola*.
Bottom = mature cleistocarp of *Meliola*

Plant Diagnostician's Quarterly (PDQ) is a nonprofit publication which serves plant pathologists in extension, regulatory and industrial clinical laboratories, private consultants, and other interested persons. PDQ is published four times a year. Yearly subscription fees are:

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Volume XIII, Number 1
March, 1992

PLANT DIAGNOSTICS QUARTERLY

FROM THE EDITOR

ATTENTION !!

Well the time I'm sure many of you have been waiting for has come: Your Faithful Editor has decided to step down from the helm of *PDQ* and hand the tiller over to someone else. This is my fifth year as editor, and I think that is probably long enough. I will be happy to remain until the end of the year, but would also consider an earlier date for retirement.

If you are interested in filling my shoes (size 40 metric), please send me a letter stating your interest in *PDQ* and a number of suggested feature articles. I would also be interested in how you would go about soliciting articles, and your editorial philosophy.

The strength of this publication has always been due to the contributions and volunteer efforts of its readers. If no one steps forward to take over the position of editor, ***PDQ* will cease to exist**. Don't assume that others will take the necessary action - most likely they will not.

I have certainly enjoyed my tenure as editor and encourage any of you who would like to be in the position of writing this column to send me your ideas.

Until I am replaced, I remain

Your Faithful Editor,

Melodie Putnam

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REGIONAL REPORTS

Northwest Region
Colette Beaupré

Laura Pottorff in Colorado reports the sudden temperature drop on October 28, 1991 has caused significant damage to many landscape plants. The full extent of injury may not be realized until spring. Scots, Ponderosa, and Austrian pines were the first to show damage, with the most recent year's needles dehydrated. The most severe browning is evident on south and west exposures. Trees stressed by recent transplanting, pruning, excess water, and fertilizer prior to the October freeze were injured the most. In most cases, however, the buds of affected evergreens appear healthy.

Bud-dehydration and branch die-back in Callery pears, Sunburst honeylocust, privet, Japanese barberry, cottoneaster, and smooth sumac was also observed.

Other notable diseases diagnosed recently include Coniothyrium canker on greenhouse roses ('Royalty'), tomato spotted wilt virus on *Phalaenopsis* orchids, Pythium and Rhizoctonia crown rots on ivy and zonal geraniums, *Alternaria* and *Fusarium* on carnations, and Rhizoctonia crown rot on basil. Interestingly, Laura is also beginning to pick up Polymixa-like fungi on bentgrass roots. Samples have been sent to Dr. Adrianna Hewing (USDA-ARS, Urbana, Illinois) for confirmation.

Stacey Fisher notes that the winter in Oregon's Willamette Valley has been extremely mild with only a couple of freezes. Crocuses have been up and flowering; and the flowering cherries have been blooming since the second week of February. A spring freeze would make diagnoses easy. Several plant samples have arrived that had aphids actively moving about. This may be a good virus year, a warm welcome to our new virologist at OSU.

Even though temperatures have been so mild, woody ornamentals that have been nipped by cold temperatures have come into the Plant Clinic (especially young trees that were over-fertilized and watered late in the fall, preventing hardening-off). *Fusarium* patch and yellow patch have been active on turf; and Berckman's blight occurred on arborvitae. *Botrytis* is moving on greenhouse crops, landscape ornamentals, and cabbage for seed. The weather has been perfect for scald, and it has occurred on orchard grass and barley. *Phoma* has been causing crown rot of sugar beet. An aerial *Rhizoctonia* almost melted a crop of greenhouse-grown Corsican mint. A weeping fig with foliar ringspots and necrotic arcs had rod-shaped virus particles (450 and 550 nm long) in the leaf tissue.

Many perennial rye grass seed growers have been noticing poor regrowth after harvest. In the past, this may have been attributed to field burning die-out; but with a decrease in burning, another factor must be involved. No pathogen has been associated at this time of the year with the die-out.

Western Washington has also been experiencing a mild winter. The weather may partially account for the record number of off-season sample submissions to the WSU Puyallup Plant Clinic. Carrie R. Foss has received several unusual disease problems, including brown rot on pear, black leaf spot on *Helleborus* sp., and *Sirococcus* blight on mountain hemlock. Rust teliospore infection was determined to be the cause of a necrotic leaf spot on coral bells. Commercial Christmas tree growers have been concerned about the physiological disease known as current-season needle necrosis affecting their true firs. Yellow patch caused by *Rhizoctonia cerealis* has been the most common problem observed recently on turf.

Ellen Bentley reports from Eastern Washington that the WSU clinic in Prosser processed 758 samples in 1991. Thirty-five percent were from commercial agriculture and 58% from consumer horticulture. Seven percent were from research or regulatory agencies. Thirty-seven percent were diagnosed as a disease, 13% as other physiological or environmental problems, and 37% as insect-caused. Thirteen percent were inconclusively diagnosed due to poor sample quality or insufficient information.

1992 has begun without winter, as temperatures and moisture have been higher than normal. Low snow pack may limit late summer irrigation availability. Pruning is well underway, as fruit buds are already swelling. Winter wheat is green and last fall's dry areas have been reseeded. Field preparation have begun for seasonal crops. The potential for a late season cold snap has growers concerned.

Clinic samples have consisted of problems observed during these "spring" activities. Many have carried over from last year including cold injury, drought stress, bacterial canker in stone fruits and late blight in stored potatoes.

The second consecutive year of late blight has been a concern due to the presence of metalaxyl-resistant strains of *Phytophthora infestans* in British Columbia and western Washington. Samples from infected circles located in the Columbia Basin were submitted to Ken Diehl (USDA, Beltsville, MD) for evaluation. Several isolates had moderate-to-high metalaxyl resistance, raising serious concern for this coming season. Historically, late blight has occurred in the region following mild winters (which fail to freeze cull piles and unharvested tubers).

Martin Draper has an update from North Dakota: The North Dakota summer season of 1991 was marked by two rather new concepts; rain (at last) and summer heat (about one month early). As a result, crop diseases were plentiful and about three weeks ahead of a normal summer (whatever that is). Tan spot on wheat was very prevalent; net blotch on barley was the worst observed in at least ten years (as far as incidence and distribution). The year progressed with additional leaf diseases causing problems on crops and ornamentals alike. Bacterial blights were so prominent on wheat and barley that producers were concerned about control measures. Both black chaff and basal glume rot were noted on samples from various parts of the state. By mid-summer the weather dried out and left us in a near drought. Row crops were running seriously short of moisture by September. Later in the summer powdery mildews were so heavy in small grains that some fields showed up to 40% severity on flag leaves.

Fire blight was very severe on apples, crab apples, and mountain ash trees. Other diseases that are typically curiosities in ND, such as elm black spot were severe in locations.

Turf diseases continued to be a problem on stressed lawns across the state. Necrotic ring spot has apparently become the most widespread disease of concern among homeowners and turf professionals in the state.

Two new diseases were reported from North Dakota in 1991. For the last several years we have been expecting the arrival of the virulent strain of blackleg of canola from adjacent areas of Manitoba. This year it was discovered in each of the fields surveyed at levels as high as 60% incidence. Canola has become a fairly well established crop in the northeast corner of the state and the occurrence of this disease will certainly impact canola as an alternative crop. Another alternative crop is lupine. Lupine is not nearly as far along as a real cropping alternative. Nonetheless, anthracnose was found on lupines in the east central part of the state.

Overall, it was a busy year. We have seen an increase in sample number over each of the last three years. Just under 1,000 samples were sent to the lab in 1991. About 60% of those samples were plant diseases/disorders and about half were submitted through a County Agent.

Seed health testing has increased, both within dry beans and potatoes, and with new crops. A late blight assay for celery and an Ascochyta blight assay for chickpeas has been added.

Wyoming was no exception regarding mild winter weather. In the warmer parts of the state lilac buds were breaking in mid-February. Winter injury to trees and shrubs is currently low; however there is the potential for extensive winter damage in the coming weeks.

The most common specimen received this winter has been house plants with cultural problems. Willow, poplar, and Colorado Blue Spruce samples with Cytospora canker occasionally came in. *Fusarium* dry rot in stored potatoes due to bruising has been a problem for some growers.

The Wyoming Master Gardener volunteer training program occurs in March and early April. Our plant pathology workshop will be reduced from eight to six hours. Emphasis will be on Wyoming's most common garden diseases (currently about 70% of clinic samples are from home gardeners). This will hopefully result in more time for the clinic to address crop problems. (Collette M-S Beaupré)

Southwest Region

Steven Koike

Drought-stricken California welcomed some much needed rain this winter. Due to these rains, planting of annual vegetable crops has been slightly delayed in some areas. Downy mildew (*Peronospora effusa*) of spinach, which damaged last winter's crop, has occurred only sporadically in most areas. Presumably this is due to the combination of preventive fungicide programs and new race 4-resistant cultivars. Incidence of lettuce infectious yellows has been slight in the desert lettuce growing

regions. Researchers are not sure of the reasons behind this reduction. It may have to do with the decreased transmission ability of the new whitefly biotype and the reduction of reservoir hosts.

Oklahoma experienced very cold temperatures earlier in the winter (November), causing noticeable damage to trees, including significant damage to peach trees. Some miscellaneous detections include pine with pine wood nematodes, *Colletotrichum* causing dieback on *Pyracantha*, Botryosphaeria canker on rose, Dothistroma needle blight on pine, and Mycosphaerella leaf spot on strawberry.

Disease detection reports from Arizona include Texas root rot on *Brachychiton* trees and *Cassia* species, *Pratylenchus* sp. on *Sophora segundiflora* and *Phyllosticta* on *Opuntia* sp.

Central Region
Karen Rane

Clinics throughout the region are still receiving samples of evergreens, primarily spruce and Scots pine, that were damaged by the early November freeze. Other conifer problems reported from the region include Dothistroma and brown spot needle blights (Iowa), Diplodia tip blight (Kansas) and pine wilt (Illinois, Kansas).

The November freeze also adversely affected the wheat crop in many states. Cold injury followed by Rhizoctonia blight has caused significant damage on wheat in Illinois and Indiana. In Missouri, heaving problems have developed due to the freeze. Wheat viruses found include soilborne mosaic (Kansas, Missouri), spindle streak (Kansas), and wheat streak in volunteer wheat (Missouri). Leaf rust has overwintered in south central Kansas.

In Indiana, several cases of equine leukoencephalomalacia have been reported. This disease is caused by ingestion of corn feed containing fumonisin, a mycotoxin produced by some species of *Fusarium*, including *Fusarium moniliforme*. Conditions were favorable for Fusarium ear rot development last fall, resulting in contaminated grain.

Problems of interest on ornamentals this winter include several samples of palms with leaf spots in Minnesota, and root knot nematode on weigela in Iowa.

Northeast Region
Anne Bird Sindermann

Many of the 1991 plant diseases in the Northeast were summarized by Margery Daughtrey in the Minutes of the NED-APS Extension Committee, including damage observed in the wake of Hurricane Bob. Wind and salt injury was widespread. Also, Margery reports that an outbreak of bacterial blight on geraniums may have been associated with hurricane wind-driven rain carrying inoculum from perennial *Geranium* spp. to geraniums in an uncovered (plastic) greenhouse. Another occurrence believed to be associated with the hurricane was bacterial infection that caused fruit collapse in New Jersey pumpkins.

Mike Likins detected tissue proliferation in Virginia grown *Rhododendron catawbiense* 'Album' that was at first inspection very much like crown gall. However, he noted some inconsistencies and the diagnostic danger flag went up. One of the differences noted differentiated vascular tissue, possibly explaining the otherwise healthy appearance of the plants. Mike is now involved in determining what causes this condition. It has been described as a gall forming phenomenon and tissue proliferation. Workshops have been held in Washington State (where the descriptive name "tissue proliferation" was coined) and recently in New England.

The abundant callus-like tissue is often accompanied by adventitious buds and/or shoots produced near the crown. The condition has been observed in some vegetatively propagated and tissue culture derived rhododendron cultivars. It is suspected that the confusion between this condition and crown gall, caused by the bacterium *Agrobacterium tumefaciens*, has resulted in the rejection of rhododendrons by plant regulatory officials. Attempts to isolate the crown gall pathogen from tissue proliferation samples have been negative, as have bioassay and DNA probes.

The present conclusions about tissue proliferation are that it is a non-pathogenic condition; plant to plant spread has not been observed under field conditions; and that plants recover after two or three growing seasons. That is, if the grower hasn't destroyed them first.

Southeast Region
Jackie Mullen

Botrytis was active in landscape plantings in many southeastern states, due to the unseasonably mild winter. Also most states reported Rhizoctonia and Pythium blights on landscape turfgrasses and golf course bentgrass, respectively. Another commonly reported disease this winter quarter was Phytophthora crown and root rot on both landscape and nursery stock woody ornamentals. The northern-most southeastern states reported some cold damage on woody ornamentals due to the freezing temperatures of early November.

In Tennessee, Beth Long reported the occurrence of winter injury and Botryosphaeria canker on woody ornamentals, needlecast diseases on spruce, Phytophthora root rot on woody ornamental nursery stock, Pythium and Rhizoctonia root rot on greenhouse bedding plants, a low incidence of tomato spotted wilt virus in bedding and greenhouse plants, and cool season Pythium and Rhizoctonia on bentgrass turf (golf greens and sod). In addition, she reported field crops with Sclerotinia stem and crown rot on alfalfa and winter injury on wheat, oats, and rye.

In North Carolina, Tom Creswell listed a variety of ornamental problems, including Botrytis (*B. cinerea* or *B. tulipae*) blight on spring bulbs, floral crops, bedding plant seedlings, and pansy; Pythium root rot on floral crops and Pythium crown/root rot on bentgrass; *Rhizoctonia* problems on bedding plant seedlings and bentgrass (*Rhizoctonia cerealis*; cool-weather *Rhizoctonia*); black root rot (*Thielaviopsis*) on pansy; pink snow mold (*Fusarium nivale*) on bentgrass; Phytophthora root rot (*P. cinnamomi*) on pansy and several woody ornamentals including false cypress (*Chamaecyparis*), false arborvitae (*Thujopsis*), and wax myrtle (*Myrica*); and root knot nematode (*Meloidogyne incognita*) on schefflera. In addition to these ornamental/turf problems, Tom reported the occurrence of barley yellow dwarf virus on rye, and downy mildew (*Peronospora parasitica*), *Sclerotinia sclerotiorum*, and black leaf spot (*Alternaria*) on collards. Also included in his list was a problem called tulip leaf withering caused by toxins produced by *Fusarium* and *Trichoderma* that colonize tulip roots. These toxins move into leaves and leaf tips and cause the leaves to shrivel.

Brian Eshenaur in Kentucky commented on the hard freeze in early November which had been preceded by mild weather. Some conifers were apparently not hardened-off and were damaged by the sudden low temperatures. Needle tip necrosis on the most recent growth and an over-all bronzing of the foliage were noted on spruce, pine, and *Taxus*. With greenhouse crops, Brian reported the unusual occurrence of Cylindrocladium root rot on spathiphyllum.

James Blake in South Carolina reported diseases of ornamentals resulting from mild winter weather. His list included Phytophthora root rot on azalea, boxwood, ivy, rhododendron, holly, and Leyland cypress; downy mildew on rose; Lophodermium (*Ploioderma*) needle cast on slash pine; algal leaf spot on camellia and magnolia; Cercospora leaf spot on *Ligustrum*, pittosporium, rhododendron and turnip; tar spot on Chinese hollies; Phomopsis dieback on dogwood, camellia, and azalea; and Sclerotinia stem root rot on collards. Also, James reported seeing crown gall on rose and apricot, and mushroom root rot on azalea.

Clayton Hollier in Louisiana reported a particularly high incidence of *Botrytis* on pansies and Rhizoctonia brown patch on turf grasses, especially St. Augustine.

In Mississippi, M. V. Patel saw greenhouse tomatoes with Cladosporium leaf mold, Botrytis gray mold, Pythium root rot, and Fusarium crown rot. On golf course bentgrass, M. V. reported Rhizoctonia brown patch and Pythium blight.

Florida, as usual, reported a large number of disease problems. Some of the more interesting and serious problems are mentioned here. Tomato spotted wilt virus, gemini viruses, Phytophthora spp. on woody ornamentals, and Benlate-related diagnoses required considerable time and attention. Some of the unusual problems seen include *Cattleya* sp. with odontoglossum ringspot virus and cymbidium mosaic virus; cucumber with *Alternaria cucumerina* leaf blight and *Corynespora cassiicola* leaf spot; mahogany (*Swietenia mahagoni*) with *Pseudocercospora subsessillis* leaf spot; St. Augustinegrass with *Sclerotinia homeocarpa* dollar spot and *Gaeumannomyces* sp. decline; tomato with *Erwinia carotovora* pv.

carotovora hollow stem, dieback and blight of sweet basil (*Ocimum basillicum*) due to *Sclerotinia sclerotiorum* ; nephthytis (*Syngonium podophyllum*) with leaf spot (*Myrothecium roridum*), soft rot (*Erwinia chrysanthemi*), and leaf spot (*Pseudomonas cichorii)* ; and petunia with *Rhizoctonia solani*, *Pythium*, and *Phytophthora parasitica* root rots, and blight due to *Sclerotinia sclerotiorum* .

As with the rest of the South, Alabama also experienced a mild winter. Included in our list of diseases are powdery mildew (*Sphaerotheca*) on rose, algal leaf spot (*Cephaleuros*) on southern magnolia, Phytophthora root rot on juniper, Botrytis blight on pansy, slime mold on centipede grass, Cercospora leaf spot on azalea, Colletotrichum leaf spot on dwarf nandina, Heterosporium leaf spot on iris, Pythium root rot on pansy, Pythium blight on bentgrass and ryegrass, and Rhizoctonia brown patch on centipedegrass. With the greenhouse situation, we saw *Botrytis* on chrysanthemum and Phytophthora, Pythium, and Rhizoctonia root rots on spathiphyllum. Cercospora leaf spot was common on turnips as it usually is at this time of year. In addition, we recently received some Irish potatoes purchased by an Alabama grower from a Wisconsin seed producer. The problem concerns the Fusarium dry rot and bruising which was present when the seed arrived in Alabama but not when it left Wisconsin (J. Mullen).

MEMORANDUM

TO: *PDQ* Readers
FROM: Jackie Mullen, The Plant Diagnostic Lab, Auburn University
RE: Hymexazole

I am trying to find a supplier for hymexazole (tachigaren). We use it in a selective media for *Phytophthora* spp. as hymexazole will selectively inhibit many *Pythium* spp. I have written to the Sankyo Co., Ltd. No. 7-12, Genza 2-Chome, Chuo-Ku, Tokyo 104, Japan (as listed in the Farm Chemicals Handbook) on two separate occasions with no results. If you know how to contact the supplier (or a local U.S. supplier) please call me at (205) 844-5508 or FAX me at (205) 844-1947; my Internet address is JMULLEN@ACENET.AUBURN.EDU.

Thanks.

DIFFUSION

A rhabdovirus inducing vein yellowing in croton. Croton plants (*Codiaeum variegatum* cv. Fred Sander) in Italy were found to have virus-like symptoms: leaf malformation, dwarfing, and vein yellowing. A. Bertaccini and M. G. Bellardi (Ist. Patologia vegetale, Univ. Bologna, Italy), found virus particles in sap of naturally infected plants and those inoculated with sap from infected plants. The authors were able to transmit the virus mechanically to *Nicotiana glutinosa* and *Chenopodium amaranticolor*; the virus was transmitted via leaf grafts to healthy crotons. Virus particles were bacilliform and 70-80 x 180-240 nm in size. The name croton vein yellowing virus is proposed for the virus. Additional characterization studies are underway. Plant Pathology 1992, **41**:79-82.

Interaction of management factors on dollar spot disease severity in tall fescue turf. A. D. Brede (formerly of Dept. Horticulture and L/A, Oklahoma State Univ., Stillwater) evaluated the interaction of two tall fescue cultivars, seeding and fertilizer rates, and cutting height on severity of disease caused by *Lanzia* and *Moellerodiscus* spp. (No information is provided on fungal identification, or even which fungi were actually present.) Plots were established outdoors using fescue cultivars Kentucky-31 and Mustang; seeding rates were 5, 29, and 78 g seed/m^2; fertilizer was applied at 4.9 and 24.4 g N/m^2/year. Dollar spot severity was evaluated visually during natural epiphytotics over the course of two years. As with other turf diseases, management factors did influence disease severity. Specifically, severity increased with higher cutting height (57 vs 19 mm) and higher seeding rates. 'Mustang' was more susceptible of the two cultivars. HortScience 191, **26**:1391-92.

Comparison of trunk injected and soil applied macronutrients. Use of trunk injection to deliver various compounds into the vascular system of trees has increased in recent years. Trunk injection is usually used to deliver pesticides and micronutrients. E. T. Smiley *et al.* (Bartlett Tree Res. Lab., Charlotte, NC) recently looked at soil application and trunk injection for effectiveness in correcting macronutrient deficiency in trees with limited root space. Twenty five chlorotic willow oaks (*Quercus phellos*) in parking lot islands were treated with either 28-9-9 (N-P-K) slow release fertilizer injected eight inches below the soil surface, or with a 0.4-0.6-0.6 soluble fertilizer injected into the trees twice over a nine month period. Leaves of treated trees were analyzed by Clemson University (South Carolina) for total N, available P_2O_5, soluble K_2O, Cu, Fe, Mn, and Zn. The authors found that "one soil application of a complete slow release fertilizer increased the foliar nitrogen level and improved color more than two applications of trunk injected macronutrients." (There was basically no effect on either phosphorus or potassium levels for either treatment). It was estimated that increasing N levels in deficient trees from 1.4% to 2.0% would require 2,500 standard 6 ml capsules of 0.4% N. J. Arboriculture 1991, **17**:322-324.

5th Annual Workshop on Rapid Diagnostic Assays: Survey Results

Compiled by
Sally A. Miller, Workshop Chair

The fifth consecutive "Rapid Diagnostic Assays for Plant Pathogens Workshop" sponsored by the Diagnostics Committee of American Phytopathological Society was held at the APS meeting in St. Louis. As in previous years the workshop, which required pre-registration, was filled to capacity. At this event, a survey was developed and distributed to participants in the workshop. The purpose of the survey was to 1) determine the segments of APS served by the Workshop, 2) judge the value of the Workshop in its current format to the participants, and 3) ask for suggestions on improvements for future Workshops.

The survey was completed by 40 of the 70 registered participants. The results show that the Workshop was attended by a wide range of professionals working in academic, industrial and state institutions (Figure 1). Eighty percent of the participants work in the United States. About one-third (35%) of the participants were involved in clinical diagnostics more than 50% of the time, one-third (37.5%) were involved but less than 50% of the time, and nearly one-third (27.5%) were not involved in clinical diagnostics.

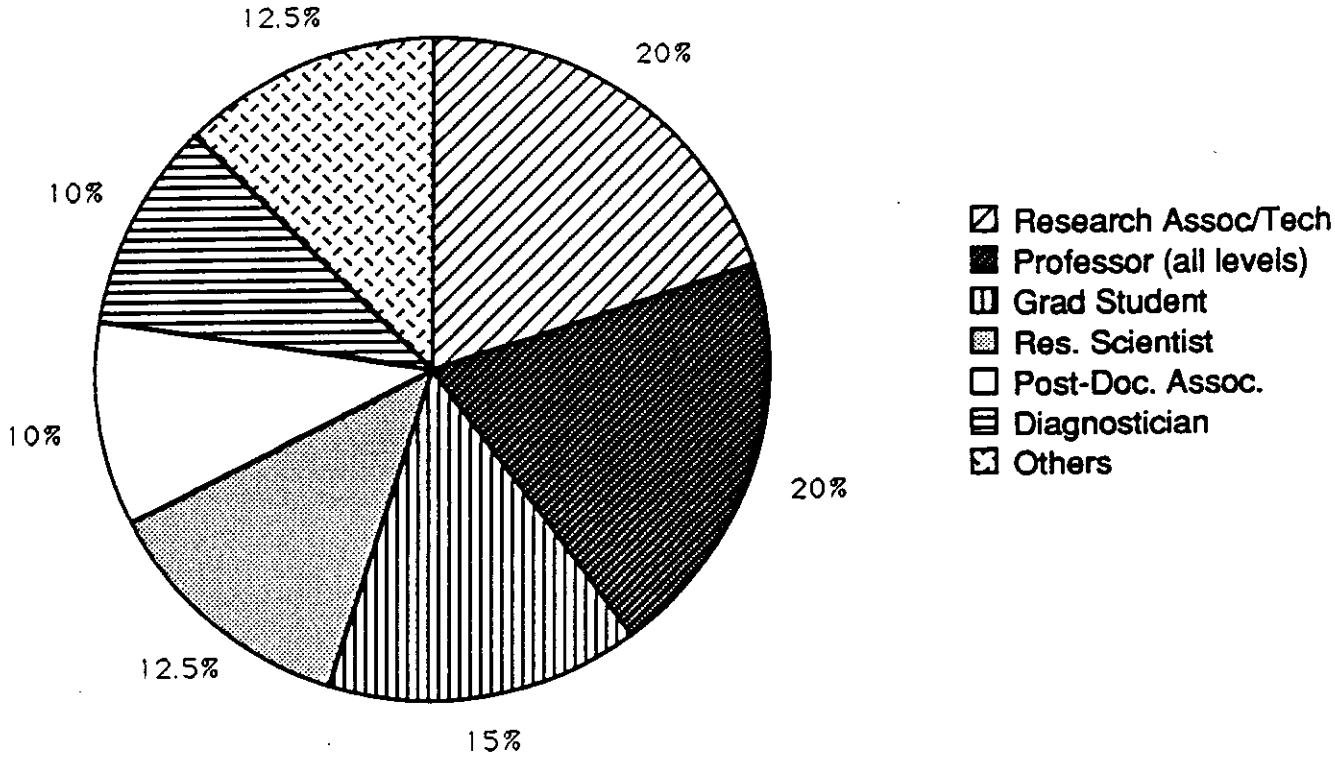
The majority (77.5%) of the participants were interested in learning about currently available products as well as new technologies that might become available in the future. Nearly all of the participants ranked each of the sessions attended as meeting his or her expectations. Ninety-five percent of the participants felt that the workshop should be offered again at the next APS meeting (5% did not respond to the question).

There were a number of suggestions made for improving the workshop next year. One consistent problem over the years has been the noise level - with six or seven presentations going on at once in a single room, the volume goes way up. We will try next time to locate and use dividers between the workstations to reduce the level of noise. Another problem, was with "walk-ons". Quite a few people who had not registered stopped by to hear some of the presentations. This resulted in some very crowded sessions. We'll try to find a way to handle this better in Portland. There were also suggestions to include more handouts, and to provide a session that addresses improvements in "classical" techniques and "tricks of the trade" for ELISA and other methodologies.

The subcommittee responsible for the Workshop (S. Miller (chair), Larry Brown, Steve Nameth, Melodie Putnam and Chet Sutula) is open to additional comments or suggestions.

The proposed line-up of participants for the Workshop in Portland follows on the next page.

Positions Held by Workshop Participants



Institution

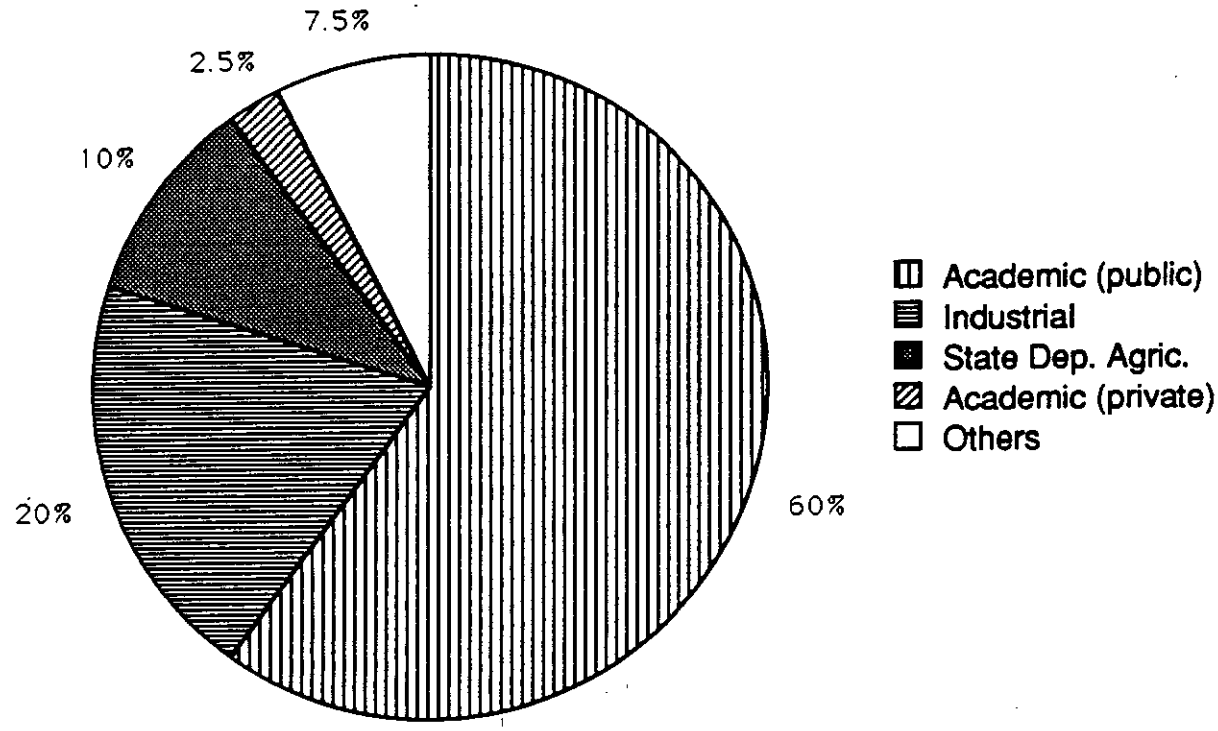


Figure 1. Segments of APS served by the Workshop: Rapid Diagnostic Assays for Plant Pathogens

WORKSHOP - Speakers for Rapid Diagnostic Assays for Plant Pathogens

APS - Portland, OR (1992)

Speaker #1: DuPont's rapid membrane test for plant disease (Botrytis) and pesticide residue (Lannate). Craig Lamison. Melissa Joerger and Laure Kenyon, E. I. DuPont, Wilmington, DE 1980

Speaker #2: Virus ELISA. Chet Sutula, AGDIA Inc., Elkhart, IN 46514

Speaker #3: Use of rapid on-site assay for detection of Xanthomonas campestris pv. pelargonii on geranium. Tony R. Joaquim, Agri-Diagnostics Assoc., Cinnaminson, NJ 08077

Speaker #4: Principles of the PIN ELISA system for plant diagnostics. Franz Fux, BIOREBA Inc., Chapel Hill, NC 27515

Speaker #5: An ELISA based diagnostic kit to detect mature ascospores of Venturia inaequalis (apple scab). L. P. Berkett, A. K. Gotlieb, and J. A. Bergdahl, Dept. Plant and Soil Science, Univ. of Vermont, Burlington, VT 05405.

Speaker #6: Nucleic acid methods for detection of plant pathogens. R. L. Gilbertson and E. J. Paplomatas, Dept. Plant Pathology, University of California-Davis, Davis, CA 95616.

Speaker #7: PCR use in plant virus diagnosis. Laurene Levy and Ed Podleckis, USDA-ARS, Beltsville, MD 20705.

Speaker #8: Introduction to light microscopy of viral inclusions. Larry Brown[1] and Gary Simone,[2] Florida Dept. of Agriculture and Consumer Services, Gainesville, FL 32602,[1] and Dept. Plant Pathology, University of Florida, Gainesville, FL 32611[2]

Plant Disease Diagnostic Sheets

On the following pages are the last of the Diagnostic Sheets that have been produced by the subcommittee of the Plant Disease Diagnostics Committee (American Phytopathological Society). (Please see the December, 1990 issue for additional information.) As mentioned last time, the subcommittee hopes that if practices described in these sheets are less useful than those you are using to diagnose these diseases, that you will step forward to revise, edit, or update the appropriate diagnostic sheet.

Comments on these sheets, including any on editorial style, factual information, and other miscellaneous tidbits, should be addressed to Charles Semer IV, Department of Plant Pathology, University of Florida, Gainesville, FL, 32611; phone (904) 392-7241.

Disease: Fusarium Wilt of Tomato

Primary Economic Host: Lycopersicon esculentum Mill. (tomato)

Pathogen: Fusarium oxysporum (Schlecht.) f. sp. lycopersici (Sacc.) Snyder & Hansen

Symptoms and signs:

Seedlings: Infected seedlings are stunted, the older leaves droop, curve downward, and some may turn yellow. The vascular tissue becomes dark brown and the brown discoloration usually extends to the stem tip. The base of an affected stem often enlarges and the plants frequently wilt and die.

Mature Plants: Symptoms on older plants generally become apparent during the interval from blossoming to fruit maturation. The earliest symptom is the yellowing of the older leaves. The yellowing often develops on only one side of the plant, and the leaflets on one side of the leaf frequently turn yellow before those on the other side. The one-sided yellowing of plant and leaf is highly characteristic of the disease. The yellowing gradually includes most of the foliage and is accompanied by wilting of the plant during the hottest part of the day. The wilting progressively becomes more severe until the plant dies. The vascular tissues usually are dark brown, with the browning extending far up the stem. It is especially noticeable in a petiole scar. The pith remains healthy. Fruit infection occasionally occurs and can be detected by browning of the vascular tissue within the fruit.

Isolation:

Excise small chips of tissue from the base of symptomatic stem tissue, soak in 0.52% sodium hypochlorite for 5 minutes, and place directly onto the isolation medium (generally potato dextrose agar (PDA)). Rinsing the disinfested chips in sterile water before plating usually is not necessary. Optimum temperature for growth is 28 C.

Identification:

The mycelia are delicate white to pink often with a purple tinge, and are sparse to abundant. Microconidia are borne on simple phialides arising laterally, abundant, oval-ellipsoid, straight to curved 5-12 x 2.2-3.5 µm, and nonseptate. Macroconidia, which are sparse to abundant, are borne on branched conidiophores or on the surface of sporodochia. They are thin walled, 3-5 septate, fusoid-subulate and pointed at both ends, with a pedicellate base, and 27-46 x 3-5 µm in size if three septate or 35-60 x 3-5 µm if five septate; three septate spores are more common. Chlamydospores, both smooth and rough walled, are abundant, terminal or intercalary, and generally are solitary, but occasionally in pairs or chains. Optimum temperature for growth is 28 C.

Pathogenicity:

Three physiological races of this pathogen have been reported. Race 1 is the most widely distributed race, having been reported from most geographical areas. Race 2 was found in Ohio as early as 1940, but did not cause economic damage until its discovery in Florida in 1961. Thereafter it was reported in several states of the United States and in several other countries, including Australia, Brazil, Great Britain, Israel, Mexico, Morocco, the Netherlands, and Iraq. Race 3 was reported in 1966 in Brazil. Since then it has been found in Australia and the United States (Florida and California). Inoculation of differential tomato cultivars is necessary for race identification. The cultivar 'Bonny Best' has the I-1 gene for resistance to race 1, but is

susceptible to races 2 and 3. 'Walter' possesses the I-2 gene (and presumably the I-1 gene) and is resistant to races 1 and 2, but susceptible to race 3. 'Florida 13R-1' is a breeding line with the I-3 gene (and presumably the I-1 and I-2 genes) and is resistant to races 1, 2, and 3.

Inoculation:

Uproot 2-week old seedlings, dip the injured roots (the roots can be further damaged by snipping with scissors, but this is not needed) and swish them around in a spore suspension containing 20 to 60 million micro- or macrospores/ml, and transplant into a light, acid (pH 4.0 - 6.0) soil or medium. Incubate at 28 C. The spore suspension is prepared by comminuting one or two PDA plates of Fusarium in 100 ml water. Symptoms will become apparent in 10 to 14 days. Field inoculation can be accomplished by raising Fusarium on sterile vermiculite saturated with a nutrient solution high in ammonia-nitrogen and micronutrients. After 7 to 10 days incubation at 28 C put 100 ml of the infested vermiculite into each plant hole two weeks after the field has been pasteurized with a broad-spectrum fumigant or with steam.

Storage of organism:

Maintain cultures in tubes of sterile soil or soil-medium mixes. Cultures also can be kept on PDA, but frequent culture renewal is required.

Selected references:

Alexander, L. J., and Tucker, C. M. 1945. Physiological specialization in the tomato wilt fungus Fusarium oxysporum f. lycopersici. J. Agric. Res. 70:303-313.

Beckman, C. H. 1987. The Nature of Wilt Diseases of Plants. APS Press. St. Paul. 175 p.

Engelhard, A. W., J. P. Jones, and S. S. Woltz. 1989. Nutritional factors affecting Fusarium wilt incidence and severity. Pages 337-352 in: Vascular Wilt Diseases of Plants. E. C. Tjamos and C. H. Beckman, eds. NATO ASI Series, Vol. H28. Springer-Verlag. Berlin, Heidelberg, New York, London, Paris, Tokyo.

Jones, John Paul, and S. S. Woltz. 1981. Fusarium-incited diseases of tomato and potato and their control. Pages 157-168 in: Fusarium: Diseases, Biology, and Taxonomy. P. E. Nelson, T. A. Toussoun, and R. J. Cook, eds. The Penn. State Univ. Press. University Park and London.

Jones, John P., Arthur W. Engelhard, and S. S. Woltz. 1989. Management of Fusarium Wilt of vegetables and ornamentals by macro- and microelement nutrition. Pages 18-32 in: Soilborne Plant Pathogens: Management of Diseases with Macro- and Microelements. Arthur W. Engelhard, ed. APS Press. St. Paul, MN.

Toussoun, T. A., and Paul E. Nelson. 1968. Fusarium: A Pictorial guide to the Identification of Fusarium Species. The Penn. State Univ. Press. Univ. Park and London. 51 p.

Walker, J. C. 1971. Fusarium Wilt of Tomato. The American Phytopathological Soc. Monograph No. 6. 56 p.

Contributed by: John Paul Jones and S. S. Woltz

Disease: Leaf Rust of Soybean

Primary Economic Host: Soybean (Glycine max (L.) Merr.)

Pathogen: Phakopsora pachyrhizi Sydow

Symptoms and signs:

Initially leaf lesions are small (0.5 mm^2), polygonal in shape, and light in color turning gray to tan or brown as the lesions enlarge. Lesions are often restricted by leaf veins and develop to a size of 2-5 mm^2. In mass the lesions appear tan to dark brown or reddish brown with one to many erumpent, globose, ostiolate uredia. Under field conditions, heavily infected leaves are off color, often become yellowed, and drop from the plant prematurely.

Isolation:

The fungus has not been cultured on artificial media, but can be maintained on inoculated detached leaves or on living plants inoculated in the greenhouse or planted year-round in a disease garden.

Identification:

Uredia are globose, subepidermal, erumpent, light cinnamon to reddish brown, more abundant on lower leaves, and range in size from 100-200 µm. Paraphyses form a dome-like covering over the sporophore. Uredospores are globose, subglobose, ovate, or ellipsoidal, hyaline to light yellow-brown, finely echinulate, and range in size from 20-28 x 17-23 µm.

Telia do not frequently occur unless induced by cooler temperatures (< 21^{o} C) for at least several days or weeks. Telia are subepidermal, and are often masked among the uredia. Initially they are orange to brown in color turning black with age. The teliospores are smooth-walled; one-celled; yellow to brown; clavate, oblong, or angulate in shape; and 20-35 x 8-15 µm. Basidia and basidiospore production has been induced, but has not been recorded to occur in nature.

Pathogenicity:

Infected leaves (those with abundant uredia) are placed inside polyethylene bags overnight and the next day soaked in water for 5-10 minutes. The suspension is filtered and used to atomize plants. All ages of plants are susceptible. Atomized plants should be covered for 12-24 hours and kept at about 22^{o} C. Symptoms occur 4-10 days after inoculation.

Storage of pathogen:

Urediospores can be harvested and stored dried and will last for long periods of time in a deep freeze or in liquid nitrogen. The fungus can also be maintained on live tissue (either detached leaves or continuous inoculation of living plants).

Reported host range:

Soybean, other Glycine species, and numerous other legumes.

Geographic range:

Primarily the eastern hemisphere, but reported also from Puerto Rico, Brazil and other South American countries, and Africa.

Suggested taxonomic references:

Arthur, J. C. 1917. Uredinales of Puerto Rico based on the collections made by H. H. Whetzel and E. E. Olive. Mycologia 9:55-104.

Sydow, H., and Sydow, P. 1914. A contribution to knowledge of the parasitic fungi of the island of Formosa. Ann. Mycol. 12:108.

Bibliography:

Bromfield, K. R. 1984. Soybean Rust. Monogr. 11. American Phytopathol. Society, St. Paul, MN. 65 pp.

Hartman, G. L. and Wang, T. C. 1991. Soybean rust development and the quantitative relationship between rust severity and soybean yield. Plant Dis. 75:(in press).

Koch, E., and Hoppe, H. H. 1987. Germination of the teliospores of Phakopsora pachyhizi. Soybean Rust Newsl. 8:3-4.

Saadaoui, E. M., and Tschanz, A. T., eds. 1987. Bibliography of Soybean Rust. 1885-1986. Asian Vegetable Research and Development Center, Shanhua, Taiwan. 103 pp.

Tschanz, A. T., and Shanmugasundaram, S. 1985. Soybean ruts. Pages 562-567 in: Proc. World Soybean Res. Conf. III. R. Shibles, ed. Westview Press, Boulder, CO.

Vakili, N. G., ed. 1978. Proc. Workshop Soybean Rust West. Hemisphere. U. S. Department of Agriculture, Agriculture Research Service, Mayaguez Institute of Tropical Agriculture, Puerto Rico. 81 pp.

Yeh, C. C., Sinclair, J. B., and Tschanz, A. T. 1982. Phakopsora pachyrhizi: Uredial development, uredospore production and factors affecting teliospore formation on soybeans. Aust. J. Agric. Res. 33:25-31.

Contributed by: G. L. Hartman

Disease: Red Leaf Blotch of Soybean

Primary Economic Host: Soybean (Glycine max [Glycine max (L.) Merr.])

Pathogen: Dactuliochaeta glycines (R. B. Stewart) Hartman & Sinclair (Syn. Pyrenochaeta glycines R. B. Stewart)

Symptoms and signs:

Lesions on leaves initially are pink to red, circular to angular, and 1-3 mm in size. Lesions enlarge and coalesce to form large necrotic blotches up to 2 cm in diameter. Lower leaves usually have more lesions and often drop from the plant prematurely. Pycnidia and sclerotia may occur on the lower or upper side of leaves associated with coalesced lesions.

Isolation:

The fungus can be isolated from lesions by 1) removing sclerotia from infected leaves and plating them directly on medium; or 2) lesions can be incubated under moist conditions which causes conidia to ooze from pycnidia. The conidia can be transferred with a sterile needle to water, and then a drop streaked on water agar. Single conidia can be isolated and transferred to PDA.

Identification:

On infected leaves, both pycnidia and sclerotia can be measured and compared to the fungal description. In culture, morphological sizes vary, and both pycnidia and sclerotia are often not produced. Pycnidia are 87 to 298 µm in diameter with aseptate to multiseptate setae clustered mostly around the ostiole. Conidiogenous cells are monophialidic and ampulliform. Conidia are ellipsoidal, one-celled, and 4-8 x 1-3 µm. Sclerotia are 96 to 357 µm in diameter, mostly spherical, and dark brown to black with setae 5-36 µm long.

Pathogenicity:

Plants can be inoculated in several ways. A simple way is to collect sclerotia from infected leaves and place them on test plant leaves under moist conditions. The sclerotia will germinate and mycelium will infect causing lesions to develop 2-4 days later. In pure culture, some isolates regularly produce pycnidia that sporulate. Conidia collected from these pycnidia can be suspended in water and atomized on plants kept at a high relative humidity for 1-2 days.

Storage of pathogen:

Sclerotia survive for long periods of time and can probably be stored under refrigeration indefinitely.

Reported host range:

Soybean and a perennial legume, Neonotonia wightii (Arnott) Lackey, in nature. Other hosts have been reported using detached leaves.

Geographic range:

Limited distribution to countries in Central and Southern Africa, primarily Zaire, Zambia, Zimbabwe.

Suggested taxonomic references:

Datnoff, L. E., Levy, C., Naik, D. M. and Sinclair, J. B. 1986. Dactuliophora glycines, a sclerotial state of Pyrenochaeta glycines. Trans. Br. Mycol. Soc. 87:297-301.

Hartman, G. L., and Sinclair, J.B. 1988. Dactuliochaeta a new genus for the fungus causing red leaf blotch of soybeans. Mycologia 80:696-706.

Leakey, C. L. A. 1964. Dactuliophora, a new genus of Mycelia Sterilia from tropical Africa. Trans. Br. Mycol. Soc. 47:341-350.

Stewart, R. B. 1957. An undescribed species of Pyrenochaeta on soybeans. Mycologia 49:115-117.

Bibliography:

Datnoff, L. E., Naik, D. M., and Sinclair, J. B. 1987. Effect of red leaf blotch on soybean yields in Zambia. Plant Dis. 71:132-135.

Hartman, G. L., Datnoff, L. E., Levy, C., Sinclair, J. B., Cole, D. L., and Javaheri, R. 1987. Red leaf blotch of soybeans. Plant Dis. 71:113-118.

Contributed by: G. L. Hartman

Disease: Red ring disease, Little leaf

Primary Economic Host: Cocos nucifera (Coconut palm),
Elaeis guineensis (African oil palm)

Parasite: Bursaphelenchus cocophilus (Cobb) Baujard
(=Rhadinaphelenchus cocophilus)
(Nemata: Aphelenchida: Aphelenchoididae)

Pathogen Common Names: Red ring nematode, coconut palm nematode

Vector Common Name: Palm weevil (Rhynchophorus palmarum)

Symptoms and signs:

Symptoms vary with host species, cultivar, and environmental factors. Classical red ring symptoms in African oil palm include progressive premature yellowing and death of older leaves. These leaves often break at the petiole and hang for a long period of time while the symptoms progress to younger leaves. New leaves in diseased trees are usually more of a pale yellowish-green color than those observed in healthy palms. Examination of a stem cross-section will reveal a brown, cream, or rose-colored ring which is a few centimeters in thickness and concentric to the periphery of the stem. The ring is not necessarily continuous throughout the entire stem length. Feeding by the larval stage of the weevil vector often destroys the apical bud. Generally oil palms older than 5 years are infected and death can occur within several months. Third-stage juveniles of the red ring nematode can be harvested from tissues adjacent to and within the ring from the stem of African oil palm and range in number from 0-5,000 nematodes/gram of tissue. Little leaf symptoms and a combination of red ring and little leaf symptoms have been documented in red ring nematode-infested African oil palms. Little leaf symptoms can also be caused by a rot of the anterior stem, recovery from spearleaf rot, Fusarium wilt, boron deficiency, and attack by some species of insects. In oil palms with typical little leaf symptoms, leaf color remains normal. However, palms begin abnormal production of very short leaves which give crowns the unusual appearance of a feather duster. As the disease progresses, there is a decrease in leaf size and surface area to the point where the leaf is reduced to a leafless rachis with suberized lesions over most of its surface. New leaves and inflorescences are aborted and palms become unproductive. Red ring nematodes can be recovered from necrotic lesions in the middle and distal parts of unpresented leaves (-11 to -2; counting backwards from the spearleaf [=1]).

Classical red ring symptoms in coconut palm often include premature nut fall (in nut bearing palms), whithering of inflorescences, and yellowing, bronzing, and death of progressively younger leaves. Yellowing of leaves usually starts at the tips of the pinnae and moves inward to the rachis and then to the base of the petiole. Several of the dying or dead leaves will often break close to the petiole and remain hanging from the stem. A stem transverse section will reveal a discrete brick to brownish-red ring which is 2-6 cm wide and occurs 2-6 cm within the stem periphery. The cortex of roots and leaf petioles can also be discolored yellow to brownish-red. In longitudinal section, discoloration is usually continuous throughout the length of the stem, appearing as two bands which unite at the base and form discontinuous lesions near the crown. Coconut palms 3-10 years-old usually die within several months of infection. Severe damage to the crown of red ring-diseased coconut palms is caused by larval feeding of the weevil vector. In areas such as El Salvador, older palms (> 20 years old) have been reported with red ring disease displaying less definitive symptoms with a more prolonged death. Third-stage juveniles can be harvested from the discolored tissue of the ring (up to 10,000 nematodes/g of tissue), from leaf petioles, or roots to confirm disease diagnosis from symptoms. Little leaf symptoms have also been reported for coconut palms. Nematodes can be recovered from areas around lesions on the

middle to distal regions of the younger leaves. Coconut and African oil palms younger than 2.5 years-old can not be infected experimentally with the red ring nematode nor have symptoms been observed in palms of this age in the field.

Isolation:

Tissue from the discolored area in the stem is chopped into thin chunks (3.0 x 3.0 x 0.5 cm) with a cutlass, soaked in tap water for ca. 4 hours, and filtered through nested nos. 12 and 400 USA standard testing sieves. The nematodes are backwashed off the no. 400 sieve into a large Baermann funnel with a piece of absorbent cotton positioned at the stem base of the funnel. The apparatus is left overnight at room temperature (27° C) and the nematodes are collected for observation. Mostly third-stage juveniles are harvested from the discolored tissue whereas adults, eggs, and juveniles can be harvested from regions of the stem where the lesions have not coalesced to form a ring.

Identification:

Bursaphelenchus cocophilus is a very long, thin nematode; the females and males have been reported to be 60-139 and 65-179 times longer than wide, respectively, with the greatest body width being less than 15.5 µm with a rounded terminus. Males have seven caudal papillae; one ventral preanal papilla, one pair of subventral preanal or adanal papillae, and two pairs of subventral postanal papillae. The distal ends of the spicules in the males are heavily sclerotized and the caudal alae form a spade-shaped flap (= bursal flap). Third-stage juveniles from coconut palm usually range from 700-920 µm and have a pointed tail with or without a mucron. The metacorpus is usually not well developed in third-stage juveniles from the palm or the weevil vector and the stylet is not visible.

Storage and culture of organism:

Survival of third-stage juveniles of B. cocophilus is very poor under unsterile conditions in water at room temperature or in the refrigerator (100% mortality in < 7 days). Survival can be prolonged for 70-80 days at room temperature when nematodes are surface-sterilized and stored in autoclaved red ring nematode-diseased stem tissue extract (R) or R plus D-glucose or lactose (Giblin-Davis et al. 1989). Red ring nematode can be inoculated into and cultured in coconut palms older than 3 years old, or in husks of nearly mature coconut fruits, or in an exised leaf stalk (leaf 6 to 13 on a nut-bearing coconut) which has been trimmed of the pinnae and the cut ends have been parafin coated. Cultures in immature fruits or leaf stalks must be subcultured about once every 4 weeks.

Reported host range:

Acrocomia aculeata
Attalea sp.
Astrocaryum standleyanum
Cocos nucifera
Elaeis guineensis
Euterpe pacifica
Guilielma gasipaes
Guilielma sp.
Jessenia polycarpa
Mauritia flexuosa
Mauritia mexicana
Maximiliana maripa
Oenocarpus distichus
Phoenix canariensis
Phoenix dactylifera
Roystonea oleraceae
Roystonea regia

Vector:

Rhynchophorus palmarum (Arthropoda: Coleoptera: Curculionidae). Red ring diseased palms are attractive to R. palmarum adults which oviposit in and colonize them. The developing weevil larvae are parasitized by third-stage juveniles of B. cocophilus which persist in the insect through metamorphosis, and appear to aggregate around the genital capsule of the adult weevil. The adult weevils emerge from their cocoons in rotted palms and disperse to apparently healthy or stressed and/or wounded palms where females deposit nematodes during oviposition. In the case of healthy palms, the nematodes enter oviposition wounds and begin their life cycle, eventually causing the wilt associated with red ring disease which attracts more weevils to colonize and thoroughly destroy the palm.

Geographical distribution:

The red ring nematode is co-distributed with its insect vector, R. palmarum, in the lower Antilles, and Mexico southward, through Central America, into South America, where it has been reported from Colombia, Surinam, Guyana, Ecuador, Venezuela, and Brazil.

Suggested references:

Bernard, E. C. 1987. Proposed common names for plant-parasitic nematodes. Nematology Newsletter 33 (3):21-23.

Brathwaite, C. W. D. and M. R. Siddigi. 1975. Rhadinaphelenchus cocophilus. C.I.H. Description of Plant Parasitic Nematodes, Set 5, No. 72.

Chinchilla, C. 1988. El Sindrome del anillo rojo-hoja peque:a en palma aceitera y cocotero. Boletin Technico 2:113-136.

Dean, C. G. 1979. Red ring disease of Cocos nucifera L. caused by Rhadinaphelenchus cocophilus (Cobb, 1919) Goodey, 1960. An annotated bibliography and review. Technical Communication No. 47 of Commonwealth Inst. Helminthol. 70 pp.

Esser, R. P. and J. A. Meredith. 1987. Red ring nematode. Fla. Dept. Agric. & Consumer Serv., Div. Plant Industry. Nematology Circ. No. 141.

Fenwick, D. W. and S. Mohammed. 1964. Artificial infections of seednuts and young seedlings of the coconut palm with the red-ring nematode Rhadinaphelenchus cocophilus (Cobb). Nematologica 10:459-463

Gerber, K. and R. M. Giblin-Davis. 1990. Association of the red ring nematode, Rhadinaphelenchus cocophilus, and other nematode species with Rhynchophorus palmarum (Coleoptera: Curculionidae). J. Nematol. 22:143-149.

Gerber, K., R. M. Giblin-Davis, and J. Escobar-Goyes. 1990. Association of the red ring nematode, Rhadinaphelenchus cocophilus, with weevils from Ecuador and Trinidad. Nematropica 20:39-49.

Gerber, K., R. M. Giblin-Davis, R. Griffith, J. Escobar-Goyes, and A. D'Ascoli Cartaya. 1989. Morphometric comparisons of geographic and host isolates of the red ring nematode, Rhadinaphelenchus cocophilus. Nematropica 19:151-159.

Giblin-Davis, R. M., K. Gerber, and R. Griffith. 1989. In vivo and in vitro culture of the red ring nematode, Rhadinaphelenchus cocophilus. Nematropica 19:135-142.

Giblin-Davis, R. M., M. Mundo-Ocampo, J. G. Baldwin, K. Gerber, and R. Griffith. 1989. Observations on the morphology of the red ring nematode, Rhadinaphelenchus cocophilus. Revue Nematol. 12:285-292.

Giblin-Davis, R. M. 1991. The potential for introduction and establishment of the red ring nematode in Florida. Principes 35:(In press).

Griffith, R. 1987. Red ring disease of coconut palm. Plant Disease 71:193-196.

Contributed by: Robin M. Giblin-Davis

Disease: Rice Blast

Primary Economic Host: Oryza sativa (rice)

Pathogen: Pyricularia oryzae

Symptoms:

a) On leaves. The fungus produces spots or lesions which are typically elliptical with more or less pointed ends. The center of the spot is usually gray or whitish, and the margin is usually brown or reddish brown. Both the shape and color of the spots vary, depending upon environmental conditions, age of the spots, and degree of susceptibility of the rice cultivar. The spots usually begin as small, water-soaked, whitish, grayish or bluish dots. They enlarge quickly under moist conditions on susceptible cultivars. Fully developed lesions reach 1-1.5 cm long, 0.3-0.5 cm broad, and usually develop a brown margin. Spots on susceptible cultivars growing under moist, shaded conditions show very little brown margin, but instead sometimes have a yellow halo around the spot. On highly resistant cultivars, only minute brown specks of pin-head size may be observed.

   Numerous spots may appear on a leaf which may soon be killed; this is followed by drying of the leaf sheath. Seedlings or plants at tillering stage are often completely killed.

b) On stem. Infected nodes turn black. The stem easily breaks on the infected nodal point.

c) On panicles. Any part of the panicle may be infected, producing brown lesions. Areas near the panicle base are often attacked, causing the "rotten neck" or "neck rot" symptoms, and the panicles often fall over. Panicle branches and glumes are also attacked.

Signs:

To confirm identification mode through symptoms, spores of the causal fungus should be observed from the lesions. Infected leaves newly collected from the field may not yield spores, which may have been dislodged; hence, the following procedure is suggested.

a) Incubate specimens in a moist chamber (a plastic bag, petri dish, or plastic-covered dish) at room temperature.

b) After 24-48 hr, examine for fungal spores using a compound microscope. Saprophytes may overgrow the lesions if leaves are incubated longer.

Spore identification:

Conidia obclavate, tapering at apex. Truncate at base, 2-septate. 20-22 x 10-12 µm. Produced in clusters.

Isolation:

In case no spores are observed, isolate the fungus by tissue plating method as follows:

a. Cut lesions into halves to include healthy tissues.

b. Disinfest the cut leaf sections and plate onto potato dextrose agar.

c. After 3-4 days, observe for hyphal growth.

d. To produce spores, expose culture plates to fluorescent light continuously for 3 days.

e. Examine for spores of the fungus using compound microscope.

Note: Currently, single-spore isolation method is used to isolate the fungus by picking up spores from infected leaves and streaking on plates of water agar using glass rod with rounded end, and transferring single spore to plated prune agar using capillary tube.

Pathogenicity:

Inoculate with a conidial suspension in water. Optimum spore germination 26-28 C. Invasion time 10 hr at 32 C, 8 at 28 C, 6 at 24 C. Maximum infection 24-28 C with 16-24 hr continuous wetting.

Host Range:

Oryza sativa (rice). Numerous strains of the fungus and varietal host resistance. Grasses of 38 species.

Distribution:

One of the most widely distributed plant diseases. Found in 85 countries.

Selected Reference:

Ou, S. H. 1985. Rice Diseases. 2nd ed. C.A.B. International Mycological Institute. Great Britain. 380 p.

Contributed by: P. Teng

Disease: Root Nematode

Primary Economic Host: Oryza sativa (rice)

Pathogen: Hirschmaniella oryzae

Symptoms:

There is no specific symptom of the disease on the above-ground parts except a gradual retardation of growth. Based on separate experiments using inoculated plants, the following symptoms were observed: root tissue became discolored; rate of tillering was reduced by 50-60%; height and weight of foliage and roots were reduced

Root rot of rice induced by physiological factors (autumn decline) becomes more serious when this nematode is present.

Isolation from soil:

Baermann funnel method - requires only a 12-15 cm glass funnel, a 6 cm circular piece of screen, a piece of rubber tubing and a pinchcock, a stand to the funnel, and some cheese-cloth.

1. Place wet-strength tissues (Scotties or Kleenex) on screen in funnel.
2. Put 100 cc soil sample on the tissue paper.
3. With pinchcock closed, flood the soil with water and allow to stand overnight. The live nematodes will migrate through tissues into the clear liquid and sink to the bottom just above the pinchcock. Over 90% of the nematodes can be recovered by drawing off 5 ml of water.

Combination of sieving and Baermann funnel method:

1. Place 200 ml soil sample in plastic pail and thoroughly mix with water.
2. After mixing, set aside for 30 seconds; then, decant the suspension through the mesh sieves no. 50, 200, and 325, set one on top of each other from coarse to the finest.
3. Wash the residue collected in 325 mesh sieve using rubber hose.
4. Transfer nematodes with crop debris caught in 325 sieve in a breaker using a wash bottle.
5. Set the suspension in PVC pipe ring with nylon mesh.
6. Transfer setup in petri plate and add water up to the level above the nylon mesh.
7. Examine suspension after 24 hrs.

Isolation procedure from roots:

Maceration or blender method:

1. Cut 3-gm root sample into small pieces

2. Dispense in beaker and add 150 ml water
3. Blend for 15 seconds
4. Transfer materials in PVC pipe ring with nylon mesh
5. Transfer set up in petri plate and add water up to the level above the nylon mesh
6. Examine suspension after 24-48 hrs

Disease cycle:

The nematode enters the roots and lay eggs in relatively short time, larvae hatch and live in the root cortex for a time, leave the roots when mature and become adult in the soil. H. oryzae has 2 generations a year.

The nematodes are still alive in the soil after 10 weeks in the absence of host plants. Usually, they overwinter in dead rice roots in larval or adult form.

The application of nitrogen fertilizer seems to increase the nematode population, while potash fertilizer or compost maintains a low population level throughout the season. Based on separate studies the nematode population was observed to be low in fields applied with:

-calcium silicate or compost (applied annually)
-soil conditioners containing spores of nematode-trapping fungi
-neem and mustard seed cakes

H. oryzae is an ectoparasitic nematode. Diseases caused by ectoparasitic nematodes are considerably to diagnose because the association between the plant and nematode is circumstantial, i.e. nematodes are found only in the soil.

Rarely do they adhere to root surfaces that are pulled to be examined. The presence of microscopic root lesions gives some support for a pathogenic relationship, but is not conclusive evidence.

Distribution:

Widely distributed in all rice-growing regions of the world.

Suggested Taxonomic Reference:

Luc, M.; Goodey, J. B. (1962) Hirschmannia n.g. differentiated from Rhadopholus Thorne, 1949. (nematoda: Tylenchoidea). Nematologica 7,197-202.

Suggested References:

Ou, S. H. 1982. Rice Diseases. C.A.B. International Mycological Institute. Great Britain. 380 p.

Contributed by: P. Teng

Disease: Sheath Rot

Primary Economic Host: Oryza sativa (rice)

Pathogen: Sarocladium oryzae (Acrocylindrium oryzae)

Symptoms:

The rot occurs on the uppermost leaf sheaths enclosing the young panicles. The lesions start as oblong or somewhat irregular spots, 0.5-1.5 cm long, with brown margins and gray centers, or they may be grayish brown throughout. They enlarge and often coalesce and may cover most of the leaf sheath. The young panicles remain within the sheath or only partially emerged. An abundant whitish growth may be found inside affected sheaths and young panicles are rotted.

Identification:

Mycelium white, sparsely branched,septate, 1.5-2 µm in diameter. Conidiophores arising from the mycelium, slightly thicker than vegetative hyphae, branched once or twice, each time with 3-4 branches in a whorl. The main axis 15-22 x 2-2.5 µm, terminal branches tapering towards the tips, 23-45 µm long, 1-5 µm at the base. Conidia may be borne simply on the tip, produced consecutively, hyline, smooth, single-celled, cylindrical, 4-9 x 0.5-1.6 µm.

Conidia 2.1-8.5 x 0.5-1.6 µm from host, 1.8-13 x 1-1.6 µm from culture. Borne simply on the tip, produced consecutively, hyaline, smooth. Optimum growth: 30-31 C.

Pathogenicity Testing:

Take rice grains with husk intact. Place into a beaker or flask, cover in water. Allow to imbibe for 24 hours. Drain excess water, autoclave. After the rice has cooled, inoculate with mycelial plugs containing the pathogen. Cover and incubate at 30-31 C for two weeks. After incubation spread out the infested grains and allow them to dry.

Use a single infested rice grain culture and place it between the leaf sheath and culm at the tillering to panicle initiation stage. Constant, severe infection is produced.

Note: Wounding of host facilitates infection. Most of the infected plants in Southeast Asia were also infested by stemborers or had other injuries on stems.
Some infected plants were also infected by yellow dwarf, tungro, and ragged stunt viruses in seperate studies.

Host Range:

Did not infect other Gramineae tested.

Distribution:

Japan, Southeast Asia, Indian subcontinent, USA, West Africa.

Suggested Taxonomic Refereences:

Gams, W., Hawksworth, D. L. (1975) The identity of Acrocylindrium oryzae Sawada and a similar fungus causing sheath-rot of rice. Kavaka 3,57-61. (Review of Plant Pathology 55, 1010.)

Suggested References:

Ou, S. H. 1982. Rice Diseases. C.A.B. International Mycological Institute. Great Britain. 380 p.

Contributed by: P. Teng

Disease: Sheath Blight

Primary Economic Host: Oryza sativa (rice)

Pathogen: Thanatephorus cucumeris (Rhizoctonia solani)

Symptoms:

The pathogen causes irregularly shaped spots on the leaf sheath which are, at first, greenish-gray, varying from 1-3 cm long. The center of the spot becomes grayish-white, with a brown margin. Lesions are also formed on the upper leaf sheaths and on the leaf blades. The presence of several large spots on a leaf sheath usually causes the death of the whole leaf, and in severe cases all the leaves of a plant may be blighted in this way.

Signs:

Sclerotia are formed on or near the lesions but are easily detached. Under humid conditions, the mycelia of the fungus may grow over the surface of the leaf sheaths and can spread a considerable distance (several cm) in 24 hours.

Taxonomy:

Morphology of mycelium and sclerotia varies greatly. The mycelium is colorless when young, becoming yellowish brown when older, 8-12 µm in diameter, with frequent septations. Three types of mycelium are produced. Straight runner hyphae, at intervals, give rise to short, swollen much branched or lobate mycelia from which penetration pegs arise. The lobate mycelium infects tissue and produces lesions. It is produced in patches of varying sizes and shapes which determine the sizes and shapes of the lesions. On an infected stem, the runner hyphae may cover most parts of the stem but the lobate mycelium is found only on the lesions. The third type of mycelium consists of monilioid cells involved in the formation of sclerotia. Sclerotia are superficial, more or less globose but flattened below, white when young becoming brown to dark brown. The teleomorph, described by Sawada (1912) and Matsumoto (1932) has the measurements; basidia 10-15 x 7-9 µm; sterigmata 4.5-7 x 2-3 µm numbering 2-4; basidiospores 8-11 x 5-6.5. Survival of the pathogen is as mycelium or sclerotia in soil.

Isolation:

Affected leaf sheaths can be collected, surface sterilized in 0.5% sodium hypochlorite for 1 minute, rinsed in sterile water and blotted dry. Cut the lesion so that both fresh tissue and a part of the lesion are removed. Place the selected tissue on Potato Dextrose agar and incubate at 25 C. Sclerotial production is affected by the avaliable carbon and nitrogen sources. A medium containing proline will produce many vary large sclerotia (3.5 x 3.1 mm). Abundant moderately sized sclerotia were produced on medium with sodium nitrate, serine, glutamine, alanine and aspartic acid.

Pathogenicity Testing:

Using seedlings three weeks old, inoculate with a culture of the fungus on rice straw by placing the straw on the soil surface at the base of the seedlings. Readings are taken 2 weeks after inoculation.

Host range:

The fungus isolated from rice has been shown to infect many other plants and similar fungi isolated from other crops have been shown to infect rice.

Storage of pathogen:

Storage of sclerotia in dry soil has been reported with survival of viable propagules reported up to nine months.

Suggested Taxonomic References:

Parmeter, J. R., Jr.(ed.) (1970) Rhizoctonia solani, Biology and Pathology. Berkeley, Los Angeles and London. University of California Press. 255 pp.

Sneh, B., Lee Burpee, and Atera Ogoshi (in Press) Identification of Rhizoctonia species. APS Press.

Contributed by: P. Teng

Disease: Tungro Virus Disease (Penyakit merah)

Primary Economic Host: Oryza sativa (rice)

Pathogen: Tungro Virus

Symptoms:

The major symptoms on tungro-affected rice plants are stunting and discoloration of the leaves; the colors range from various shades of yellow to orange. Discoloration starts from the tip of the leaf and may or may not extend to the lower part of the leaf blade; often only the upper portion is discolored. Young leaves may have a mottled appearance and old leaves show rusty-colored specks of various sizes. Stunting is severe in susceptible cultivars, but slight on those which are more resistant.

The number of tillers produced by infected plants is usually slightly reduced but does not differ greatly from the number produced by healthy plants.

In susceptible cultivars, stunting and leaf discoloration usually persist throughout the life of the plant, and severely diseased plants may die at an early or later stage of growth.

When infection occurs late in the growing season, no symptoms develop even in susceptible cultivars. Infected plants have delayed flowering. The panicles are small and not completely developed, and bear mostly sterile or partly-filled grains often covered with dark brown specks.

Diagnostic procedure:

To confirm tungro as diagnosed through symptoms, the iodine test is carried out as follows:

a. Collect infected leaves by cutting smoothly at the base.

b. Dip cut end in iodine solution diluted at 1:4 (1 part iodine and 4 parts water).

c. Observe results after 5 minutes; for positive result, dark discoloration develops at the leaf base spreading upward mainly through the midrib.

The iodine test is based on the principle that tungro-infected leaves contain large quantities of starch, which turns black or dark brown when stained with iodine.

For a more accurate test to confirm tungro using infected leaves or to detect the presence of tungro particles in leaves without symptoms, latex test can be carried out as follows:

a. Macerate infected leaves and add few drops of water.

b. Dispense sap in small test tube and add an equal amount of latex suspension [latex + antiserum (sensitized with either spherical or bacilliform virus particles or both)].

c. Shake tube for 30 minutes.

d. Transfer one drop on glass slide and examine under a compound microscope; for positive result, there will be clumping of latex particles.

Causal agent:

Both isometric and bacilliform particles present.

Vector:

Nephotettix virescens is principal vector.

Host range:

Many species of wild rice. Eleusine indica (L.) Gaertn., Echinochloa colonum (L.) Link and Echinochloa crusgalli Beauv. are considered to be alternate hosts.

Distribution:

Tungro and similar diseases are limited to the countries of Southeast Asia.

Suggested Taxonomic References:

Basu, A. N., Mishra, M. D., Naizi, F. R., Ghosh, A. (1976) A proposed Key to the strains of rice tungro virus. International Rice Research Newsletter 1(2), 6-7.

Suggested References:

Ou, S. H. 1985. Rice Diseases. C.A.B. International Mycological Institute. Great Britain. 380 p.

Contributed by: P. Teng

Disease: Target Spot of Tomato

Primary Economic Hosts: Cucumber, Tomato

Pathogen: Corynespora cassiicola

Symptoms and signs:

On cucumber, target spot symptoms begin as small, yellow leaf flecks that enlarge to about 1 cm in diameter. They often become quite angular and care must be taken to avoid confusion with angular leaf spot. Individual mature lesions are very light tan with a thin brown margin. Eventually, these lesions may coalesce and form large areas of necrotic tissue. Microscopic examination of fungal growth from the diseased tissue is important in making a firm diagnosis. On tomato leaves, early symptoms consist of small, necrotic spots that later develop sunken tan to light brown centers. In some cultivars, a yellow halo surrounds the leaf spots. Coalescence of lesions leads to a general blighting of leaves. Especially in the early stages, target spot can easily be confused with bacterial spot. It is important that laboratory tests confirm suspected target spot. Symptoms on tomato fruit are quite striking and not easily confused with other problems. Small, brown, sunken flecks are seen first. As fruits mature, lesions become larger and darker. Affected areas can become deeply sunken and a gray to black growth of the fungus is sometimes seen growing in the lesion center. A recessed zone of healthy looking tissue will often surround the diseased area of the fruit.

Isolation:

Wash symptomatic tissue to remove superficial soil and other surface debris. Tissue samples can then be surfaced-sterilized in 0.5% aqueous sodium hypochlorite for 30 sec to 2 min. Sections should be rinsed in sterile, deionized water and blotted dry. Acidified potato dextrose agar and V-8 juice agar are suitable recovery media. Plates can be incubated in the dark at ca. 27 C. However, sporulation is greatly enhanced by exposure to a 12-hour photoperiod under fluorescent lights. An additional 7 days or so of incubation may be needed to allow for sufficient spore production and maturation.

Identification:

Historically, C. cassiicola has been called a number of different names, including Helminthosporium and Cercospora. Only an imperfect stage is known. The mycelium is light brown, branched, and septate. The conidiophores are 110-850 µm long, typically about 8-septate and have an iridescent appearance under a dissecting microscope. Conidia are born singly or in chains. Conidial morphology varies considerably with species of Corynespora. C. cassiicola spores are typically straight or slightly curved and pale olive to dark brown. The most diagnostic feature is the appearance of 4-20 "pseudosepta" in the conidia: these are septations that appear to divide the conidia into separate cells but do not actually extend all the way to the outer walls. Conidia should be examined carefully at 400X to determine if pseudosepta occur. Conidia are 40-220 µm long, 9-22 µm thick in the broadest part.

Pathogenicity:

Leaves of cucumber and tomato are best inoculated with a suspension of conidia in sterile water prepared by scraping the surface of 12-day old PDA cultures with a sterile glass slide. The suspension should be filtered through two layers of cheesecloth and applied to test plants with a hand atomizer. Fruit infection studies are more successful if the fruit are wounded prior to

application of inoculum. Use a fine grade of sand paper to gently abrade the fruit surface and immediately follow with a spray of conidial suspension as described for foliar infection.

Storage of pathogen:

The pathogen has been stored on PDA slants for several months and serially transferred to fresh slants. Storage at -70 C or lyophilization should work very well.

Selected crops in host range:

cucumber	cowpea	Aeschynanthus
tomato	papaya	cotton
soybean	snap bean	hibiscus
banana	azalea	impatiens

Geographic range:

Not completely known. Reports exist from:

North America
Caribbean Basin
Africa

Suggested taxonomic references:

Ellis, M. B. 1971. Dematiaceous Hyphomycetes. Commonw. Mycol. Inst., Kew, Surrey, England. 608 pp.

Wei, C. T. 1950. Notes on Corynespora. Commonw. Mycol. Inst. Myco. Pap. 34. 10 pp.

Suggested references:

Blasquez, C. H. 1969. Corynespora cassiicola on bananas. (Abstr.) Phytopathology 59:1347.

Blasquez, C. H. 1970. Varietal resistance to three cucumber foliar diseases in southwest Florida. Plant Dis. Rep. 54:52-55.

Blasquez, C. H. 1972. Target spot of tomato. Plant Dis. Rep. 56:243-245.

Blasquez, C. H. 1977. A blight of tomatoes caused by Corynespora cassiicola. Plant Dis. Rep. 61:1002-1006.

Jones, J. P., and Jones, J. B. 1984. Target spot of tomato: Epidemiology and control. Proc. Fla. State Hortic. Soc. 97:216-218.

Pohronezny, K., and Simone, G. W. 1988. Target spot of several vegetable crops. Ext. Plant Pathol. Fact Sheet No. PP-39. Univ. of. Coop. Ext. Serv., Gainesville, FL. 4 pp.

Volin, R. B., Pohronezny, K., and Simone, G. W. 1989 Severe spotting of fresh market tomato fruit incited by Corynespora cassiicola after storm-related injury. Plant Dis. 73:1018-1019.

Contributed by: K. Pohronezny

Disease: Crown gall, Gall, Root Knot, Root gall

Primary Economic Host: Apple, Cherry, Peach, Apricot, Plum, Pear, Qunice, Grape, Assorted Berries, Rose, Clematis, *Euonymus*, *Populus*, Almond, Olive, and Walnut

Pathogen: Agrobacterium tumefaciens

Symptoms and Signs:

A. tumefaciens infections trigger excessive proliferation of plant cells and cell enlargement resulting in a gall. Gals appear as small outgrowths within 2-4 weeks of infection when temperatures are at or above 20 C. Symptom development is greatly delayed at temperatures below 15 C, and infection is inhibited above 32 C. Latent infections may not show signs of gall until the third growing season, but this situation is observed rarely.

Anatomically, the gall consists of a sphere of parenchyma tissue containing disorganized vascular elements. The gall texture varies from soft and spongy to hard, depending upon the content of vascular tissues. The tissue is initially whitish, changing to tan and becoming brownish with age; aerial galls on herbaceous plants can appear green, depending upon chloroplast content. Small galls require careful diagnosis because they may be confused with excessive wood callus or with galls induced by nematodes or insects. The surface of galls on Mark rootstock can be covered with small bead-like protuberances of tissue.

Severe galling of young plants weakens the plant and results in stunting and occasional death. Severely galled rose plants become increasingly debilitated with time.

Roots: Galls are located most commonly along the main root, especially at the point where the main root was pruned off prior to transplanting. Root galls are typically rounded and vary in diameter from a few mm to 10 cm, with smaller galls located on lateral roots and the larger galls positioned along the main root.

Trunk/Stems: Galls that develop above ground are often close to the soil surface, rounded and smooth or fissured. Galls on woody perennial plants become more woody and fissured with age, sometimes reaching a diameter of 10 cm and girdling the stem.

Aboveground galls on roses and caneberries are typically globose. Occasionally galls appear at the site of pruning wounds. Galls on grape vines are usually elongate, erumpent ridges of tissue bursting through outer stem tissues, but galls near the soil can be globose. Erumpent gall tissue may cause berry canes to split and dry-out as moisture is lost. The split canes produce small, low quality fruit. Eventually, caneberry gall tissue turns brown and begins to deteriorate, especially those in or near the soil; new gall tissue typically develops the next spring from the margins of the old gall.

Leaves: Leaves of most plants are unaffected, but galls develop occasionally on petioles. Galls can be common on leaf surfaces of greenhouse grown chrysanthemums.

Laboratory Signs: Neither bacterial streaming nor ooze is associated with gall tissue, probably because of the relatively low number of Agrobacterium cells present in gall tissues.

Isolation:

Agrobacterium strains can be isolated from galls, vascular fluids (grape) and above- and below ground surfaces of symptomless plants. When isolating from galls, take samples of whitish

tissues. Populations of A. tumefaciens are low in necrotic, decomposing tumor tissues and may be overwhelmed by accompanying saprophytes.

Wash the gall surface in running tap water to remove soil and debris; trim away dead, darkened tissue. Washing is preferred to surface sterilization, thus reducing the risk of killing the target bacteria. If surface sterilization is desired, immerse the tissue in 1% sodium hypochlorite (or 20% household bleach) for 10-20 minutes then rinse the tissue sequentially with several changes of sterile water. Remove subsamples of tissue from two or three sites on the gall, dice these into about 2 x 2 mm pieces and suspend in sterile distilled water, covering the diced tissue with 1-2 mm layer of liquid. Vortex the sample preparation and let stand for 30 min. to allow bacteria to diffuse from the tissue. (Diffusion for up to 6 hrs may be necessary for old galls or when Agrobacterium populations are low in the tissue.) Charge an inoculating loop from the gall suspension and streak to a semi-selective medium (Fahy and Persley 1983; Lelliott and Stead 1987: Moore *et al.*1988). Alternatively, 0.1 ml of the suspension can be spread over the semi-selective medium, or diluted serially before spreading. A word of caution is appropriate on the use of selective media: Some strains grow poorly or not at all on these media. We have recently isolated strains of A. tumefaciens that don't grow on the selective medium; instead they require a medium high in salts (Canfield and Moore 1989). Both biovar 1 and 2 strains of A. tumefaciens can be isolated from galls of plants grown in the Pacific Northwest, but biovar 2 strains predominate.

Identification:

Colonies on potato dextrose agar are usually white or cream-colored, convex, glossy, with entire margins depending upon the kind of isolation medium used. Colony pigmentation can be white, tan, yellow, pinkish, light green, reddish-orange, or have a reddish center, but colony morphological features remain constant. To aid colony recognition, unknown isolates should be compared to known biovar strains that are streaked to the same medium at the time of isolation.

Methods have been published recently for development of strain-specific polyclonal antibodies (Bouzar and Moore 1987) and monoclonal antibodies (Bishop *et al.* 1989; and Elsman *et al.* 1987) for identification of Agrobacterium. DNA probes for identification of A. tumefaciens strains have also been used successfully (Burr *et al.*1990; Canfield *et al.* 1990).

The nomenclature for A. tumefaciens is confused. There are three biovars within the genus which are biochemically distinct groups. Although the biovar is a subspecies taxon, the groups equate taxonomically with three separate species. Biochemical tests for classification of strains to biovar status are found in Fahy and Persley (1983) and Moore *et al.* (1988). Current methods of classification to species is capricious because species taxonomy is based on whether the strain is pathogenic. Pathogenicity is encoded by genes carried on a large extrachromosomal piece of DNA referred to as the tumor-inducing (pTi) plasmid. If the pTi is lost, the strain is no longer pathogenic and would qualify to be called A. radiobacter, a nonpathogenic species. Conversely, transfer of a pTi to an A. radiobacter strain confers pathogenicity on the strain, and it would then qualify as an A. tumifaciens. Other taxonomic schemes have been suggested, but historical use of the current taxonomy and/or the cumbersome nature of the new suggestions have prevented them from succeeding.

Pathogenicity:

Pathogenic and non-pathogenic agrobacteria are usually isolated from a gall, and pathogenicity assays on tomato or other herbaceous hosts (Anderson and Moore 1979) or colony-hybridization (Burr *et al.* 1990; Canfield *et al.*1990) need to be performed to determine pathogenicity.

The isolation of nonpathogens is especially troublesome from apple galls because nearly all the strains isolated are nonpathogenic, thus making it difficult to fulfill Koch's postulates. These galls also lack opines (Moore *et al.* 1986). Continual isolation of nonpathogenic Agrobacterium from apple galls has led us to speculate that: (i) A. tumefaciens may infect apple then mutate to avirulence, (ii) the pathogen didn't survive in the gall, (iii) the pathogenicity assay for apple strains was inadequate, or (iv) the true causal agent was not isolated.

Storage of Organism:

a. Lyophilization: We have not used lyophilization for storage of agrobacteria, but it should work well. The method does require equipment expenditure and technical skill. It is essential to use a suitable suspending medium to protect the bacteria during the freeze-drying process (Fahy and Persley 1983).

b. Storage at -80 C: Agrobacterium strains are grown for 48 hr on MGY agar slants (10 g mannitol, 2 g glutamate, 1 g yeast extract, 0.5 g k KH_2PO_4, 0.2 g $MgSO_4.7H_2O$, 0.2 g NaCl, and 15 g agar made up to 1 liter with distilled water and adjusted to pH 7.1), and 2 loops full of bacterial growth suspended in 30% sterile glycerol. The suspension is placed in a chest freezer at -80 C for long term storage.

c. Sterile Water: Agrobacterium strains are grown for 48 hours on MGY agar slants, and 2 loops full of bacterial growth are suspended in sterile distilled water contained in small screw-capped vials (lids have teflon liner for good seal). Vials are stored at 4 C. Cultures of Agrobacterium have remained viable for 15+ years.

d. Agar Slants: Working cultures of Agrobacterium are maintained for 6 months on slants of PDA + 5% $CaCO_3$ in small screw-capped culture tubes. Tubes are stored at 4 C.

Reported Host Range:

Host range is strain dependent; some strains have a wide host range while others have a narrow host range (Anderson and Moore 1979; Loper and Kado 1979). The total host range listed for the species collectively is very wide (Decleene and DeLey 1986) and includes shrubs, ornamental flowers, fruit trees (temperate to tropical), tubers, vines, tobacco, annuals, members of the gymnosperms, etc. Bradbury (1986) lists 391 susceptible plant genera, many of which have multiple susceptible species within the genus. Crown gall disease has been observed on only a few of these species in their natural habitat. Conclusions about susceptibility of most of the hosts listed are based upon gall development following wounding and inoculation during scientific investigation.

Vector: Nematodes (Vrain and Copeman 1987), grubs and other chewing insects (Moore and Cooksey 1981) have been implicated as passive carriers of A. tumefaciens which subsequently infect feeding wounds.

Geographic Range:

The disease occurs world-wide.

Suggested Taxonomic Keys:

Fahy, P. C. and G. J. Persley. 1983. Plant Bacterial Diseases: A Diagnostic Guide, 393 pp. Academic Press, New York.

Moore, L. W., C. I. Kado and H. Bouzarr. 1988. Agrobacterium. pp.16-36. In Laboratory Guide for Identification of Plant Pathogenic Bacteria. N. W. Schaad, Ed. 2nd ed. Am. Phytopathol. Soc. Press. Minneapolis.

Suggested Literature:

Bradbury, J. F. 1986. Guide to plant pathogenic bacteria. C.A.B. International Press, Slough, Britain 332 pp.

Leiliott, R. A. and D. E. Stead. 1987. Methods for the diagnosis of bacterial diseases in plants. Methods in Plant Pathology. Palo Alto, Blackwell Scientific Publications, 216 pp.

Moore, L. W. and D. C. Cooksey. 1981. Biology of Agrobacterium tumefaciens: The Biology of Rhizobacteria, supplement to International Review of Cytology. Giles ed. Academic Press, New York.

Contributed by: L.W. Moore

Disease: **Rhizoctonia Damping-off, Rhizoctonia Crown and Root Rot**
Rhizoctonia Foliage Blight

Primary Economic Host: Beta vulgaris (sugarbeet)

Pathogen: Rhizoctonia solani (teleomorph: Thanatephorus cucumeris)

Symptoms and signs:

a) Damping-off: Both pre-emergence and post-emergence seedling disease occurs, but usually the post-emergence phase is the most damaging. Infection occurs below the soil line and moves up the hypocotyl with a definite margin between diseased and healthy tissue. R. solani anastomosis group 4 (AG-4) usually predominates in isolations from diseased seedlings and most isolates are highly virulent on beet seedlings. However, some isolates of AG-2-2 and Ag-1-1C (if AG-1-1C is present in the particular region) also can cause damaging post-emergence damping-off. Other R. solani AG and binucleate Rhizoctonia spp., which have been isolated from diseased seedlings in U.S.A., are reported to be much less virulent or avirulent.

b) Crown and root rot: Crown rot occurs on older plants, associated with the developing canopy. The disease occurs in elongated patches of diseased plants within affected fields. Disease patches tend to extend along rows farther than across rows. Diseased areas range in size from those involving 2-4 plants to areas of 6 meters or more in diameter. Crown rot symptoms begin with formation of black disease lesions on the outer (older) petioles in contact with soil. More petiole bases and the crowns become diseased, the plants wilt and foliar yellowing develops. The wilted leaves collapse, forming a flattened, chlorotic rosette on the soil. The leaves eventually die, dry and turn brown or black. Crowns and roots rot and become dead hulks. Less frequently invasion of roots occurs at the soil line on beets with raised crowns. Also invasion and rotting of roots may occur below the soil surface. Infection of roots often is accompanied by occurrence of large cracks or fissures in the beets. In some areas of U.S.A. sunken cankers or crater spots of limited decay develop rather than a general spreading rot. Under high moisture conditions hymenia of the teleomorph may develop on healthy tissue adjacent to disease lesions on the underside of affected petioles. Hymenia are appressed, white to beige colored, superficial, membranous to felty fungal growths, which can readily be removed with a dissecting needle for microscopic examination or culture. Basidiospores formed on hymenia may be disseminated to beet foliage and may cause foliage blight if conditions are favorable. In U.S.A. and Japan the primary AG causing crown and root rot disease is AG-2-2, although other AG such as AG-4, AG1-1C, and AG-5 can be isolated in some geographic areas.

c) Foliage blight: Two distinct types of foliage blight can occur. In the first type young heart leaves in the center of the leaf rosette exhibit marginal leaf burn and blackening, often being reduced to narrow deformed leaves or petiole stubs. This symptoms is associated with attack by R. solani AG-4. A few to possibly 5% of the plants within fields in Ohio will be affected depending on seasonal environmental conditions.

Under very moist conditions a second type of leaf blight symptom can develop, consisting of large, irregular, collapsed, water-soaked and blackened lesions on large leaf blades. As the blight progresses the lesions tend to dry out and disintegrate and the blighted leaves then appear ragged or shredded. In most instances, hymenia occur on the underside of leaves on healthy tissue adjacent to the leaf blade lesions. This second type of foliar blight can be caused by either AG-4 (as reported in Colorado) or AG-2-2 (as reported in Japan and Ohio). In the case of Ag-2-2, hymenia were first detected on the underside of petioles near the crown 10 days to 2 weeks before foliage blight was evident. In Japan, the obvious leaf blight phase is preceded by small (1 mm dia.) lesions initiated by basidiospores, followed by secondary chlorotic lesions with brown

edges. Under continued moist conditions the obvious, large blackened lesions previously described rapidly develop. Secondary spread by basidiospores occurs as long as conditions remain suitable.

Isolation:

For seedlings and pieces of diseased petioles and beets, wash them thoroughly on an immersed sieve under running cool tap water for at least 10-15 min. Initially adding several drops of Tween® will improve washing. Float seedlings on water in petri plates and incubate or blot dry on paper toweling and plate on 2% water agar. Fifty µg/ml each of streptomycin sulfate and chloramphenicol can be added for bacterial control. After 24 hr at room temperature examine plates for growth of R. solani. Incubation may be extended to 48 hr, but contaminating bacteria, fungi and nematodes may cause problems with extended incubation. Transfer colonies from isolation plates to 2% water agar plates, one colony transfer (centered) per plate. With a sterile spatula cut around the agar and flip the agar layer over, trapping the inoculum piece in a air bubble under the agar. Cut the agar layer approximately 1 cm from the plate edge entirely around the layer. The agar layer will now seal around the isolation inoculum piece. After sufficient growth, carefully (do not completely penetrate the agar layer) hyphal tip the culture and transfer to PDA or other nutrient media. The isolate should now be free of bacteria (repeat agar flip if not) and can be maintained on PDA slants, sterile barley grain cultures, soil-bran cultures dried and kept frozen at -10°C, cryogenic (-70°C) or liquid nitrogen storage.

Some prefer use of selective media (several have been described) to water agar for isolation of R. solani, but such media may not be equally suitable for all AG types and will exclude other possible pathogens which may be present.

Identification:

According to Parmeter, criteria for distinguishing R. solani include the presence of a prominent doliform septal pore apparatus, multinucleate vegetative cells, branching near the distal septum of cells in young vegetative hyphae, constriction of the branch and formation of a septum in the branch near the point of origin, some shade of brown in color of colonies. Other characteristics usually present include moniliod (barrel) cells, undifferentiated sclerotia, hyphae greater than 5 µm in diameter, rapid growth and pathogenicity. Characteristics never present are clamp connections, conidia, differentiated sclerotia, rhizomorphs, pigments other than brown and any perfect state (teleomorph) other than T. cucumeris.

Pathogenicity:

For seedling assays grow the isolates on 2% water agar plates until the colonies reach the plate edge. Partially fill a 10 cm diam. pot or similar sized container with potting mix soil. Place the intact colonized water agar layer on and covering the soil surface. Add about an inch of soil and place seeds on its surface. Cover the seeds. After 2 to 3 weeks record surviving seedlings.

For older plants used dried colonized barley seed inoculum to infest (3-6 grains/pot) single sugarbeet plants (6-8 wk-old) in 15 cm pots for greenhouse tests or sidedress rows of sugarbeets in the field prior to layby cultivation, both sides of row with whole grain colonized barley inoculum at ca. 45 kg/ha.

To produce inoculum add 300 ml water to 500 ml barley grain, stir, let stand overnight. Autoclave at 121°C for 1 hr. Transfer several pieces of agar colonies of appropriate isolates to the sterile barley. Incubate 2 wk, air-dry, screen to separate grains. Inoculum viability will remain high (stored in paper bags at room temperature) for at least 2-3 months.

Reported host range:

R. solani AG-4 causes seedling diseases of many hosts, having an extremely wide host range. The R. solani AG-2-2 host range is more restricted but includes several crops including:

Beta vulgaris	sugarbeet
Arachis hypogaea	peanut
Glycine max	soybean
Nicotiana tabacum	tobacco
Zea mays	corn

and weed hosts, including:

Amaranthus retroflexus	rough pigweed
Chenopodium album	lambsquarters

(Evidence is accumulating that the division of AG-2-2 into AG-2-2 III B and AG-2-2 IV made by Japanese researchers may reflect host range differences of importance.)

Geographic range: Worldwide distribution

Suggested references:

Butler, E. E. 1980. A method of long-time culture storage of Rhizoctonia solani. Phytopathology 70:820-821.

Gaskill, J. O. 1968. Breeding for Rhizoctonia resistance in sugarbeet. J. Am. Soc. Sugar Beet Technol. 15:107-119.

Herr, L. J. 1986. Characteristics of hymenial isolates of Thanatephorus cucumeris on sugar beets in Ohio. Plant Disease 66:246-249.

Kotila, J. E. 1947. Rhizoctonia foliage blight of sugar beets. J. Agric. Res. 74:476-780.

Ogoshi, A. 1985. Anastomosis and intraspecific groups of Rhizoctonia solani and binucleate Rhizoctonia. Fitopatologis Brasileira 10:371-390.

Ogoshi, A. 1987. Ecology and pathogenicity of anastomosis and intraspecific groups of Rhizoctonia solani KHhn. Annu. Rev. Phytopathol. 25:125-143.

Parmeter, J. R., Jr. 1970. Rhizoctonia solani: Biology and Pathology. University of California Press, Berkeley, Los Angeles, and London. 255 pp.

Contributed by: Leonard J. Herr

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Fla. Dept. Agric. & Consumer Serv.
Division of Plant Industry

Botryosphaeria Cankers and Dieback

T. J. Proffer[1]

Four or more species of Botryosphaeria have been associated with cankers (Fig. 1) and/or dieback on a wide range of trees and shrubs in Florida (1,4). These fungi can also cause leaf spots and fruit rots which will not be addressed in this circular. This cosmopolitan group of generally nonspecialized pathogens has an extremely wide host range. Due to its unique location, Florida hosts both the more common temperate and tropical species, with two or more species often being reported from the same host. Botryosphaeria is commonly recovered from stem cankers and terminal branch segments exhibiting dieback on plants which have been injured by other biotic or abiotic factors (3,5). Wounds caused by mechanical injury (pruning wounds, equipment wounds, etc.), environmental injury (freezing or high temperatures), insect injuries (borers, oviposit wounds, etc.), and chemical injuries (herbicide injury to thin-barked species) are common infection sites (3,5). Plants which are stressed by cultural conditions are also more commonly affected (poor site factors, moisture stress, nutritional problems, transplanting, etc.) (3,5). Based on their widespread occurrence and opportunistic nature (coupled with a ready resource of stressed plants), Botryosphaeria spp. rank high on the list of important fungal pathogens of woody plants in Florida.

PATHOGENS: Botryosphaeria cankers and dieback have been attributed to a number of Botryosphaeria species. The taxonomic distinctions between species are not universally accepted and problems with nomenclatural precedence are frequently noted in the literature (2,3,5,6). Species of Botryosphaeria (Class: Loculoascomycetes, Order: Dothideales) are most frequently encountered in their conidial or asexual stage. Conidia are produced in pycnidia in all cases, however, there is a good deal of variation (both within and between species) in the organization of the pycnidia (single or stromatic groups) and in the color, shape, and septation of the conidia (Fig. 2). As a result of this variation, anamorphs of Botryosphaeria are found in the form genera: Botryodiplodia, Diplodia, Dothiorella, Fusicoccum, Macrophoma, and Sphaeropsis (3,4,5,6). The nomenclatural status of these various form genera are also not totally agreed upon by all authors.

Fig. 1. Botryosphaeria canker on redbud (Cercis canadensis L.).

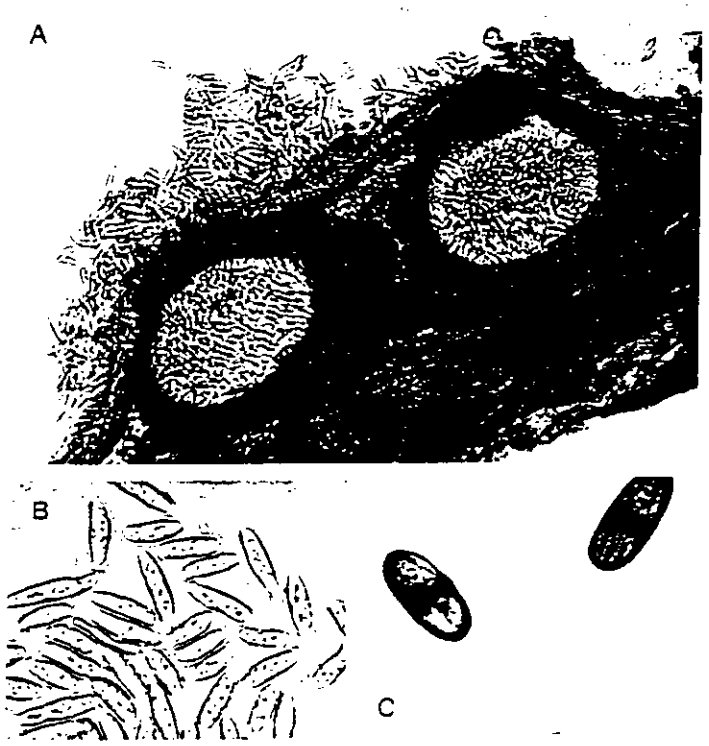

Fig. 2. A) Pycnidia, B) conidia of Botryosphaeria ribis; C) conidia of B. rhodina.

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The two most frequently encountered species of Botryosphaeria in Florida are B. rhodina (Cooke) Arx and B. ribis Gross. & Duggar (1). Botryosphaeria rhodina is a common tropical to subtropical species which has been reported on at least 280 genera of plant hosts worldwide (5). The asexual stage of B. rhodina is distinct in that the mature conidia are oval (18-30 X 10-18µm), dark, one septate, and longitudinally striate at maturity (3,5). The conidia, however, do not mature until after they have been discharged from the pycnidium. As discharged in the conidial cirrus or within the pycnidial centrum, the conidia are hyaline, aseptate, and densely granulate. Botryosphaeria ribis is more temperate in its distribution (5) and the nomenclatural status of the species is more tentative. It is often considered as a synonym of B. dothidea (Moug.:Fr) Ces. & De Not. and B. berengeriana De Not. (2,3,6). This taxonomic question has yet to be satisfactorily resolved, the reader is reminded, therefore, that the names noted here may overlap in the literature. The asexual stage of B. ribis displays much variability. Two types of conidia are often observed, macroconidia which are generally oblong and slightly fuscoid [17-25 X 5-7 µm (5,6), 16-31 X 4-8 µm (3)] and microconidia [2-3 X 1 µm (3,5,6)]. The conidia are hyaline and nonseptate. A good deal of variability is noted among conidia, even from the same pycnidium. Other species of Botryosphaeria reported in Florida (1) include: B. corticis (Demaree & Wilcox) Arx & Muller, B. disrupta (Berk. & Curt.) Arx & Muller, and B. quercuum (Schwein.) Sacc.

DISEASE SYNDROME: Species of Botryosphaeria are considered facultative parasites. They survive as saprophytes on dead plant tissues and should be considered as being ubiquitous in the environment. Even in healthy plants there are dead tissues which may be colonized. If a plant is subsequently stressed or injured the fungus may invade healthy tissues.

Symptoms caused by Botryosphaeria canker vary with the species of host infected and the degree of predisposing stresses. As noted previously, wounds, either natural or inflicted, are often the site of canker initiation. Cankers may vary from small lesions surrounding the wound, to sunken elliptical lesions delimited by a callus ridge, to spreading sunken lesions which are not contained by callus. Generally the bark over the canker will be discolored, this is most readily seen on thin barked species. Spreading diffuse Botryosphaeria cankers are often narrow and elongate, the fungus moving quickly along the length of the stem. Beneath the bark, the cortical/cambial tissues of cankered stems are brown as compared to the white or pale green healthy tissues. Xylem tissues can also be discolored by Botryosphaeria canker and a brown wedge-shaped section may be observed in a stem cross section.

Fruiting bodies of the fungus form on dead tissues and may produce viable spores over a period of years (3,5). These fruiting bodies are generally small (0.5-3.0 mm) (6) and on the bark surface appear as small raised black spots. The asexual spores (conidia) are most frequently implicated in the spread of disease. The conidia are exuded from the pycnidia when the tissues are moistened and are spread primarily via rainsplash.

CONTROL: Growers should try to minimize wounding and maintain vigorous plants. Pruning should be done in dry weather with tools which are frequently sterilized. If Botryosphaeria canker is present, the affected tissues should be aseptically pruned out. The infested debris should be destroyed, the fungus can produce spores on these pruned out branches for long periods of time (3). A protectant fungicide can be applied to cut surfaces. Effective chemical controls are lacking, good sanitation and good cultural practices are the best methods to prevent Botryosphaeria canker.

SURVEY AND DETECTION: The initial visible symptom of Botryosphaeria canker is often a wilting and flagging of leaves on branches distal to the canker. A closer examination of the stems will generally reveal a slightly sunken and often discolored area on the bark. A pocket knife should be used to cut away the bark to reveal the cortical and cambial tissues (do not cut all the way to the xylem), a brown discoloration indicates a pathogen may be at work.

LITERATURE CITED

1. Alfieri, S. A. Jr., K. R. Langdon, C. Wehlburg, and J. W. Kimbrough. 1984. Index of Plant Diseases in Florida. FL Dept. of Agric. and Consumer Serv., Div. of Plant Industry, Bull. No. 11, 389p.

2. Arx, J. A. von and E. Muller. 1954. Die Gattungen der Amerosporen Pyrenomyceten. Beitr. Kryptogamenfl. Schweiz 11:1-434

3. Brown, E. A., II and K. O. Britton. 1986. Botryosphaeria Diseases of Apple and Peach in the Southeastern United States. Plant Disease 70:480-484.

4. Farr, D. F., G. F. Bills, G. P. Chamuris, and A. Y. Rossman. 1989. Fungi on Plants and Plant Products in the United States. APS Press. St. Paul, Minn. 1252p.

5. Sinclair, W. A., H. H. Lyon, and W. T. Johnson. 1987. Diseases of Trees and Shrubs. Cornell University Press. Ithaca, N.Y. 574p.

6. Sivanesan, A. 1984. The Bitunicate Ascomycetes and their anamorphs. J. Cramer. 701p.

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Fla. Dept. Agric. & Consumer Ser.
Division of Plant Industry

PHYLLOSTICTA LEAF SPOT OF CHINESE WISTERIA

S. A. ALFIERI, JR.[1]

Chinese wisteria, *Wisteria sinensis* (Sims) Sweet, is a woody, twining vine, native to China, and is commonly cultivated as an ornamental for its foliage and striking, drooping racemes of white, pink or lavender sweetpea-like flowers. It is hardy in the North, but most common in the southeastern states where it has become naturalized.

One of the more striking foliage diseases of Chinese wisteria is caused by *Phyllosticta wistariae* Sacc. (2,3), which is considered more important in the South. This disease has been reported from Florida (1), Massachusetts, Missouri, New Jersey and Texas (2,4).

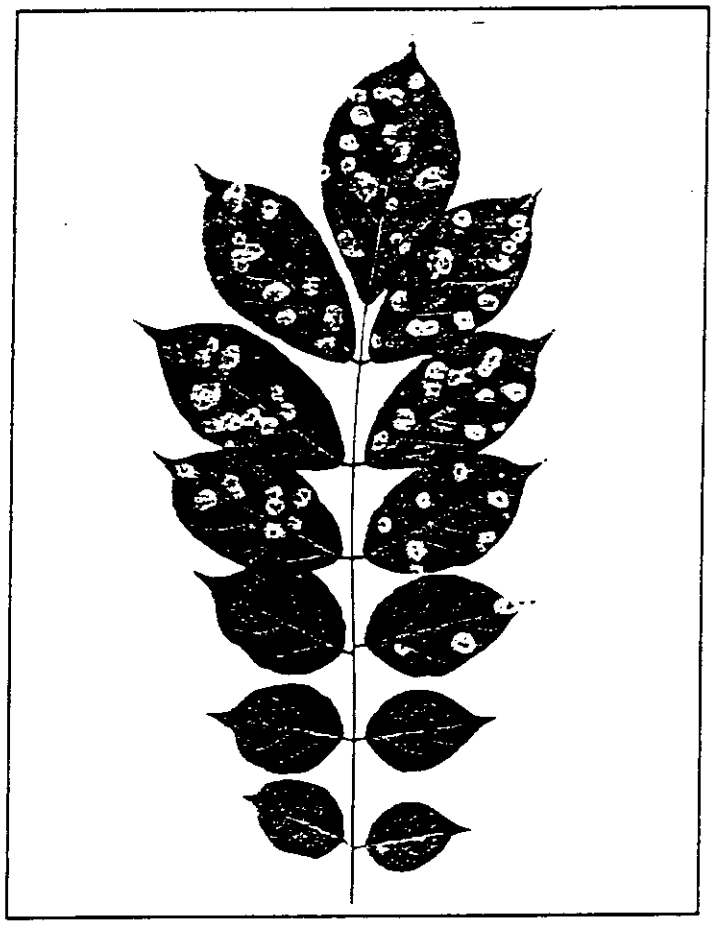

Figure 1. Phyllosticta leaf spot of Chinese wisteria. DPI File #702540.

SYMPTOMS: Leaf spots caused by *Phyllosticta wistariae* are very distinct in appearance. They are usually numerous, subcircular to irregular, yellow lesions with a somewhat brown-speckled center, rarely coalescing, and up to 6 mm in diameter (Fig. 1).

CONTROL: This disease does not appear to occur frequently at high incidences; however, if leaf-spotting does occur at seriously high levels, consulting with the local county extension director for control measures is recommended.

SURVEY AND DETECTION: The appearance of usually numerous, yellow leaf spots with a brown speckled center is evidence of this disease.

LITERATURE CITED

1. Alfieri, S. A., Jr., Langdon, K. R., Wehlburg, C. and Kimbrough, J. W. 1984. Index of Plant Diseases in Florida. Fla. Dept. Agric. & Consumer Services. Div. Plant Industry. Bull. 11. 389 p.

2. Farr, D. F., Bills, G. F., Chamuris, G. P. and Rossman, Amy Y. 1989. Fungi on Plants and Plant Products in the United States. APS Press, St. Paul, Minn. 1252 p.

3. Saccardo, R. A. 1884. Sphaerioideae to Melanconiaceae. Sylloge Fungorum 3:11.

4. Westcott, Cynthia. 1979. Westcott's Plant Disease Handbook (Revised by R. K. Horst). Van Nostrand Reinhold Co., New York. 803 p.

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Division of Plant Industry

BACTERIAL BLIGHT OF SWEDISH IVY CAUSED BY *PSEUDOMONAS CICHORII*

J. W. Miller[1]

Swedish ivy, *Plectranthus australis* R. Br., is a native of Australia belonging to the mint family, Labiateae. It is a creeping herb used for hanging baskets (1). Other species of *Plectranthus*, including *P. oertendahlii* T.C.E. Fries, are grown on occasion. A serious disease was observed in nurseries from scattered locations in Florida which resulted in leaf spotting and blighting. *Pseudomonas cichorii* (Swingle) Stapp was consistently isolated from diseased plants and was confirmed as cause of the problem.

Figure 1. *Pseudomonas cichorii* on *Plectranthus australis* showing dark gray leaf spots and blighted leaves.

SYMPTOMS: The disease begins as dark gray, rather dry spots which occur either within or along the margin of the leaves (Fig. 1). The lesions may enlarge and lead to blighting of the plant, with defoliation of older leaves (2). Artificial inoculations showed susceptibility of other members of the family Labiateae including *P. oertendahlii*, *Coleus* X *hybridus* Voss. 'Golden Bedder', and *Rosmarinus officinalis* L. (rosemary), but not *Ajuga reptans* L. (bugleweed).

CONTROL: It is best to rogue diseased plants or leaves and keep foliage as dry as possible to retard disease spread. Take cuttings only from healthy plants. No chemicals are registered for control of this disease on Swedish ivy.

SURVEY AND DETECTION: Look for dark gray, dry spots on leaf blades or margins which may lead to blighting and defoliation.

LITERATURE CITED

1. Graf, A. B. 1974. Exotica Third. Roehrs Co., Rutherford, N. J. 1833 p.

2. Miller, J. W. and Virgona, R. J. 1978. Bacterial blight of Swedish ivy caused by *Pseudomonas cichorii*. (Abstr.) Phytopathol. News 12:135.

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Fla. Dept. Agric. & Consumer Serv.
Division of Plant Industry

SEPTORIA LEAF SPOT OF BLUEBERRY

S. A. Alfieri, Jr.[1]

Blueberry (*Vaccinium spp.*) production is expanding into a significant agricultural industry in Florida. Presently constituting approximately 2,100 acres, it is a relatively young, growing, and vibrant commercial industry. Not much information is known or available on diseases affecting blueberries grown under Florida conditions (1) as compared to other well known extensive production areas (4). However, there are a number of serious disease problems that affect blueberries in other important blueberry-growing areas of the United States (3,4,7).

Among the leaf-spotting organisms of importance in blueberry production is *Septoria albopunctata* Cooke (2,3,6), the cause of eyespot disease. On some varieties, *S. albopunctata* could have a very debilitating effect on plant vigor, such as shown on the foliage of the highbush species *Vaccinium corymbosum* L. in Fig. 1.

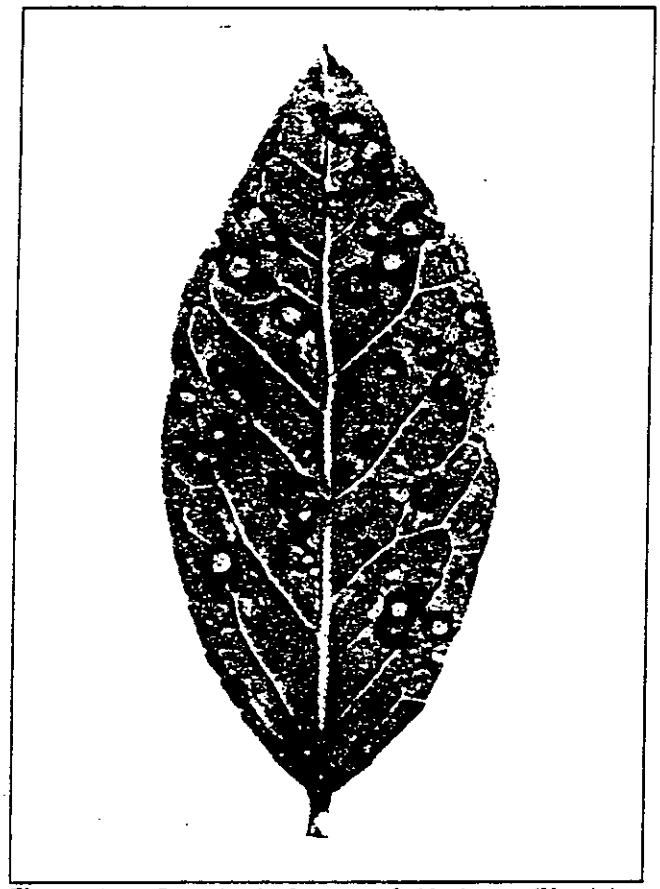

Figure 1. Septoria leaf spot of blueberry (*Vaccinium corymbosum* L.). DPI File #702346-1.

SYMPTOMS: Septoria leaf spot on blueberry is denoted by numerous circular to subcircular, light to medium brown lesions with a broad, purplish brown margin, up to 3 mm in diameter, and often coalescing upon enlargement (Fig. 1).

CONTROL: Presently, there does not appear any evidence that this leaf spot or other leaf-spotting diseases cause reductions in yield in Florida, thus the use of fungicides does not appear warranted. However, cultural control such as collecting or mulching over infected, fallen leaves would help reduce the fungus inoculum during the following growing season (5). In the nursery production of blueberry plants, chemical control of leaf-spotting diseases may be desirable or necessary. In such cases, consulting a County Extension Director or a current Plant Disease Control Guide (5) is advisable.

SURVEY AND DETECTION: The appearance of usually numerous brown, often coalescing, leaf spots with a broad purplish brown margin, approximately 3 mm in diameter, is evidence of this disease.

LITERATURE CITED

1. Alfieri, S. A., Jr., Langdon, K. R., Wehlburg, C. and Kimbrough, J. W. 1984. Index of Plant Diseases in Florida. Fla. Dept. Agric. & Consumer Services. Div. Plant Industry. Bull. 11. 389 p.

2. Cooke, M. C. 1884. New American fungi. Grevillea 12:22-83.

3. Demaree, J. B., and Wilcox, Marguerite S. 1947. Fungi pathogenic to blueberries in the eastern United States. Phytopathology 37:487-505.

[1]Plant Pathologist, Emeritus, FDACS, Division of Plant Industry, P. O. Box 1269, Gainesville, FL 32602

4. Farr, D. F., Bills, G. F., Chamuris, G. P. and Rossman, Amy Y. 1989. Fungi on Plants and Plant Products in the United States. APS Press, St. Paul, Minn. 1252 p.

5. Kucharek, T., Simone, G. 1989. Plant Disease Control Guide (1989). Inst. Food and Agric. Sci., Fla. Coop. Ext. Serv., Univ. Fla.

6. Saccardo, P. A. 1884. Sphaerioideae to Melanconiaceae. Sylloge Fungorum 3:493.

7. Taylor, J. 1958. Stem canker and related blueberry diseases. North Carolina Agric. Exp. Sta. Tech. Bull. 132. 24 p.

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BACTERIAL LEAF SPOT OF BOUGAINVILLEA

S. E. Walker[1]

Bougainvillea Comm. ex Juss is a member of the Nyctaginaceae family. A native of South America with large colorful floral bracts, it is among the most spectacular of flowering vines and shrubs used in landscape designs. The floral bracts are arranged in triplets surrounding the small, tubular white flowers (1, 2). Various bract shades of red, purple, pink and orange, as well as white and some bicolored varieties are available. The climbing vines are anchored with sharp thorns rather than coiled tendrils. Leaves are ovoid to elliptic-lanceolate, may be variegated, and vary considerably in size between varieties. Although there are many species of *Bougainvillea*, most plants in the ornamental trade today are hybrids of three horticulturally important species: *B. glabra*, *B. spectabilis*, and *B. peruviana*. In the tropics and subtropics bougainvilleas are used in borders, on fences, and as espaliers (2). Bougainvillea has recently gained popularity in colder climates where it is used as hanging baskets and pot plants for patio gardens during the summer months. Accompanying the recent increase in bougainvillea production in Florida, a bacterial leaf spot disease has emerged on the popular variety 'Barbara Karst'. The causal agent, *Pseudomonas andropogonis* (E. F. Sm.) Stapp. has since been recovered from at least 12 different varieties at several locations throughout Florida and the southeastern United States.

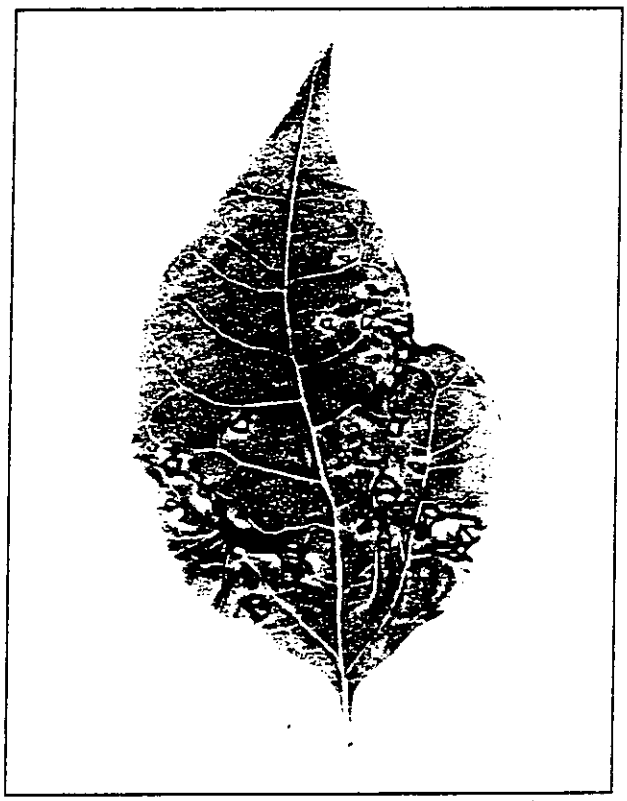

Figure 1. Tan colored leaf spots with reddish-brown borders on bougainvillea. Note the puckered, distorted leaf shape caused by early infection of expanding leaves.

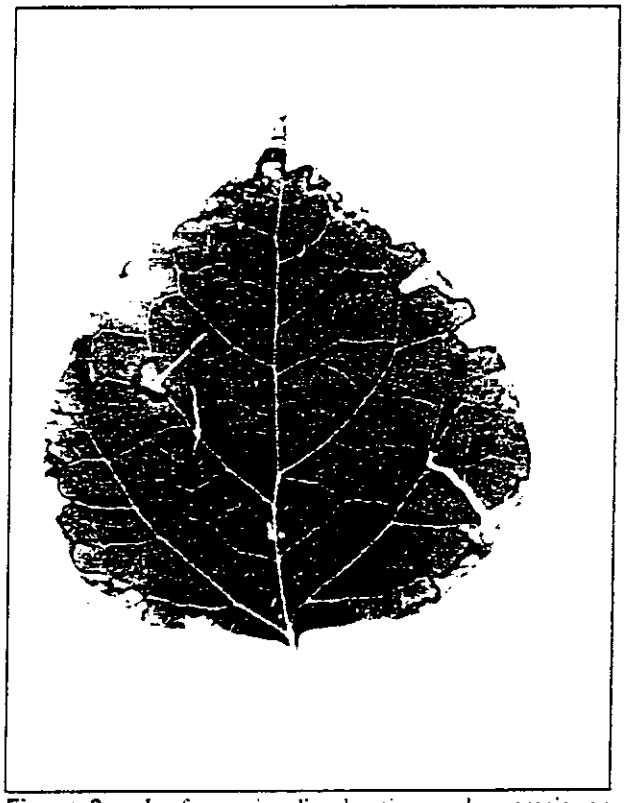

Figure 2. Leaf margin discoloration and necrosis on bougainvillea.

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SYMPTOMS AND DISEASE DEVELOPMENT: The early symptoms are small reddish-brown leaf spots which usually occur on younger foliage. These enlarge into circular or irregular dark necrotic spots. When environmental conditions are drier and less favorable leaf spots develop slowly. Lesions have a tan center surrounded by a dark red-brown margin, and are sometimes bordered by a chlorotic halo (Fig. 1) (5, 6). Necrotic leaf margins, resembling excess soil soluble salts damage, are so common that reddish-brown marginal necrosis is often the main symptom present (Fig. 2). In time, leaf edges may become ragged as the necrotic tissue turns dry and papery. Under conditions of high rainfall or relative humidity the lesions develop quickly and are often black and vein delimited. Infection of developing leaves and bracts results in puckered, distorted growth (5, 6). Nursery plants grown under frequent overhead irrigation may exhibit this type of disease development. Defoliation will occur when leaf spotting, blighting or marginal necrosis becomes severe (5, 6).

PATHOGEN: *P. andropogonis* is worldwide in distribution and causes an array of leaf spots, necrotic stripes, and streaks on a variety of host plants. Some important agronomic hosts are corn, sorghum, velvet bean, chick pea, and clover. The pathogen also infects many ornamentals including carnations, tulips, statice, ruscus, and orchids (3).

Some isolates of *P. andropogonis* are host specific while others have a broad host spectrum. Inoculation of bougainvillea with isolates from diverse hosts and locations show that bougainvillea is susceptible to some but not all isolates (5).

CONTROL: Maintaining dry foliage is the primary control measure. The pathogen is spread by water splash, handling, and propagation from diseased stock plants (6). Starting with clean stock plants and avoidance of overhead irrigation will minimize chances of disease outbreaks. Good sanitation and removal of infected leaves and/or plants from the growing area reduces the risk of infecting healthy plants. In frost-free climates where bougainvillea is perennial, disease incidence drops during cool and/or dry weather (6).

SURVEY AND DETECTION: The most common symptoms of this disease are red-brown leaf margin discoloration and/or necrosis. Leaf spots are circular to irregularly shaped with tan centers and reddish-brown borders. Small leaf spots would be reddish-brown to black in color. Angular, black necrotic leaf lesions with puckered, distorted growth are also characteristic of the disease. Infected bracts show tan or black lesions with distorted growth (5, 6). *Note for clinical diagnosis:* Microscopic observation shows profuse bacterial streaming from the edges of leaf spots. Bacterial growth on nutrient agar plates will not appear until the second day and is strongly inhibited in the presence of other bacteria. Colonies are butyrous becoming viscid (4) and the bacteria quickly die out when kept in culture for very long. *P. andropogonis* is a non-fluorescent pseudomonad. Hypersensitivity can be demonstrated using fresh isolates on tomato and sometimes tobacco, but this ability is lost quickly in older cultures. For biochemical tests refer to Lelliot and Stead (4).

LITERATURE CITED

1. Baily, L. H. 1976. Hortus Third. Macmillan Publishing Co. Inc., New York. p. 174.

2. Everett, T. H. (ed) 1981. The New York Botanical Garden Illustrated Encyclopedia of Horticulture. Vol. 2. Garland Publishing, Inc., New York. p. 472-474.

3. Fahy, P. C. and Persley, G. J. (eds) 1983. Plant Bacterial Diseases. Academic Press, New York. p. 113-117.

4. Lelliot, R. A. and Stead, D. E. 1987. Methods for the Diagnosis of Bacterial Diseases of Plants. Blackwell Scientific Publications, Oxford. p. 63-64.

5. Moffett, M. L., Hayward, A. C., and Fahy, P. C. 1986. Five new hosts of *Pseudomonas andropogonis* occurring in eastern Australia: host range and characterization of isolates. Plant Pathology 35:34-43.

6. Rothwell, A. and Hayward, A. C. 1964. A bacterial disease of bougainvillea. Rhodesian Journal of Agricultural Research 2:97-99.

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