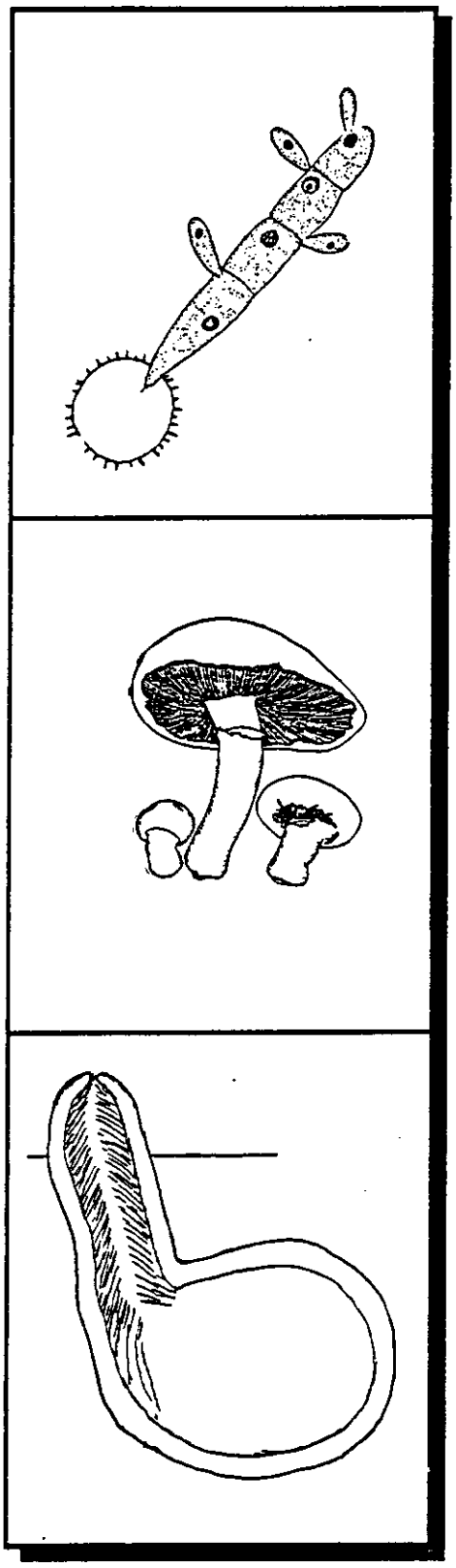

June, 1992
Volume XIII, Number 2

PLANT DIAGNOSTICS QUARTERLY

Features

University Plant Disease and Soil Testing Labs

On the cover: Fungi associated with turf:
Top = production of sporidia in *Ustaligo*
Middle = *Agaricus*, a common "fairy ring" mushroom
Bottom = a mature perithecium, immersed except for the tip of the beak, of *Gaeumannoymces graminis*

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FROM THE EDITOR

Hello again,

It's that time of year, so I'll be brief.
The proposed agenda for the Diagnostics Committee is within.
The response to my request for a new editor for *PDQ* was truly uninspiring.
The weather here has just turned decent.
Hope to see you in Portland.

Your Faithful Editor,

Melodie Putnam

REGIONAL REPORTS

Northwest Region
Colette Beaupré

The spring diagnostic season in Colorado has been extremely busy in terms of the number of samples handled (the samples this year are double what they were last year at this time). The season, however, has been very uneventful in terms of the plant drought stricken turf and the associated diseases of Kentucky bluegrass. Trees and shrubs around the region are loaded with Cytospora canker as a result of two previous summer's worth of hail damage and accompanying adverse Colorado growing conditions. Many deciduous trees and shrubs failed to leaf out as a result of the freeze we experienced in October 1991. Some of the more interesting plant diseases diagnosed include: sharp eye spot of wheat, tomato spotted wilt virus on various greenhouse crops, Botrytis stem rot of peony, and anthracnose on strawberry runners. Those of us who work in the Denver metro area continually pray for rain so that our summer diagnostic season will be full of interesting diseases instead of the usual abiotic problems we encounter in the arid West. (Laura Pottorff)

In the past few months the Oregon State University Plant Clinic has found stem nematode in a few central Oregon alfalfa fields. Much of the wheat in eastern Oregon has shown yellowing due to sulfur deficiency. We have also observed a lot of Cephalosporium stripe, which is uncommon for the area. To the west of the Cascades, there is a high level of rust and scald in many grass seed fields. This is earlier than usual, due to overwintering inoculum. Downy mildew has been found in many crops.

It is a brown spot and shot hole spring for stone fruits. Lophodermella needlecast is active on pines. We have seen Pseudomonas leaf spot and Thielaviopsis root rot on container-grown magnolia. Aspens have been troubled with Venturia shoot blight. Purple blotch occurred on blackberries, even though we did not have a winter to speak of. An impatiens strain of tomato spotted wilt virus (TSWV) has been found in English daisy, geranium, New Guinea impatiens, double impatiens, peppermint, and verbena. A lettuce strain of TSWV was found in double impatiens. Geraniums have been plagued with black leg, leaf spot, stem rot and Verticillium wilt. Ramularia leaf spot has been active on sugar beet and parsnip. (Stacey Fisher)

The Utah State University Plant Disease Clinic has had about 35 samples so far this year. Highlights include Fusarium dry rot in stored potatoes, pink and gray snow molds in wheat, Botrytis rhizome rot in iris, blackleg of geranium, tomato powdery mildew (sample was from Montana), and a very strange growth of yeast on turf!

We are concerned especially about an outbreak of virus, probably wheat streak mosaic virus, in wheat; but have not positively identified it yet. The amount of acreage affected seems to be limited, however. Our biggest worry now is drought. We had minimal rain in March and April; and phenological events like fruit tree bloom, codling moth flights, and powdery mildew on sour cherry are about three weeks early.

Anyone with experience to share on controlling Botrytis rhizome rot of iris, culturally or chemically, please contact me. (Karen M. Flint)

Rhododendron and *Prunus* samples have been the predominant hosts received into the Washington State University Puyallup Plant Clinic. Damage at the crown by root weevils and Phytophthora root rots have resulted in rhododendron death. A range of problems have been observed on *Prunus*, including brown rot infections, leafroller damage, and shot hole symptoms. High inoculum levels of *Monilinia* sp. and favorable environmental conditions have led to a significant number of brown rot twig and leaf infections from blossom blights. Shot hole symptoms on *Prunus* have resulted from bacterial, viral, physiological, and fungal infections. *Pseudomonas* infections in young leaf and shoot growth damaged by frost injury during April has been diagnosed on a range of hosts.

Master Gardener volunteers have been recruited to work in the WSU Puyallup Plant Clinic from June through August 1992. They will be assisting walk-in clients with diagnostic request forms, bulletin purchases, and diagnosis of common problems. The clinic volunteers will have the option to attend weekly diagnostic training sessions. We anticipate a busy summer season and look forward to having their assistance in the plant clinic. (Carrie R. Foss)

Our wimpy winter (at WSU - Prosser) turned into a brutal spring as April temperatures plummeted during peak cherry bloom, and the beginning of apple bloom and grape emergence. Growers had to finish pruning and applying cover sprays through the lingering haze from the previous night's frost protection. A temperature inversion added to the poor air quality for the rest of us, grouchy from listening to 7-9 hours of wind machines for many consecutive nights. Damage assessments vary, with the soft stone fruits hardest hit. Bacterial canker and *Pseudomonas* blast continue. One fire blight infection period has occurred.

Conifers and ornamentals are also showing the effects of the erratic spring. Hot weather prior to the initiation of the irrigation season in many areas has compounded the existing drought conditions and last fall's cold injury. It also encouraged weekend gardeners to ask questions about all those problems they ignored last year. Recent rains have resulted in extensive peach leaf curl, sycamore anthracnose, apple and pinpoint (*Venturia pini)* scab, powdery mildew on roses and turf, and shot hole blight (*Wilsonomyces carpophilus*).

Reduced potato acreage is in the ground and seasonal vegetable planting is underway. Botrytis blight and white rot (*Sclerotinia cepivorum*) are plaguing the Walla Walla onions (Do I hear a cheer in Vidalia and Texas?).

So much for the "green" winter wheat reported last time. To quote Russ Karow (OSU Agronomist), "A spot is a spot, but a treatable disease it may be not". It looks like Septoria and/or tan leaf spot with a liberal dose of micronutrient deficiency and a dash of barley yellow dwarf virus. It's the "no name" disease currently called physiological leaf spot. Etiology remains elusive but the acreage is extensive. It certainly provided days of coffee chatter for the wheat growers and headaches for the field men and us. BYDV and dryland root/foot rot (*Bipolaris* and *Fusarium*) are also occurring in dryland fields.

An unidentified virus destroyed a greenhouse crop of *Oxalis* prior to the "wearing of the green" (ouch). Leaves displayed mottle and ringspots before chlorosis and death. Successful transfers have been made to beans, tobacco, *Chenopodium quinoa*, and other indicator plants. (Ellen Bentley)

The University of Wyoming Plant Disease Clinic has responded to 50 samples since early March. Prevalent were drought-related problems of evergreens and bluegrass lawns.

We are finalizing revision of UW Clinic Form, so that it will be more "user-friendly". Also, the University is requiring that we charge for clinic services beginning July 1, 1992. We are currently awaiting approval for both the form and the fee schedule.

We are now using LOTUS 1-2-3 for the Clinic's Electronic record keeping to establish a working data base for Wyoming plant diseases. (Collette M-S Beaupré)

Southwest Region

Steven Koike

In Oklahoma, substantial rains in late May and into June have created conditions favorable for disease development. As a result, sooty mold (*Alternaria*) on wheat heads has been common, as has crown rot (*Bipolaris*). Black rot of grape, cedar-apple rust of apple, leaf scorch of strawberry, and bacterial leaf spot of peach are some of the prevalent diseases on fruits. Commonly reported diseases of ornamental plants include the following: bleeding necrosis (*Botryosphaeria*) on sweet gum; fire blight on crabapple and ornamental pear; *Phomopsis* twig blight on juniper; *Dothistroma* needle blight of pine; *Botrytis* on lisianthus and begonia; *Pythium* root rot on many hosts (such as hibiscus, kalanchoe, gerbera, and azalea); *Phytophthora* root rot of china doll; dollar spot and spring dead spot on bermuda turf; brown patch on bentgrass turf. Vegetable diseases include: bacterial leaf spot, tomato spotted wilt, and *Septoria* leaf spot of tomato; anthracnose, cucumber mosaic, and watermelon mosaic 1 on watermelon; and maize dwarf mosaic on sweet corn.

Three diseases of note have been unusually severe in parts of Texas this year. These include: rose downy mildew, caused by *Peronospora sparsa*; Entomosporium leaf spot (pathogen *E. maculatum*) on photinia and Raphiolepis; and Northern corn leaf blight caused by *Exserohilum turcicum.*

In Nevada an intensive white rot of garlic survey is in its third year. This state wide survey intends to identify all infestations in order to fumigate those areas. In fields that contain such *Sclerotium cepivorum* infestations the remainder of the crop can be harvested for dehydration only (these bulbs

cannot be used for seed). Verticillium wilt (*V. albo-atrum* var. *alfalfae*) has now been confirmed in four Nevada counties. Experiments in White Pine County are investigating the effectiveness of Verticillium-resistant alfalfa cultivars.

New Mexico continues to experience unusually rainy weather. The primary result of this wet condition has been many cases of water-logging and other problems associated with poorly aerated, saturated soils. There are also scattered reports of bacterial leaf spot on pepper and tomato, and Pythium damping off. Powdery mildew has been severe on many ornamentals and even some desert shrubs.

California also experienced spring rains. Downy mildew of rose (*Peronospora sparsa*), Heterosporium leaf spot (*H. variable*) of spinach, bacterial leaf spot (*Xanthomonas campestris* pv. *vitians*) of lettuce, and other diseases benefited from this moisture. Downy mildew of lettuce (pathogen *Bremia lactucae*) was unusually severe in March and April in the coastal counties. The first report of spinach anthracnose (*Colletotrichum dematium*) was also made at this time. Moving into May and June, lygus bug populations climbed and resulted in heavy damage on coastal vegetables. Damage caused by this insect was sometime considered to be a disease. Also, beet leafhoppers were in abundance in some of the tomato and pepper growing areas. Reports of curly top disease are common from these regions.

Central Region

Karen Rane

Clinics throughout the Central Region are continuing to receive numerous samples of brown-needled evergreens. Several periods of drought over the last three years plus the unusual cold weather last autumn (as described in previous *PDQ* Regional Reports) have taken their toll on spruces and pines. In Ohio, death of entire spruce trees has been attributed to these adverse.weather conditions. Deciduous trees and shrubs are also showing similar weather-related problems. In some instances, marginally hardy species such as peach and apricot produced new leaves this growing season, then collapsed and died suddenly.

Frost injury on woody and herbaceous ornamentals was also widespread throughout the region. Warm temperatures in late February and early March encouraged early foliar development, and a return to normal March temperatures resulted in damage to new growth. Anthracnose on maple and ash has been severe in some parts of the region as well. Other woody ornamental problems of note this spring include Dothistroma needle blight (Illinois, Minnesota), Diplodia tip blight (Illinois, Kansas, Michigan, Minnestota), Phomopsis canker on spruce (Michigan), Cytospora canker on cottonwoods and poplars (Kansas) ash yellows (Minnesota) and pine wilt (Kansas, Missouri).

English ivy plantings in Indiana, Kansas and Ohio were found to be severely affected by anthracnose (*Colletotrichum* sp.). Botrytis blight of Artemesia 'Silver Mound' in Illinois, foliar nematodes in phlox in Missouri, and Fusarium crown rot of phlox in Illinois were of interest this spring. In Indiana, Iceland poppy plants showing crown rot symptoms were found to be infected with a *Dendrophion* sp. *Dendrophion papaveras* is an important seed-associated fungal pathogen of opium poppies in Europe and the Middle East.

Field crops in the Central region were adversely affected by the weather. Cold injury in alfalfa was reportedly severe throughout Indiana and Missouri. In Ohio and Indiana the canola crop was virtually wiped out by the pattern of alternating warm and cold weather in the fall and winter. Wheat in Wisconsin, Kansas and Indiana also suffered cold injury. Wheat rust in Kansas is severe this spring, with yield losses expected. Septoria leaf spot has been reported from Illinois, Kansas, and Missouri. In Missouri, wheat streak mosaic and wheat soilborne mosaic viruses were common, while in Kansas wheat streak mosaic virus occurrence was low. Extensive flea beetle feeding has been observed on corn seedlings throughout the region, and Stewart's wilt is expected to be a serious problem. Turf problems reported in the region include snow mold (Minnesota), and late greening of zoysia lawns due to cool spring temperatures (Indiana).

The University of Nebraska has recently hired a new diagnostician for the Plant Disease Clinic. Diane Merrill has a Bachelor's degree from Nebraska Wesleyan University, and a Master's degree from the University of Wisconsin. The diagnosticians of the Central Region welcome you to the fold, Diane!

Northeast Region

Anne Bird Sindermann

Adult western flower thrips (WFT) were collected in March from Adams County, Pennsylvania at a site where a population of WFT has been monitored since last year. This is the first record of WFT overwintering in Pennsylvania. WFT had been collected from peach and nectarine trees last summer after fruit showed silvering and russetting damage as a result of WFT feeding. At this time there is no evidence of TSWV in weeds at the sites of overwintering WFT.

In New Hampshire, a daffodil crop loss was attributed to *Fusarium oxysporum.* Bulbs from Holland potted in sterile mix last fall, put in cold storage, and then forced emerged with blasted tissue. Bulbs cut in half revealed that the bulb scales were rotted throughout.

Bacterial blight of zonal and ivy geraniums was reported in several northeastern states. Tiny brown leaf spots, brown wedges and yellow or wilting leaves may all be symptoms of plants infected with *Xanthomonas campestris* pv. *pelargonii.* Cultivars infected included 'Pink Expectation', 'Yours Truly', 'Cherry Blossom' and 'Snow White'.

Tobacco ringspot virus was detected in bedding impatiens from greenhouses in Maryland and Pennsylvania. Plants were stunted and in some cases displayed oakleaf mosaic patterns but symptoms were not consistently associated with plants that tested positive for tobacco ringspot virus in ELISA. The source of virus has not been determined. The symptoms were different from TSWV in that the plants were very stunted and the line pattern (when present) was more of an oakleaf mosaic. A few leaves showed ringspots but those were not the dark purple ringspots typically associated with TSWV in this host.

Few problems of woody plant material were reported other than winter damage due to the lack of snow cover in southern New England. Rhabdocline needlecast of Douglas fir has been observed in New Jersey and Pennsylvania. Due to the cool, wet weather, *Rhabdocline* sporulated in mid-April in south central PA.

Pink snow mold of turf caused by the fungus *Microdochium navalle* has been rampant in New Jersey, New York and Maryland. Cool temperature brown patch caused by *Rhizoctonia cerealis* was also a big problem in turf throughout the winter. Ascochyta leaf blight of turf was diagnosed in New Jersey. The primary symptom of this disease is bleached leaf tips. Pycnidia may be observed in the leaf tissue.

Southeast Region

Jackie Mullen

Most states in the Southeast reported cold damage after the up and down temperatures of winter and spring. For the most part winter in this area was mild with sudden freezing temperatures in March and April. These conditions were just about perfect for cold injury on landscape and field crops. In addition, powdery mildews were abundant this spring, as much of the spring in many parts of the Southeast was relatively dry. Downy mildews and secondary fungal leaf spots on senescent leaves of broadleaf evergreens were also reported to be common in many sections. Fire blight, a common occurrence in spring, was present but generally observed to be less of a problem than usual. Dogwood anthracnose (*Discula*) occurrences were reported this spring in North Carolina, Kentucky, and Alabama. Tomato spotted wilt virus (TSWV) was reported on floriculture crops in North Carolina, South Carolina, and Florida. Florida reported pocketbook flower (*Calceolaria*) with L strain and watercress (*Nasturium*) with L strain; North Carolina reported dahlia with the L strain and gloxinia and impatiens with the i strain; South Carolina noted Verbena with the i strain.

Disease reports at this time of year are long and - in the interest of space - I will report here only the unusual or noteworthy incidences.

North Carolina. Field crops: Oats - barley yellow dwarf virus; oat soilborne mosaic virus; wheat - wheat soilborne mosaic virus, wheat spindle streak virus, and wheat streak mosaic. Ornamentals:

Leyland cypress - Seiridium canker, Phytophthora root rot; Japanese maple - Verticillium wilt; lilac - bacterial blight (*Pseudomonas syringae*); wax myrtle - Septoria leaf spot; dusty Miller with Alternaria leaf spot; geranium - bacterial blight (*Xanthomonas campestris*); marigold - bacterial leaf spot (*Pseudomonas syringae* pv *tagetes*) and iris with physiologic stem collapse, which appears to be caused by rapid cell expansion during warm periods. Fruits: Strawberry with angular leaf spot (*Xanthomonas fragariae*), anthracnose (*Colletotrichum*) and multiplier disease (Mycoplasma-like organism). Vegetables: Tomato with Stemphyllium leaf spot and target spot (*Corynespora*).
(T. Creswell)

Florida. *Botrytis cinerea* on several greenhouse and field crops (geranium, basil, Exacum, onion, strawberry, rose); widespread *Rhizoctonia* on many different plants; a tobacco (predominantly) seed bed problem of *Rhizoctonia solani* called target spot (field incidence of target spot occurs also); TSWV in tobacco with a 40% incidence estimate (in previous years the over-all incidence level in tobacco was below 5%). Also, bermuda grass and St. Augustinegrass decline (*Gaeumannomyces graminis* var. *graminis*) was prevalent this past quarter. Other reports from Florida: Ornamentals: Oleander - *Pseudocercospora nerulla*; fatsia - *Alternaria panax*; Carissa - *Sphaeropsis tumefaciens*; snapdragon - *Puccinia antirrhini*; gardenia - *Mycosphaerella gardeniae*; *Rhapis* - *Phaeotrichoconis* sp.; palm - *Thielaviopsis* sp.; and canna with canna mosaic virus. Fruits: Blueberry leaves/fruit with *Pucciniastrum nerulla*; strawberry with *Xanthomonas fragariae*. Vegetables: Greenhouse tomato with *Erwinia carotovora* hollow stem. (R. Cullen, G. Simone)

Tennessee. Field Crops: Alfalfa with Sclerotinia crown and stem rot was particularly a problem this spring on fall seeded fields; wheat spindle streak virus has been common on wheat; with float bed tobacco-Rhizoctonia stem rot and Pythium stem and root rot. Ornamentals: Botryosphaeria canker and other secondary fungal cankers were commonly found on a variety of woody ornamentals, probably a result of environmental stress and winter injury; several types of greenhouse bedding plants were diagnosed with Pythium and Rhizoctonia root rot; Phytophthora root rot is being found on woody ornamental nursery stock and commercial and homeowner landscape plantings; there was a low incidence of TSWV in bedding and greenhouse plants. B. Long noted that rose rosette disease was found in one additional county (Obion) this spring in west TN on multiflora rose. Also, there was high incidence of white pine decline this past spring. A large number of white pines developed yellow needles and needle drop during spring. There were many reports of white pines dying. The majority of these problems seemed to be related to environmental or cultural stress. (B. Long)

Kentucky: More problems were noted with float system tobacco plantings. Lower stem rot due to *Sclerotinia* and *Rhizoctonia* was prevalent. The rot was often found to occur in a circular pattern involving 10 - 20 plants. With the *Sclerotinia* infection, the hard black sclerotia were often found.

With the first case of dogwood anthracnose for the season, B. Eshenaur reported that the affected branches failed to flower or leaf-out. The surface of many of the small twigs on these branches was rough with a large number of small protruding fungal fruiting bodies. (B. Eshenaur)

South Carolina. Field Crops: Wheat with soilborne wheat mosaic virus and barley yellow dwarf virus (BYDV); oats with BYDV. Ornamentals: Cercospora leaf spots on rhododendron, ligustrum, pittosporum, hawthorn; Tar spot (*Phacidium* sp.) on 'Nellie R. Stevens', 'Emily Bruner', American, and 'Burford' hollies; algal leaf spot (*Cephaleuros virescens*) on camellia and tea; camellia canker and dieback (*Glomerella cingulata*); Phytophthora root rot on azalea, ivy, camellia, juniper, holly, boxwood; root-knot nematode on boxwood and holly; ring nematode on ivy and holly; Phomopsis dieback on azalea, camellia, and dogwood; Botryosphaeria dieback on azalea, camellia, and holly; bacterial leaf spot (*Xanthomonas campestris* pv. *zinniae*) on zinnia; bacterial leaf spot (*Pseudomonas cichorii*) on vinca; bacterial blight (*Pseudomonas syringae*) on lilac; false smut (*Graphiola phoenicis*) on Canary Island date palm. Turfgrass: ring nematode on St. Augustinegrass. (J. Blake)

Louisiana. Field Crops: Rice - water mold (*Pythium* spp.) has been very active during the prolonged cool season; sugarcane - red rot is evident this spring because of the adverse weather conditions we have had since the cane was planted last fall. Ornamentals & Turf: Many of the shipments of India hawthorn into the New Orleans area had fire blight; this is the second consecutive year for that to occur; with iris, bacterial crown rot (*Erwinia* sp.) and rust (*Puccinia* sp.) were more prevalent than normal;

with rose, downy mildew (*Peronospora sparsa*) has been at epidemic proportions in south Texas and Louisiana. (C. Hollier)

Mississippi. Field Crops: Bacterial stripe and black chaff (*Xanthomonas*) was observed on wheat as an unusual occurrence. Ornamentals: Quince rust (*Gymnosporangium claviceps*) on cedar/juniper. Vegetables: *Sclerotinia sclerotiorum* was observed as a serious problem on greenhouse tomato (called timber rot) and cabbage (watery soft rot). Cold damage was observed on pine trees. (M. V. Patel)

Georgia: Unusual disease occurrences included an apparent *Pseudomonas syringae* pv. *syringae* (tentative diagnosis) leaf spot on holly which appeared in a nursery situation where wind and sand injury may have contributed to disease development. Investigations are still on-going as to the mechanisms of the disease and its development (S. McCarter).

Erwinia carotovora soft rot on iris was a problem and G. Moody speculated that depth of planting may be involved with disease susceptibility. He recommended planting the rhizome very close to the soil surface. (G. Moody)

Alabama. Field crops: Anthracnose (*Colletotrichum*) and Pleiochaeta leaf spot on lupin; suspect (not confirmed as yet) soilborne wheat mosaic virus on wheat; loose smut (*Ustilago*) on wheat. Fruits: Phomopsis (syn. *Fusicocum*) canker on peach. This canker disease was a real problem last year (spring) in certain orchards. Usually the lesions occur on dormant lateral buds or leaf scars. As cankers develop they become brown with (often) gray centers. Sometimes a zonate pattern is present and during humid, moist weather tiny black pycnidia may be seen on the spots. On susceptible cultivars, dieback was severe enough to kill last year's growth. Vegetables: Bacterial canker (*Clavibacter*) on tomato. Ornamentals: Phomopsis canker on sycamore; Phyllosticta blight on Norfolk Island pine; Heterosporium leaf spot and soft rot (*Erwinia*) on iris; Armillaria root rot on red cedar; brown patch (*Rhizoctonia*) on centipede and zoysia. Take-all patch (*Gaumannomyces graminis* var. *graminis*) was found to occur this spring in a St. Augustinegrass lawn in the northeastern section of the state. This is a noteworthy find as it was just last summer when it was identified for the first time in Alabama and Florida. Pathogenicity was recently confirmed by M. Elliott in Florida. Also this spring (April), *G. graminis graminis* has been found in Alabama (northwestern section of the state) on zoysia. The tentative diagnosis of take-all patch on zoysia has not - as yet - been confirmed by pathogenicity tests. (J. Mullen)

DIFFUSION

Transfer of several phytopathogenic *Pseudomonas* species to *Acidovorax* as *Acidovorax avenae* subsp. nov., comb. nov., *Acidovorax avenae* subsp. *citrulli*, *Acidovorax avenae* subsp. *cattleyae*, and *Acidovorax konjaci*. A group in Belgium (Lab. Microbiol. microbiele Gen. Rijksuniversiteit Gent) have been at work for a number of years examining the genus *Pseudomonas*. They have now reclassified a number of pseudomonads, including the one causing fruit blotch of watermelon. Using DNA-rRNA hybridizations, DNA-DNA hybridizations, polyacrylamide gel electrophoresis of whole cell proteins, and a numerical analysis of carbon assimilation tests, the rRNA group III *Pseudomonas* species were examined and transferred to the genus *Acidovorax* (see Int. J. Syst. Bacteriol., 1990, vol 40:384-398 for information on the proposed genus *Acidovorax*). *P. avenae*, *P. rubrilineans*, and "*P. setariae*" are placed in *Acidovorax avenae* subsp. *avenae*. *Pseudomonas cattleyae* is transferred to *Acidovorax avenae* subsp. *cattleyae*; and *P. pseudoalcaligenes* subsp. *citrulli* (the fruit blotch organism) has been placed in *Acidovorax avenae* subsp. *citrulli*. The species *Pseudomonas pseudoalcaligenes* subsp. *konjaci* is proposed as a separate species, *Acidovorax konjaci*. International Journal of Systematic Bacteriology 1992, **42**:107-119.

Bacterial leaf scorch of landscape trees caused by *Xylella fastidiosa*. James Sherald (National Park Service, Washington, D. C.) and Stanley Kostka (Crop Genetics International, Hanover, MD) have written another update on the leaf scorch organism and its hosts. Natural hosts (asymptomatic) include the following: *Sambucus canadensis*, *S. caerulea*, *Parthenocissus tricuspidata*, *P. quinquefolia*, *Ampelopsis arborea*, *A. brevipedunculata*, *Callicarpa americana*, *Baccharis halimifolia*, *B. pilularis*, *Rhus* sp. (sumac), *Solidago fistulosa*, *Rubus* sp. (blackberry), *Artemisia vulgaris* var. *heterophylla*, *Trifolium repens* var. *latum*, *Cynodon dactylon*, *Digitaria sanguinalis*, and *Paspalum dilatatum*. Diseases associated with *X. fastidiosa* are Pierce's disease of grape, almond leaf scorch, alfalfa dwarf, peach phony disease, plum leaf scald, periwinkle wilt, citrus blight; and elm, sycamore, maple, mulberry and oak leaf scorch. J. Arboriculture 1992, 18:57-63.

Tectacervulus mahoniae, *Kabatina mahoniae* and *Selenophoma mahoniae*, three new fungi on *Mahonia repens*. Those "Phyllosticta" leaf spots on Mahonia may not be *Phyllosticta*. Annette Ramaley of Durango, Colorado has created a new genus and species (*T. mahoniae*) for an acervular coelomycete found in association with light brown lesions on Mahonia leaflets collected in late spring in southwestern Colorado. Lesions were up to 2 cm diameter, often coalescing to involve the entire leaf. Acervuli were light buff in color and remained hidden beneath the intact epidermis. *Kabatina mahonia sp. nov.* was associated with large, light to medium brown spots bordered by narrow dark brown to purple, red, and sometimes chlorotic bands. Lesions were large, up to 3 cm diameter, covering half to nearly entire leaflets. Acervuli were dark brown, erumpent. *Selenophoma mahoniae sp. nov.* was found in association with varying types of leaf spots. No pathogenicity tests were performed for any of these fungi. Mycotaxon 1992, **XLIII**:437-452.

Taxonomy and morphology of *Colletotrichum* species pathogenic to strawberry. Three different species of *Colletotrichum* cause similar looking anthracnose diseases of strawberry. Telling them apart has been a matter of opinion. P. S. Gunnell and W. D. Gubler (Univ. of California, Davis) have examined the three species involved and have found that traditional methods of distinguishing them have depended on unreliable characters. Conidial morphology, usually emphasized as a criterion for identification, was found to be variable unless cultures were grown on strawberry leaf agar (using gas sterilized portions of dried strawberry leaves). Conidial shape was more useful than size in species determination. Setae were found to be useful characters, especially for *C. fragariae* and *C. acutatum*. Setae of the former produced conidia when mature. All three species were recognized as separate. Mycologia 1992, **84**:157-165.

Cytological changes induced by wheat streak mosaic virus in cereal leaf tissues. J-G Gao and A. Nassuth (Univ. Guelph, Ontario) have documented the types of inclusions that are present in WSMV infected wheat, barley, and triticale leaves. Other changes in the cells also occur with infection. Inclusions were visible in leaf epidermal cells after staining with the O-G combination as dots or linear bodies distributed in the cytoplasmic matrix. These manifestations were interpreted as perpendicular or end views of cylindrical inclusions. Canadian Journal of Botany 1992, **70**:19-25.

DESIRED...

Here's an Offer You Can't Refuse

Let's get back to the barter system. Illinois has some 230 Reports on Plant Diseases. You can obtain a listing by sending a card to: Phyllis Henegar, N-533 Turner Hall, 1102 South Goodwin Ave., Urbana, IL. Just put a check mark by those you would like copies of, return it to Phyllis, and we'll send them to you free of charge - if you will send us an equal number of your newer "fact sheets." Fair enough? No exchange of money - let's keep it simple.

Malcolm Shurtleff

Call for Cultures...

Dr. Thomas C. Harrington of Iowa State University has asked for cultures of *Ceratocystis fagacearum*. He is initiating a study on genetic variation in the oak wilt pathogen and could use all the isolates he can get his hands on. Pure cultures with appropriate collection information from any location would be greatly appreciated. Diagnosticians in the Midwest would be most able to help.

NOTE: It is illegal to ship culture of plant pathogens (or diseased plant material) across state lines without a permit from the Federal Government. The person receiving the cultures must fill out a form PPQ 526 and send it to the appropriate state authority who will then forward it on to the USDA.

April 15, 1992
Announcement #91/92/45

POSITION AVAILABLE:

EXTENSION PLANT PATHOLOGIST
TENURE-TRACK POSITION

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Jerry G. Schickedanz, Chairman
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N. M. State University, Box 3AE
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JUNE 30, 1992 or until suitable candidate is found

EDUCATIONAL REQUIREMENTS:
Ph.D. in Plant Pathology or closely related field.

BASIC QUALIFICATIONS:
- Strong interest in Extension and in design and establishment of educational programs.
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- Provide training and assistance to county agents and others in areas of plant pathology.
- Maintain liaison and close working relationship with the NMSU Entomology, Plant Pathology and Weed Science Department, as well as with State and Federal agencies and the agriculture industry.
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Preliminary Agenda For APS Diagnostics Committee Meeting

August 1992

I have outlined below a preliminary agenda for the APS Portland Diagnostics Committee Meeting. Please look this over and contact me if you have suggestions/additions. Also, please be thinking about nominations for the new vice-chair (vice-chair 1993; chair - 1994) for the committee. Nominations and elections will be held at the committee meeting in August. If you plant to nominate someone, please contact the individual ahead of time to be sure they will be willing & available to serve if elected.

1. Introductions
2. Roll Call of Committee - C. Semer
   Designation of new incoming committee members
   Designation of outgoing committee members
   (Distribute diagnostics committee roster list update)
   Circulate attendance sheet for committee records
3. Review of minutes of St. Louis Meeting for approval - C. Semer
4. Old Business
   a. Registry of Plant Pathologists
   b. Discussion Session at St. Louis "Interpretation of Fungal and Bacterial ELISA Results" - J. Mullen
   c. Diagnostic Lab Roster - C. Sutula
   d. Symposium at St. Louis "Tomato spotted Wilt Virus" - Margery Daughtrey.
   e. PDQ title change - Melodie Putnam
   f. Extension Plant Pathology Reflector with E-Mail System - W. Miller
5. New Business
   a. Diagnostics Committee Account with APS - C. Semer
   b. Diagnostic Manual Subcommittee - C. Semer
   c. PDQ Report - M. Putnam, G. Ruhl
   d. Diagnostic Reference Pathologist Listing - J. Mullen
   e. Spotlight on Diagnosis - M. Hansen
   f. Diagnostics Committee sponsored events at Portland
      1. Rapid Diagnostic Assays for Plant Pathogens Workshop - S. Miller, Chairman of Subcommittee
      2. Diagnostics Committee Poster (J. Mullen, E. Dutky)

3. Plant Disease Diagnostic Contest - B. Eshenaur

g. Possible Future Events for Consideration in Nashville
   1. Turfgrass Disease Workshop?? - Karen Kackley - Dutt
   2. Pythium sp. Workshop?? - J. Mullen
   3. Phytophthora sp. Workshop??
   4. Xylem - limited Bacteria Discussion??
   5. Fusarium spp. Workshop??
   6. Applications of Molecular Techniques to Diagnostic Situations and Diagnostic Clinics??
   7. Rapid Diagnostic Assays for Plant Pathogens Workshop?
   8. Diagnostics Committee Poster??
   9. Plant disease Diagnostic Contest??
   10. Diagnostics Committee Reception??

h. Nominations & Election of New Vice Chair 1993 (Chair in 1994) - C. Semer

i. Meeting: International Congress of Plant Pathology July 28 - August 6, 1993 - C. Semer

j. Other New Business

k. Adjournment

C. Semer

Interpretation of Fungal and Bacterial Enzyme-Linked Immunosorbent Assay (ELISA) Results: Summary of the Discussion Session, 1991 APS Meeting, St. Louis, MO

Jackie Mullen

This discussion session was sponsored by the Diseases of Ornamentals & Turf Grasses Committee and the Diagnostics Committee. Co-Chairs for the session were Bill Shane (Ohio State University) and Jackie Mullen (Auburn University). The topics presented are listed below. Following this list is a summary of each presentation which was provided by each contributor. Approximately 120 people attended the session .

Research Perspectives
- -Sampling Procedures for Detecting *Phytophthora* spp. in Container-Grown Plants.
  Jim MacDonald, University of California, Davis, CA.
- -*Phytophthora cinnamomi*: Detection in Azalea with ELISA.
  Mike Benson, North Carolina State University, Raleigh, NC.
- -*Pythium, Leptosphaeria korrae* on Turfgrass
  Bill Shane, Ohio State University, Columbus, OH.
- -*Rhizoctonia, Lanzia/Moellerodiscus* on Turfgrass
  Bruce Clarke, Rutgers University, New Brunswick, N.J.
- -*Xanthomonas* on Ornamentals
  Steve Nameth, Ohio State University, Columbus, OH.

Diagnostic Laboratory Perspectives
- -ELISA in Plant Disease Diagnostic Labs/Clinics
  Jackie Mullen, Auburn University, AL.
- -Confident Use of ELISA Kits
  Seong Hwan Kim, Pennsylvania Dep. Agriculture, Harrisburg, PA.

Industry Perspectives
- -Criteria for Development of Fungal Immunoassay Interpretations
  James Adams, Agri-Diagnostic Associates, Cinnaminson, N.J.
- -ELISA with Bacteria Pathogens
  Chet Sutula, Agdia, Inc., Elkhart, IN.
- -Use of ELISA for Pseudocercosporella in Wheat
  Dave Saunders, E. I. du Pont de Nemours and Co., Newark, DE.

RESEARCH PERSPECTIVES

Sampling Procedures for Detecting *Phytophthora* Spp. in Container-Grown Plants

J. D. MacDonald
Dept. of Plant Pathology
University of California, Davis

Antibody-based kits, for diagnosis of *Phytophthora* root rots, have recently become commercially available. The kits are produced by Agri-Diagnostics Associates, of Cinnaminson NJ, and are genus-level probes. There are two ways these kits can be used in disease control programs: 1) for rapid disease diagnosis in plants having symptoms of root or crown rot and 2) for early pathogen detection in crops - prior to disease onset. This second use is critical for optimized use of fungicides, and is an underlying concept in integrated pest management strategies. However, because these kits employ a relatively new technology, effective usage and interpretation guidelines still need to be developed for growers.

When testing plants which do not have obvious disease symptoms, one is faced with the problem of sample collection and processing which maximizes the probability of pathogen detection. We have

researched this question focusing on several crop species, including hibiscus, tam juniper, gardenia, and cotoneaster. A large number of plants growing in one gallon containers have been collected from commercial nurseries and root samples were taken from the upper, middle, and lower parts of the root balls. We found that *Phytophthora* was detected more commonly in samples from the lower parts of the root ball than from upper or middle parts. Such zonation is logical since roots in the bottom of containers are in a chronically saturated zone which is most conducive to *Phytophthora* activity, and where roots may be exposed to predisposing oxygen stresses.

We also found that attempts to subsample roots, selecting those which were necrotic (and assumed to be infected), did not significantly increase the likelihood of detection over simple randomly-collected tissue samples. We attribute this to an inability to reliably distinguish roots infected by *Phytophthora* from those killed by exposure to heat, drought, or oxygen stress, or those which die as part of natural root turnover. For these reasons, we feel that performing assays on root segments extracted at random from the lower portion of the root ball will give the best chance for detecting *Phytophthora* spp, if any are present. Research is currently under way to develop sampling methods for *Phytophthora* detection at the "whole crop" level.

Detection of *Phytophthora cinnamomi* in Azalea with Commercial Serological Assay Kits

D. M. Benson
Dept. of Plant Pathology
North Carolina State Univ., Raleigh NC27695-7616

Two commercial serological assay kits were compared to a culture plate method for detection of *Phytophthora cinnamomi* in root samples from inoculated azaleas. Both the multiwell E kit and the rapid assay F kit detected *P. cinnamomi* on azalea roots beginning 1 wk after inoculation. Agreement between immunoassay kits and culture plate results for detection of *P. cinnamomi* was most consistent beginning 3-5 wk after inoculation. Root symptoms, but not foliar symptoms, of Phytophthora root rot were evident during this period. There was a positive correlation between root rot severity in greenhouse trials and root sample absorbance (multiwell) or meter reading (rapid assay) but not between symptom severity and immunoassay results. Although color reactions in the rapid assay detectors became increasingly darker after completion of the test, results after 5 min were as reliable as those after 60 min, since readings for uninoculated controls used to determine test thresholds also increased with time. The multiwell kit detected *P. cinnamomi* in root samples containing as little as 1.0% infected root tissue. In a commercial nursery survey, 5% and 15% of the azalea root samples at two nurseries had positive ELISA values that were unconfirmed by culture plate. The rapid assay kit detected *P. cinnamomi*, was easy to use, and gave results in a short time.

Pythium and *Leptosphaeria korrae* on Turfgrass

Dr. William W. Shane
Department of Plant Pathology
The Ohio State University
Columbus, OH

Our studies have focused on the development and use of antibodies for monitoring two diseases of turfgrass--warm weather Pythium blight caused by *Pythium* species and necrotic ring spot, caused by *Leptosphaeria korrae* .

Studies on *Pythium* have utilized the Agri-Diagnostics *Pythium* multiwell ELISA kit C and its dipstick predecessor (2). Methods for sampling turfgrass tissue were compared for their effectiveness in monitoring Pythium blight epidemics using ELISA. Sample areas consisted of marked strips on golf course fairways and tees with bentgrass (*Agrostis palustris* L.) and annual bluegrass (*Poa annua* L.) naturally infested by *Pythium aphanidermatum*. Samples consisted of: 1) whole plants picked by hand and assayed as whole plants; components; and 2) leaf clippings collected with a reel mower set at a 1.2 cm cutting height. ELISA readings for mowed samples generally matched those for whole-plucked samples. Several episodes of *Pythium* antigen increase were detected by ELISA assays of mowed samples

although signs and symptoms of Pythium blight were not evident. However, increases in ELISA readings for *Pythium* coincided with, but did not necessarily precede, the onset of blight symptoms with a two- to three-day sampling interval. Too infrequent sampling at critical times, and dilution of infected tissue by the sampling protocol may be to blame for the inability to reliably detect epidemic onset before symptoms appear.

At Ohio State University, we have developed a monoclonal antibody, MAB LKc50, against *Leptosphaeria korrae* (1). *L. korrae* is the causal agent of necrotic ring spot on Kentucky bluegrass (*Poa pratensis* L.) and one of the causal agents of spring dead spot of bermuda grass (*Cynodon dactylon* (L.) Pers). The assay has a limit of detection less than 2 µg/ml of lyophilized mycelial homogenate. MAb LKc50 provides a means for rapid detection of *L. korrae*, an ectotrophic root-invading fungus that is difficult to identify using conventional methods. Identification of *L. korrae* based on cultural characteristics *in vitro* is only tentative. *L. korrae* has no known anamorph. Definitive diagnosis of necrotic ringspot requires the presence of the teleomorph which is rarely detected on field samples. Production of the teleomorph in culture can take months using current techniques.

Recently John Stier and I have compared antibody-aided detection versus conventional isolation techniques for detection of *L. korrae* on turgrass (3). ELISA and isolation techniques yielded similar results when samples were moderately to heavily colonized by the pathogen. Isolation techniques were more sensitive than ELISA when samples were lightly colonized by *L. korrae*. Assays with ELISA for necrotic ring spot are fast, but should be followed up by isolation techniques when antibody assay results are not definitive.

References

1. Nameth, S. T., Shane, W. W., and Stier, J. C. 1990. Development of monoclonal antibodies for diagnosis of necrotic ring spot of turfgrass. *Phytopathology* 80:1208-1211.
2. Shane, W. W. 1991. Prospects for early detection of Pythium blight by antibody-aided monitoring of Pythium blight on turfgrass. *Plant Disease* 75:921-926.
3. Stier, J. C., and Shane, W. W. Horizontal and vertical distribution of *Leptosphaeria korrae* in turfgrass determined using monoclonal antibody and isolation and techniques. (manuscript in review).

Reducing Fungicide Inputs on Bentgrass Putting Greens Using Disease Prediction Models and ELISA-based Monitoring

Karen A. Plumley and Bruce B. Clarke
Department of Plant Pathology, Rutgers University

The golf course environment is one of the most intensively managed systems in the world. It requires extensive resources, both financial and agronomic, to maintain turf quality at an optimum level. At the same time, turf managers have come under increasing pressure to reduce pesticide usage. Calendar-based applications of fungicides, in particular, have been targeted as wasteful and ecologically unsound. In response to this concern, studies were initiated at Rutgers University to develop and refine disease detection and prediction methods to reduce fungicide inputs. Brown patch, a devastating disease of turfgrass caused by the fungus *Rhizoctonia solani*, was used as a model system for this research.

A commercially available enzyme linked immunosorbent assay (ELISA), marketed under the trade name Reveal, was used to monitor the population of *R. solani* on a bentgrass green from 1989 to 1991. Turf was maintained at a 0.635 cm cutting height throughout the study. Foliage was randomly sampled from five locations within four 1 x 3 m plots. Subsamples were combined within plots and assayed using the Reveal kits. Turf was assayed on a tri-weekly basis (Monday, Wednesday, Friday) between June and August. Pathogen levels, as measured by the color intensity of the assays, were quantified using an Agri-Meter II reflectometer.

Disease progress curves developed for the 1989, 1990, and 1991 growing seasons displayed a strong correlation (R^2 = 0.691-0.740) between percent turf area infected by *R. solani* and mean Agri-Meter II readings. In most cases, meter readings corresponded to visual brown patch symptoms only. However, the ELISA assays did occasionally predict disease outbreaks 24 to 48 hr prior to visual symptom expression. The failure to consistently predict brown patch epiphytotics was presumably due to the sampling interval rather than an inability of the assay to detect low pathogen levels.

In 1991, ELISA assays were also utilized to schedule the application of propiconazole (Banner), a fungicide commonly used to control brown patch on golf course greens. Using the assay procedures

described above, propiconazole was applied to turf at a rate of 0.8 kg ai/ha (2.0 fl oz product/1000 ft^2) whenever a mean Agri-Meter II disease threshold value of 23 was reached or exceeded. Within these parameters, the incidence of brown patch (> 5% turf area diseased) was reduced from five outbreaks (for non-treated turf) to one outbreak. When this was compared to a 14-day calendar-based schedule typically used by turfgrass managers, the ELISA-based format reduced the number of fungicide applications from four to two, while disease incidence remained the same (one epiphytotic each).

The ELISA and calendar-based schedules were also compared in 1991 to a computer-based disease forecasting model developed in association with Neogen Corporation. The computer model utilized hourly weather data (including air temperature, relative humidity, and rainfall) to predict when conditions were conducive for brown patch development. Propiconazole was applied in response to model predictions, providing seven days had elapsed since the previous fungicide application. Throughout the 1991 season, the model predicted each brown patch outbreak 24 to 48 hr before symptoms developed on untreated turf. Although turf treated in response to model predictions remained disease free, a total of five propiconazole applications were made during the summer.

It is apparent that both the ELISA and computer-based disease forecasting formats have advantages over the 14-day calendar-based spray schedule currently being used on golf courses to control brown patch. While the computer model effectively targets environmental conditions that are conducive to brown patch development, it does not take pathogen populations into account and, therefore, may overestimate the number of fungicide applications needed. Monitoring pathogen levels on a daily basis throughout the growing season with ELISA, however, is too costly and labor intensive. As a result, a combination of the two methods would appear to offer the most efficient and cost effective means of reducing fungicide inputs on bentgrass greens. This hypothesis will be tested next season by using the ELISA format to intensively monitor pathogen populations once disease development is predicted.

Detection of *Xanthomonas* on Geranium with ELISA: A Research and Clinic Perspective

S. T. Nameth
Department of Plant Pathology
The Ohio State University

A polyclonal antibody-based direct enzyme-linked immunosorbent assay was developed to detect the presence of *Xanthomonas campestris* pv. *pelargonii* (*Xcp*) in infected geranium. The assay was used to determine the presence of *Xcp* in symptomatic and asymptomatic plants. Movement of *Xcp* in root inoculated geranium cuttings was also examined. Results indicated that *Xcp* could be detected within eight days post inoculation. Inoculated plants did not show symptoms of *Xcp* infection until 12 days post inoculation. Geranium tissue samples taken from the basal portion of the stem were the most reliable for *Xcp* detection. In laboratory studies no false negatives or false positives were recorded using this test.

The test was refined into a 40 min. format and used in clinical studies. In 1990, 159 geranium plants were tested, of which 99 tested positive for *Xcp*. Results of these studies indicated ELISA to be a reliable method of detecting *Xcp* in infected geraniums.

DIAGNOSTIC LABORATORY PERSPECTIVES

ELISA Use In Plant Disease Diagnostic Labs

Jackie Mullen
Department of Plant Pathology
Auburn University, AL

This topic will be presented from the broad perspective of a *PDQ* survey of clinics on their use of ELISA, and from an individual perspective on ELISA use at Auburn University's Plant Diagnostic Lab.

PDQ Survey Results

The survey was included as part of a regular distribution of *PDQ;* at least 60 diagnostic labs are in the subscriber list (of almost 140 subscribers). I received 36 replies, or a 60% response. I would like to thank all of you who did take the time to respond!

Information on ELISA use is given in Figure 1. Most responding labs (86%) are using ELISA to assist in disease diagnosis, and of these, the majority are using commercially available kits rather than developing their own antisera. A large percentage (80%) of the responding labs indicated they perform some of the ELISA testing in their own facility. More than half (59%) of the respondents indicated they used ELISA for bacterial or fungal detection.

Many labs indicated some reservation with interpretation of ELISA results. Comments included the need to interpret conservatively; one person suggested that positive results should be greater than three times the negative control value. The importance of including proper controls for each test was stressed. In addition, the value of a plate reader was noted since the eye cannot always estimate color levels accurately, and numerical results hold more value for many clients.

See Figure 2 for a list of problems that were noted by the respondents. A weak positive color reaction was of concern - a low level of color is not enough on which to base a diagnosis. Cross-reactivity is a concern, as are false positives from healthy plant tissues and false negatives. Several mentioned the cost of ELISA kits as prohibitive.

Many feel that the ELISA test should be used in conjunction with other tests (Figure 3). Many respondents preferred use of the multiwell kit when labs are in their busy season, since samples can be run in bulk numbers. The rapid type of kit is used by some lab personnel in the field or when samples are not accumulating very quickly. Some reported that ELISA results are sometimes considered preliminary, to be followed up with other tests. Figure 4 shows the distribution of ELISA product used in diagnostic labs. All responding clinics (21) use ELISA primarily for commercial plant samples , but the test is also used on homeowner samples by many. On average, respondents reported that about 4% of their plant samples were tested with ELISA (Figure 5). About half of the labs charge a fee in order to finance the cost of buying commercial ELISA kits.

My conclusions from the comments made on this survey are positive. I believe most would agree that ELISA is a good diagnostic tool - when in the hands of a good diagnostician.

ELISA at Auburn

For the past three years we have used ELISA on 26%, 13%, and 11% of our plant samples. These figures are high due to our lab's involvement with a state-wide survey for peanut tomato spotted wilt virus the last three years. If you look at our normal client samples, we have used ELISA on only 4-5% of them. (Figure 6)

Phytophthora root rot of nursery and greenhouse samples is a problem we deal with every year, and one that often requires a quick reply. Because of this, we became interested in comparing *Phytophthora* detection using ELISA, isolation, and apple bait techniques. Over the past three years we have compared 171 ornamental samples using these three techniques. We have found good agreement between these techniques when plant samples in the early to middle stages of root rot are tested. It has been often difficult for us to obtain good agreement between culture, apple baits, and ELISA when plant roots are dead or near dead. With tissues in these conditions, it is difficult to detect *Phytophthora* using culturing or apple bait techniques, while the *Phytophthora* ELISA will often give a positive result. Also, as a result of our nursery/greenhouse concerns, we have been looking at the effects of Subdue 2E treatment on subsequent *Phytophthora* detection. Repeated tests have shown that culture results after Subdue treatment may not always be reliable.

As a result of the *PDQ* ELISA survey and our work at Auburn, I believe that ELISA, specifically fungal and bacterial ELISA, can be a valuable diagnostic tool. As with any tool or method, accurate interpretation of the results must depend on knowledge of the plant material, sample condition, and limits of the test.

Figure 1. ELISA Use in Diagnostic Laboratories

ELISA Use	Percent	Number of responses*
Yes, ELISA is used	86	32
Used in Lab (V., B., F.)	80	29
Viral ELISA	65	24
Bacterial ELISA	33	12
Fungal ELISA	39	14
Bacterial & Fungal ELISA	26	9
Testing Service Only (V., B., F.)	17	6
ELISA not used	11	4
Multiwell†	64	23
Rapid assay††	39	14

* The number of labs responding (n) = 36.
†Preferred when multiple samples of a particular disease are expected.
††Preferred when samples are coming in only occasionally or when used in the field; comments included that this test system is more expensive.

Figure 2. Problems/Concerns with ELISA

1. 'Weak' positive results - interpretation of
2. Cross-reactivity of *Pythium* and *Phytophthora*
3. False positive results
4. False negative results
5. Positive control values vary in rapid kits
6. Interpret results in view of sample condition
7. ELISA is a tool to be used along with other tests
8. Question the sufficiency of testing at the developmental stage
9. Kits too expensive for many situations; shelf life and cost prevent use
10. Fungal kits - questionable accuracy
11. Non-pathogenic binuclear *Rhizoctonia* spp. detected
12. To be cost effective, must save samples before testing
13. In many situations, the testing company can process samples more efficiently than a diagnostic lab.

Figure 3. Importance of ELISA in Final Diagnosis

When using ELISA, how important are the results in making the final diagnosis?*

Diagnosis is based solely on ELISA & symptoms (V.)	18%
Diagnosis is based on ELISA & other tests (V.,B.,F.)	50%
Importance of ELISA in diagnosis varies (V.,B.,F.)	27%
Preliminary diagnosis is based on ELISA (B.,F.)	10%

*n = 22.

Figure 4. Source of ELISA Used*

AGDIA	92%
Agri-Diagnostics	50%
Research Antibiotics	38%
ATCC†	4%

*n =26.
†=American Type Culture Collection

Figure 5. Percentage of Plant Samples Tested with ELISA*

Range	<1 - 50%
Average	4.2%
Median	2.0%

*n = 30.

ELISA Use at Auburn University

Figure 6. Number of ELISA Tests (Client and Survey Samples) at Auburn University*

Sample Type	1988	1989	1990
Viral test	477	201	161
Bacterial Test	47	19	6
Fungal Test	43	41	44

*ELISA was used on 26% (564) of all samples in 1988; 13% (261) in 1989; and 11% (211 samples) in 1990.

Interpretation of ELISA Results For Plant Disease Diagnosis - Confident Use of ELISA Kits

S. H. Kim
Pennsylvania Department of Agriculture, Harrisburg, PA 17110-9408

Plant disease diagnosis requires the components of speed, accuracy, and conclusion (interpretation). Use of ELISA kits as tools in diagnosis may well satisfy the need for speed, but the results of the tests, whether positive, negative, or indiscernible, need interpretation.

Speed. Conclusive diagnosis of a disease caused by *Phytophthora* needs confirmation that the pathogen is located in infected tissues. AgriDiagnostics' Alert kit accurately detected *Phytophthora* much faster than isolation procedures. Time consumed in detection of *Phytophthora* from wilting Japanese umbrella pines by the Alert kit, by sporangia induction on the infected tissue, and by isolation on a selective medium (PARP, which contains pimaricin, ampicillin, rifampcin, and pentachloronitrobenzene) took 0.5, 17, and 48 hrs., respectively.

Selection of ELISA for Target Pathogen. Interpretation of ELISA results may vary depending on which ELISA kit is used and on the type of diseased tissue tested. Proper selection of an ELISA kit requires knowledge of the disease, as in the case of the root rot complex of *Rhizoctonia, Pythium* and *Phytophthora*. Both the *Phytophthora* and *Pythium* Alert kits reacted positively to a sample of Phytophthora blight of English ivy hosting a saprophytic *Pythium* species.

Selection of Infected Tissue. Confident use of the ELISA kit largely depends on the diagnostician's ability to select the infection court where the highest pathogen content, living or dead, is located. The infection court of a diseased plant varies from plant to plant. For example an Alert kit detected *Phytophthora* from the rotted stem tissues of Fuchsia, but not from rotted crown or root tissues; an Alert kit also detected *Phytophthora* from the crown of a Rhododendron but not from roots or a composite tissue sample of crown and root.

Sensitivity. A clearly discernible color change of the Alert ampule reactive spot has been obtained (AgriMeter II reading 29-100) when tissue was selected from the infection court for the test. However, an indiscernible or negative color change may occur when the infection court was not targeted for the Alert test.

Geraniums that were acutely infected with *Xanthomonas pelargonii* contained 10^{12} cfu/g tissue. The color reaction of an Agdia dot blot indicated that the leaf debris collected from the geranium contained dead *X. pelargonii*, 10^{12} eq. cfu/g. However, bacteria could not be recovered from the leaf debris upon isolation which indicated that most of the bacteria were dead. An indiscernible visual color change has been experienced from both the Agdia dot blot and AgriDiagnostics multiwell ELISA when the bacteria concentration was $<40^4$.

Conclusion. ELISA is a diagnostic tool which will detect a pathogen regardless of whether it is living or dead, something not possible with traditional cultural methods. However, ELISA cannot replace a diagnostician.

INDUSTRY PERSPECTIVE

Criteria for Development of Fungal Immunoassay Interpretations

J. Adams, T. Joaquim, J. Rittenburg, F. Petersen and G. Grothaus
Agri-Diagnostics Associates, Cinnaminson, NJ

Expanding applications of immunodiagnostics in agricultural applications has resulted in an increased awareness of the critical nature of assay interpretation. There are several critical phases in immunoassay development, including: immunogen development; antibody development; reagent formatting and assay development; sample preparation; assay validation; and development of manufacturing specifications. Ideally, interpretation criteria are set at the beginning of assay development, and successful completion of each phase is dependent on producing a "product" that fits performance objectives.

In general, the basic evaluations performed on a fungal assay to define performance characteristics vary little from one product to another. However, the criteria used to evaluate performance (and develop interpretations) will vary depending on where and how the immunoassay product is used. Interpretation criteria should be based on three practical considerations: needs of the end-user; system

criteria; and assay limitations. A thorough understanding of these three areas prior to development results in realistic interpretation criteria and focused product development.

Prior to issuing interpretation guidelines for a product, performance characteristics of the immunoassay are evaluated. The characteristics of greatest interest are specificity, sensitivity and reproducibility. Specificity is to a large degree determined by the first two developmental stages: immunogen production and antibody development. It is rare to encounter specificity concerns in later stages of development if phases one and two are completed satisfactorily. Specificity does, however, directly impact interpretation of results.

Sensitivity and reproducibility are of greatest concern when evaluating immunoassay performance and developing interpretations. Examples of "sensitivity" criteria include: statistical determination of +/- cut-off in the absence of fungal antigen; effect of background or non-specific binding in target matrices; assay comparison with "standard" evaluation techniques; and antigen dose-response curves.

Regardless of product specifications, reproducibility is a critical issue in immunoassay interpretation. In many applications, the limiting factor is not the actual immunoassay, but the collection and/or preparation of a representative sample. Examples include: presymptomatic verses. symptomatic sampling; random vs. directed sampling; spatial variation of fungal propagules in soil; and environmental and host-related effects on colonization of the host by the pathogen.

In summary, development of interpretation criteria for fungal immunoassays is critical to their successful use in agricultural systems. Selection of specific criteria based on the actual end-use of the immunoassay will ensure that assay development and evaluation are carried out in a manner consistent with the intended use of the product.

ELISA with Bacterial Pathogens

C. Sutula
Agdia, Inc., Elkhart, IN

Agdia, Inc. introduced its first ELISA for bacteria in 1981-1982 - a test for the organism that causes ring rot in potato, *Clavibacter michiganensis* subsp. *sepedonicum*. Since then we have also worked with several tests for *Erwinia*, a number of *Xanthomonas* spp., *Xylella fastidiosa*, and several MLO's. These tests have been developed in three immunoassay formats: (1) DAS ELISA, (2) immunoblots and (3) indirect ELISA.

The above tests have been used primarily in two applications: (1) Confirm presence of bacteria in symptomatic tissue. (2) Screen/detect bacteria in non-symptomatic tissue.

In our experience, ELISA tests in nearly any format perform very satisfactorily when used to confirm bacteria in symptomatic tissue. For cases where the number of organisms is relatively large, i.e., 10^6 to 10^7 bacteria/mL, ELISA tests are very reliable. The specificity of these tests has not always allowed an unequivocal analysis, but in most cases it has been possible to use other observations to demonstrate the consistency and correctness of the ELISA result.

The set of applications in which ELISA are used to screen/detect bacteria in non-symptomatic tissue present serious problems. Generally, ELISA cannot be used to detect bacteria below 10^4 organisms/mL. Thus, ELISA cannot be used to prove the absence of bacteria in non-symptomatic plants.

These ideas were illustrated using bacterial ring rot in potato and *Xcp* infections in geraniums as examples.

Use of ELISA for the Detection and Quantification of *Pseudocercosporella* in Wheat

D. W. Saunders, Agricultural Diagnostics R&D,
E.I. du Pont de Nemours and Company, Newark, DE

The use of immunodiagnostic methods for detecting plant pathogens is becoming a valuable tool for helping growers and advisors monitor the health of their crops. The Du Pont Cereal Eyespot (Foot Rot) Antigen ELISA Kit is an immunodiagnostic kit for detecting and quantifying *P. herpotrichoides* antigen in cereal plants. The Kit offers a more reliable, sensitive, and accurate method for detecting the presence of pathogen than standard visual or culture isolation techniques.

The process of developing an immunodiagnostic method begins with the development of the immunoassay itself, and continues with the interpretation and application of the assay data in a meaningful, practical manner. Quite often, developing the data base and the expertise required to interpret the data requires more time and effort than developing the immunoassay. The same considerations hold true at the user level. At least as much effort should be placed on carefully planning a sampling strategy and analyzing the assay results as should be spent on actually running the assay.

The process of making practical use of any immunodiagnostic method begins by clearly identifying the questions that the immunoassay will be used to help answer. Once the questions have been defined, the level of required crop sampling and data analysis can be established. If the assay is being used to simply identify the presence or absence of pathogen in the crop, then a very superficial sampling strategy used in conjunction with a qualitative analysis of the assay results may be sufficient. On the other hand, if a very precise, detailed disease profile is required, a full testing program would be needed in order to supply sufficient data to make the proper analysis. Another factor which must be taken into consideration is the practical limitation on implementing a complex analysis program. A fully quantitative, detailed sampling and analysis program is required to generate the most precise disease intensity profile. Such a program can easily generate hundreds of samples for analysis. If practical consideration preclude the possibility of analyzing large numbers of samples, then the objectives of the testing program must be modified to reflect a more reasonable level of data analysis.

To make the most effective and efficient use of immunodiagnostic methods the first priority must be to clearly identify the goals of the testing program and then identify the minimum level of sample collection and analysis that will be required to provide accurate data. Since some degree of precision is likely to be sacrificed for the sake of practicality, conservative interpretations should be made from the data.

University Related Plant Disease and Soil Testing Laboratories of the 50 States and Six Canadian Provinces*

compiled by
Gail Ruhl
Senior Plant Disease Diagnostician
Dept. of Botany and Plant Pathology
Purdue University

State Remember, contact your local county extension office for procedures to submit samples to the diagnostic lab.	Department or Institution Performing Soil Test	Department or Institution Performing Plant Problem Diagnosis
Alabama	Soil Testing Laboratory Auburn University Auburn, AL 36849-5624	Plant Disease Clinic 102 Extension Hall Dept. of Plant Pathology Auburn University Auburn, AL 36849-5624
Alaska	Soil Testing Laboratory Agricultural Exp. Station University of Alaska 533 E. Firewood Palmer, AK 99645	Dept. of Plant Pathology Attn: Jenifer McBeath University of Alaska Ag. Forestry Exp. Station Fairbanks, AK 99775-0080
Arizona	Soil,Water, and Plant Tissue Testing Lab Dept. of Soils Water and Engineering University of AZ Tuscon AZ 85721	Plant Disease Clinic Dept. of Plant Pathology Univ. of Arizona Tucson, AZ 85721
Arkansas	Soil Testing and Research Laboratory University of Arkansas P.O. Drawer 767 Marianna, AR 72360	Plant Disease Clinic Lonoke Agricultural Center P.O. Drawer D; Hwy 70 East Lonoke, Arkansas
California	No soil testing service is offered by a public agency	Contact your local County Farm Advisor or Extension Specialist at the University nearest you.
Colorado	Soil Testing Labortory Colorado State University Fort Collins, CO 80523	Plant Diagnostic Clinic Jefferson County Extension 15200 W. 6th Ave. Golden, Colorado 80401 (not functioning at the present time due to lack of funding): Plant Disease Clinic Plant Science Colorado State University Fort Collins, CO 80523

Connecticut	Soil Testing Laboratory Plant Science Department University of Connecticut Storrs, CT 06268	Consumer Horticultural Center University of Connecticut Storrs, Connecticut 06269-4087 Connecticut Ag. Experiment Station Huntington Ave. New Haven, CT
Delaware	Soil Testing Laboratory University of Delaware Newark, DE 19711	Extension Plant Pathologist University of Delaware 126 Townsend Hall Newark, Delaware 19717-1303
Florida	Soil Testing Laboratory University of Florida Gainesville, FL 32611	Plant Disease Clinic Plant Pathology Department University of Florida Gainesville, Florida 32611 Three Regional Satellite Labs: Regional Plant Disease Lab Quincy Research Center Route 3, Box 4370 Quincy, Florida 32351 Regional Plant Disease Lab Southwest Florida Research Center P.O. Drawer 5127 Immokalee, Florida 33934 Regional Plant Disease Lab 18905 SW 280th Street Homestead, Florida 333031-3314
Georgia	Soil Testing and Plant Analysis Laboratory University of Georgia Athens, GA 30602	Extension Plant Disease Clinic 4-Towers Bldg University of Georgia Athens, Georgia 30602
Hawaii	Soil Testing Laboratory Agricultural Diagnostic Service Center 1910 East-West road Sherman Hall 112 Honolulu, HI 96822	Plant Disease Clinic Agricultural Diagnostic Service Center 1910 East-West Road Sherman Hall 112 Honolulu, Hawaii 96822
Idaho	Department of Plant and Soil Science College of Agriculture Moscow, ID 83843	Extension Plant Pathologist University of Idaho Research and Extension Center Kimberly, Idaho 83341 Extension Plant Pathologist Research and Extension Center Parma, Idaho 83660

Illinois	No soil testing service is offered by a public agency	(May-Sept) Plant Clinic 1401 W. St. Mary's Road (Oct-Mar) N-533 Turner Hall 1102 S. Goodwin Avenue University of Illinois Urbana, Illinois 61801
Indiana	No soil testing service is offered to homeowners by a public agency. Contact the Plant and Pest Diagnostic Laboratory for a partial listing of private soil testing labs	Plant and Pest Diagnostic Laboratory Department of Botany and Plant Pathol. 1155 LILY, Purdue University West Lafayette, Indiana 47907-1155
Iowa	Soil Testing Laboratory Iowa State University Ames, IA 50011	Plant Disease Clinic Department of Plant Pathology 105 Bessey Hall Iowa State University Ames, Iowa 50011
Kansas	Soil Testing Laboratory Agronomy Department Kansas State University Manhattan, KS 66506	Plant Disease Diagnostic Lab Department of Plant Pathology Throckmorton Hall Kansas State University Manhattan, Kansas 66506-5502
Kentucky	Soil Testing Laboratory 103 Regulatory Services Bldg. University of Kentucky Lexington KY 40546	(serving Western Kentucky): Plant Disease Diagnostic Lab University of Kentucky Research and Education Center P.O. Box 469 Hwy 91 South Princeton, KY 42445 (serving Central and Eastern Kentucky): Plant Disease Diagnostic Lab Department of Plant Pathology University of Kentucky Lexington, Kentucky 40546-0091
Louisiana	Soil Testing Laboratory Department of Agronomy Louisiana State University Baton Rouge, LA 70803	Plant Disease Clinic 249 Knapp Hall Louisiana State University Baton Rouge, Louisiana 70803
Maine	Soil Testing Laboratory Department of Plant and Soil Sciences University of Maine Orono, ME 04469	Pest Management Office University of Maine Cooperative Extension Service 491 College Ave. Orono, Maine 04473
Maryland	Soil Testing Laboratory Agronomy Department The University of Maryland College Park, MD 20742	Plant Diagnostic Laboratory Department of Botany The University of Maryland College Park, Maryland 20742

Massachusetts	Soil Testing West Experiment Station University of Massachusetts Amherst, Massachusetts 01003	No disease diagnostic service is offered by a agency to homeowners. Commercial samples are handled by individu Extension specialists at the University
Michigan	Soil Testing Laboratory Michigan State University East Lansing, MI 48824	Plant Diagnostic Clinic Department of Botany and Plant Pathology Michigan State University East Lansing, Michigan 48824-1312
Minnesota	Soil Testing Laboratory University of Minnesota St. Paul, MN 55108	For homeowners: Dial U Clinic 145 Alderman Hall University of Minnesota St. Paul, MN 55108 For commercial growers: Plant Disease Clinic Department of Plant Pathology Borlaug Hall University of Minnesota St. Paul, MN 55108
Mississippi	Soil Testing Laboratory Cooperative Extension Service Mississippi State University Mississippi State, MS 39762	Plant Disease Clinic P.O. Box 5446 Mississippi Cooperative Ext. Service Mississippi State, Mississippi 39762
Missouri	Soil Testing Laboratory Room 23 Mumford Hall University of Missosuri Columbia, MO 65211	Plant Science Unit/Diagnostic Clinics: Plant Disease Identification Room 45 Ag. Building University of Missouri Columbia, Missouri 65211
Montana	No soil testing service is offered by a public agency	Plant Disease Clinic 525 Leon Johnson Hall Department of Plant Pathology Montana State University Bozeman, Montana 59717
Nebraska	Soil Testing Laboratory Department of Agronomy University of Nebraska Lincoln, NE 68583	Plant Disease Clinic Department of Plant Pathology University of Nebraska Lincoln, Nebraska 68583-0722
Nevada	No soil testing service is offered by a public agency	Extension Plant Pathologist Dept. of Range, Wildlife and Forestry 1000 Valley Rd. University of Nevada, Reno Reno, Nevada 89512-0013
New Hampshire	Analytical Services Laboratory Nesmith Hall University of New Hampshire Durham, NH 03824	Plant Disease Clinic 322 Nesmith Hall University of New Hampshire Durham, New Hampshire 03824

New Jersey	Soil Testing Laboratory Rutgers University P.O.Box 902 Milltown, New Jersey 08850	Plant Diagnostic Lab Rutgers University P.O. Box 550 Milltown, New Jersey 08850
New Mexico	Soil & Water Testing Laboratory Crop & Soil Science Department New Mexico State University Las Cruces, NM 88003	Extension Plant Pathologist Box 3AE; Plant Sciences Cooperative Extension Service New Mexico State University Las Cruces, New Mexico 88003
New York	Soil Testing Laboratory Agronomy Department Bradfield Hall Cornell University Ithaca, NY 14853	(homeowner and commercial) Insect and Plant Disease Diagnostic Lab. Department of Plant Pathology Cornell University Ithaca, New York 14853 (commercial ornamental samples only) Long Island Horticultural Research Lab 39 Sound Ave. Riverhead, N.Y. 11901
North Carolina	Soil Testing Laboratory Agronomic Division North Carolina Department of Agriculture Raleigh, NC 27611	Plant Disease and Insect Clinic Box 7616 North Carolina State University Raleigh, North Carolina 27695-7616
North Dakota	Soil Testing Laboratory Soil Science Dept. North Dakota State Univrsity Fargo, ND 58105	Plant Diagnostic Clinic Department of Plant Pathology Box 5012 North Dakota State University Fargo, North Dakota 58105
Ohio	Soil Testing Laboratory Ohio Res. & Dev. Center Ohio State University Wooster, Ohio 44691	Plant and Pest Diagnostic Clinic Department of Plant Pathology 2021 Coffey Road The Ohio State University Columbus, Ohio 43210
Oklahoma	Soil Testing Laboratory Agronomy Department Oklahoma State University Stillwater, OK 74078	Plant Disease Diagnostic Lab. Department of Plant Pathology 110 Noble Research Center Oklahoma State University Stillwater, Oklahoma 74078
Oregon	Soil Testing Laboratory Oregon State University Corvallis, OR 97331	Plant Disease Clinic Extension Plant Pathology Cordley Hall 1089 Oregon State University Corvallis, Oregon 97331-2903
Pennsylvania	Soil Testing Laboratory College of Agriculture Pennsylvania State University University Park, PA 16802	Plant Disease Clinic 218 Buckhout Laboratory Pennsylvania State University University Park, Pennsylvania 16802

Rhode Island	Soil Testing Laboratory University of Rhode Island Kingston, RI 02881	Homeowner Clinic Cooperative Extension Education Center University of Rhode Island Kingston, Rhode Island 02881 for commercial samples contact Extension specialists at the University
South Carolina	Soil Testing Laboratory Agricultural Service Laboratory Clemson University Clemson, SC 29634	Plant Problem Clinic Cherry Road Clemson University Clemson, South Carolina 29634-0377
South Dakota	Soil Testing Laboratory Plant Science Department Box 2207-A South Dakota State University Brookings, SD 57007	Plant Disease Clinic Department of Plant Science South Dakota State University Box 2109 Brookings, South Dakota 57007
Tennessee	Soil and Forage Testing Laboratory University of Tennessee P.O. Box 110019 Nashville, TN 37222-0019	Plant and Pest Diagnostic Center University of Tennessee P.O. Box 110019 Nashville, TN 37222-0019
Texas	Soil Testing Laboratory Room 220 Soil and Crop Sciences Texas A&M University College Station, TX 77843	Texas Plant Disease Diagnostic Lab Room 101, L.F. Peterson Building Texas A&M University College Station, TX 77843-2132
Utah	Soil Testing Laboratory Dept. of Plant Soils and Biometeorology Ag Science Building Utah State University Logan, Utah 84322-4830	Plant Pest Diagnostic Lab Department of Biology Utah State University Logan, Utah 84322-5305
Vermont	Soil Testing Laboratory Dept. of Plant and Soil Science University of Vermont Hills Building Burlington, VT 05405-0086	Plant Diagnostic Laboratory Dept. of Plant and Soil Science University of Vermont Hills Building Burlington, VT 05405-0086
Virginia	Soil Testing Laboratory Dept. of Crop and Soil Environmental Science Virginia Polytechnic Institute Blacksburg, VA 24061	Plant Disease Clinic Dept. of Plant Pathology, Physiology and Weed Science VPI and SU Blacksburg, Virginia 24061
Washington	No public agency provides soil testing Contact your local county extension agent for a listing of local labs	(serving eastern Washington) Plant Diagnostic Clinic WSU-Prosser-IAREC Rt. 2 Box 2953-A Prosser, Washington 99350-9687

Washington		(serving western Washington) Plant Diagnostic Clinic WSU-Puyallup Research and Ext. Center 7612 Pioneer Way East Puyallup, Washington 98371-4998
West Virginia	Soil Testing Laboratory 1090 Ag Sciences Building West Virginia University Morgantown, WV 26506	Plant Diseae Diagnostic Clinic 401 Brooks Hall Downtown Campus West Virginia University Morgantown, West Virginia 26506
Wisconsin	Soil & Plant Anaylsis Laboratory University of Wisconsin 511 Mineral Pt. Rd. Madison, WI 53705	Plant Pathogen Detection Clinic Department of Plant Pathology 1630 Linden Drive University of Wisconsin Madison, Wisconsin 53706
Wyoming	Soil Testing Laboratory Plant Science Department University of Wyoming Box 3354 Laramie, WY 82071	Plant Disease Clinic Dept. of Plant, Soil and Insect Sciences University of Wyoming P.O. Box 3354 Laramie, Wyoming 82071-3354
Canadian Province		
Alberta	Soil and Feed Testing Laboratory University of Alberta OS Longman Bldg. 6909 116 St. Edmonton, Alberta	Plant Diagnostic Laboratory Alberta Special Crops and Horticultural Research Centre SS 4 Brroks, Alberta T1R 1E6 Regional,Crop Laboratory Alberta Agriculture Box 10 Olds, Alberta T0M 1P0 Regional Crop Laboratory Alberta Agriculture Provincial Building Box 7777 Fairview, Alberta T0H 1L0 Alberta Environmental Centre Bag 4000 Vegreville, Alberta T0B 4L0
British Columbia	Soil Testing Unit British Columbia Department of Agriculture 1873 Spall Road Kelowna, BC V1Y 4R2	Plant Diagnostic Laboratory British Columbia Ministry of Agriculture and Fisheries 17720-57th Ave Surrey, B.C. V3S 4P2
Manitoba	Department of Soil Science University of Manitoba Winnipeg, Man. R3T 2N2	Plant Pathology Laboratory Agricultural Services Complex University of Manitoba Winnipeg, Manitoba R3T 2N2

New Brunswick	Agricultural Soils Lab New Brunswick Dept. of Agriculture P.O. Box 6000 Fredericton, New Brunswick E3B 5H1	Plant Pest Diagnostic Lab New Brunswick Dept. of Agriculture P.O. Box 6000 Fredericton, New Brunswick E3B 5H1
Nova Scotia	Soil and Crops Branch Nova Scotia Agricultural College Truro, N.S. B2N 5E3	Plant Diagnostic Laboratory Nova Scotia Dept. of Agriculture and Marke Kentville Research Station Kentville, Nova Scotia B4N 1J5 Plant Diagnostic Laboratory Dept. of Biology Nova Scotia Agriculturral College Box 550 Troro, Nova Scotia B2N 5E3
Ontario	No provincial soil testing service is offered	Pest Diagnostic and Advisory Clinic University of Guelph Department of Environmental Biology Guelph, Ontario Canada N1G 2W1
Prince Edward Island	Soil and Feed Testing Laboratory Department of Agriculture Box 1600 Charlottetown, Prince Edward Island C1A 7N3	Plant Pathologist Department of Agriculture Box 1600 Charlottetown, Prince Edward Island C1A 7N3
Quebec	Canadian Industries Limited Soil Laboratory Beloeil Works McMasterville, Quebec	Laboratoire de diagnostic Le Service de Recherche en Phytotechnie de Quebec 2700, rue Einstein Ste.-Foy, Quebec G1P 3W8

*Many states have diagnostic labs associated with their State Department of Agriculture or with United States Department of Agriculture (USDA) Research facilities.

Virus Inclusion Workshop

Karen K. Rane
Plant Disease Diagnostician
Purdue University

Ten scientists from academic institutions and private industry attended a workshop on virus inclusions in Gainesville on March 24-25, 1992. The workshpo was sponsored by the Florida Extension Plant Disease Clinic and the Agronomy Department, University of Florida. Gary W. Simone, Associate Professor, Richard E. Cullen, Senior Biologist, Department of Plant Pathology, and Richard G. Christie, Senior Biologist, Department of Agronomy served as hosts and instructors for the event. The workshop focused on the use of virus inclusion techniques in diagnosing plant virus diseases.

Instruction included slide presentations of micrographs as well as actual sectioning and staining of infected plant material. Examples of viruses from several different groups (potyviruses, tobamoviruses, potexviruses, tomato spotted wilt virus, and others) were available for observation, and many viruses were presented in more than one host plant species. Each virus studied was presented individually, with discussion and slides demonstrating the characteristics of the inclusions for that particular virus, followed by time for each participant to prepare tissue and make observations on his or her own. A microscope equipped with a video camera was also used for demonstrations.

The organizers of this workshop have addressed some of the difficulties experienced by those who have attended previous short-format training sessions. Participants received two 8-hour days of intensive training rather than three hours as in previous workshops. The student-to-teacher ratio was excellent, and equipment (one microscope per two students) was adequate and in good working condition. Each person received a complete start-up kit with stains, fixatives and fine forceps along with a monograph of virus inclusions and slides prepared from virus-infected plants for reference. This enabled participants to practice the techniques immediately upon returning from the workshop. The site of the workshop, a clinic where the staff routinely uses virus inclusion staining for diagnostic purposes, allowed the instructors and participants access to clinic supplies and staff as needed.

There were a few points where the organization of this workshop could be improved. The timing of the workshop during mid-week was not convenient for keeping travel costs down. An additional half or full day would help to relax the occasionally hurried pace of the instruction. A microscope for each participant would also be an improvement. The organizers passed out an evaluation sheet at the conclusion of the workshop, and in a follow-up letter indicated their desire to adopt the suggestions made by the group in future workshops.

The true test of any workshop is whether participants have learned enough to apply the technique in their own laboratory. Since attending this workshop, I have successfully used virus inclusion staining in teaching undergraduate students in an introductory plant pathology course, and in diagnosing tomato spotted wilt virus infections in clinic specimens. I would therefore describe the training I received in this workshop as a valuable addition to my diagnostic repertoire.

AGRICULTURAL

MU Guide

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Commercial Horticulture

Turfgrass Disease Control -- Cultural

D.D. Minner, P.A. Donald and E.W. Palm
University of Missouri, College of Agriculture

To control turfgrass diseases, manage these three factors — they must occur together in just the right form for a disease to develop:

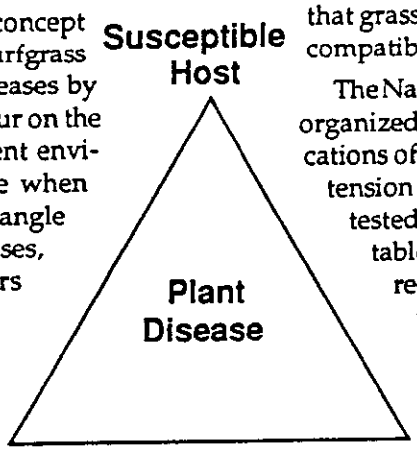
1) the host — a grass that is susceptible to the disease causing organism,

2) the pathogen — the disease causing organism (most turfgrass pathogens are fungi), and

3) the environment — such environmental conditions as temperature and moisture must be in the range for pathogens to be active and spread.

These three factors are like the sides of a triangle, as long as each is present the triangle is complete and there is potential for a disease to occur. If one of the three factors is missing then the triangle is broken and disease will not occur.

Understanding the disease triangle concept helps you properly identify and control turfgrass diseases. It can be used to identify diseases by excluding the pathogens that do not occur on the grass in question — or under the current environmental conditions that you observe when you notice disease symptoms. The triangle concept also can be used to control diseases, by removing any one of the three factors necessary for a disease to occur. For example, the pathogen's environment, especially in terms of water, can be manipulated in several ways. Effective strategies to reduce free water include (1) morning irrigation, (2) removing dew, and (3) reducing the amount and frequency of irrigation.



Disease Triangle

The host can be altered by choosing disease resistant cultivars and species. When manipulating the host and the environment do not provide effective disease management, then reduce the pathogens by applying appropriate chemicals that will either kill the organism or keep it from growing. You must properly identify the disease causing organisms to select the appropriate fungicide for control.

Consequently, before applying fungicides, consider controlling the host (type of grass) and the environment. Host and environmental considerations can be combined in a cultural disease control program that includes management decisions such as fertility, water, drainage, thatch removal, mowing, and resistant turfgrass species and varieties. See the Agricultural Guide on Chemical Turfgrass Disease Control for control measures using fungicides.

Managing the Host

Resistant Grasses: Some diseases can be completely avoided by selecting or substituting grass species that are not susceptible to certain pathogens. For example summer patch is a severe problem on Kentucky blue grass but has little effect on perennial ryegrass and tall fescue. In this situation tall fescue could be completely substituted for Kentucky bluegrass or perennial ryegrass could be over seeded to disguise the problem. Be sure that grasses used as mixtures or over seedings are compatible.

The National Turfgrass Evaluation Program was organized to test cultivar performance in several locations of the United States. Contact your local extension service for the grasses that have been tested and recommended in your area. The table below gives disease resistant cultivars recommended for Missouri. Even though these grasses are termed disease resistant it does not mean that they are 100% disease resistant, and that no infection will occur. Instead the field evaluation process identifies cultivars that show the least amount of disease when a given trial becomes infected. Thus, many of the cultivars listed below are actually *less susceptible* to specific diseases but may *not* actually be *completely* disease resistant.

Managing the Disease Environment

Water: Nearly all diseases require water for the development of fungal pathogens. Some disease problems such as pythium blight, brown patch, and dollar spot are accentuated by extended periods of free mois-

Turfgrass Diseases, Resistant Varieties, and Cultural Controls

Disease *Scientific Name* Host	Symptoms	Cultural disease control
Anthracnose *Collectotrichum graminicola* Host: Annual bluegrass, fine fescue, perennial ryegrass, Kentucky bluegrass.	The fungus is most common on dead or senescing leaves and stems. During cool, wet periods, water soaked stem lesions later turn bleached, girdle the tiller, and cause small patches of plants or individual plants to die. During warm weather plants are stressed, lesions on individual leaves are round to elongate. Reddish brown to brown blotches appear, often surrounded by a yellow halo, that may merge to blight entire leaves. Numerous, minute, black spined fruiting bodies are often observed on the affected tissue if magnified with a 10X hand lens.	**Management Practices:** Use adequate nitrogen, phosphorus and potassium. Avoid water stress on plants. **Resistant varieties** None
Brown patch *Rhizoctonia solani* Host: Bermudagrasses, bluegrasses, Zoysiagrass, perennial ryegrass, fescue.	Brownish-purple to light brown circular area ranging from a few inches to 2 feet in diameter with close-cut turf. Under high mowing conditions, the affected area may be several feet in diameter with sunken areas of matted grass. On close-cut golf greens during periods of warm, humid weather, dark purplish smoke rings may border the area especially in early morning areas.	**Management Practices:** Avoid summer application of nitrogen, especially soluble sources, and frequent irrigation, especially during hot humid conditions. Remove dew and guttation with a light irrigation at sunrise only when disease conditions exist. **Resistant varieties:** *Tall fescue:* Adventure, Maverick, Arid, Cimmaron, Mesa, Monarch, Rebel II, Tributt. *Perennial ryegrass:* Citation, Derby, Omega, Pennfine, Yorktown II, Manhattan II, Tara.
Dollar spot *Lanzia* and *Mellerodiscus spp.* (Sclerotinia homoeocarpa) Host: All turf-grasses.	On closely-cut greens (3/16" to 1/4") this disease appears as dead spots about the size of a silver dollar. On home lawns, spots will be larger, about the size of a fist. Individual leaves initially have yellow-green blotches turning to a light straw color with a dark brown margin. Lesion extends across the entire leaf's width causing an hourglass, or constricted leaf appearance.	**Management Practices:** Avoid nitrogen deficiency, drought, and night watering. If disease symptoms are present, nitrogen levels are known to be low, and water is not contributing to the problem, then a light nitrogen application (.25 to .50 lbs N/1000 sq ft) may stimulate growth and eliminate the need for fungicides. Keep thatch to a minimum. **Resistant varieties:** *Kentucky bluegrass:* Adelphi, Bonnieblue, Bristol, Eclipse, Majestic, Parade, Park, Touchdown, Vantage, Victa.
Fairy rings *Marasmius* and many other mushroom fungi. Host: All turfgrasses.	Circular rings 2-10 feet in diameter. Rings may appear as semicircles on sloped areas. Rings are either yellow or darker green. They grow at a different rate than the surrounding turf. The fairy rings are caused by colonies of mushroom fungi that live in soil and thatch. Mushrooms may be seen under conditions of high soil moisture.	**Management Practices:** Increase nitrogen fertilizer program to hide darker green ring. Core aerify and use wetting agents to increase water infiltration. Water thoroughly. More radical control measures include total removal of all the affected soil or fumigation of the affected area. **Resistant Varieties:** None

Turfgrass Diseases, Resistant Varieties, and Cultural Controls

Disease *Scientific Name* Host	Symptoms	Cultural disease control
Leaf smuts Stripe smut (*Ustilago striiformis* Flag smut (*Urocystis agropyri)* Host: Leaf smuts - bentgrass, bluegrass, ryegrass.	Infected turfgrass plants are generally slow growing, and have a yellow or grayish cast. As the disease progresses, long yellow green streaks develop on the leaves. These become gray or black as epidermal tissue ruptures releasing black spore masses. Loss of water from the ruptured epidermal tissue results in leaf death. Scattered plants or large patches may be infected. Affected plants are systemically infected and are susceptible to other stresses.	**Management Practices:** Avoid excess nitrogen and drought. Use uninfested seed or smutfree sod. If plants are infected allow grass to undergo natural dormancy periods to reduce inoculum. This practice may necessitate renovation and replanting if turf is heavily infected. **Resistant Varieties:** *Kentucky bluegrass:* Adelphi, Birka, Bonnieblue, Glade, Ram 1, Sydsport, Touchdown. Substitute ryegrasses or fescues.
Leaf spots and blights *Drechslera* and *Bipolaris* (*Helminthosporium* spp.), *Septoria*, *Ascochyta*, and *Nigrospora*. Host: Bluegrasses, bermudagrasses, bentgrasses, fescues, ryegrasses, zoysia.	Fungi invade the leaves producing spots with definite margins. The spots may enlarge until the entire blade is affected. The leaf blight stage is not always the most important stage and plant death can occur when the crowns and roots are infected.	**Management Practices:** Avoid excess nitrogen, especially in the spring; excessive use of benomyl, thiophanates, or triadimefon; and night watering. Mild leaf spotting may not require fungicide application. Heavy spring leaf spotting should be treated in the spring since it may result in severe turf loss during the summer from crown rot. Collect and destroy clippings during periods of infection. **Resistant Varieties:** *Kentucky bluegrass:* Birka, Bonnieblue, Bristol, Enmundi, Fylking, Majestic, Parade, Ram I, Touchdown. *Perennial ryegrass:* Diplomat, Manhattan II, Omega, Player, Score, Sprinter, Yorktown II. *Fine fescue:* Atlanta, Biljart, Centurion, Checker, Jamestown, Scaldis.
Powdery mildew *Erysiphe graminis* Host: Bluegrasses, fescues, bermudagrasses.	The fungus appears as isolated tufts of gray-white growth on the upper surface of the grass blade. The mycelial growth may rapidly cover the entire blade surface giving the area a greyish-white cast. Black dots may be seen interspersed with the mycelial growth.	**Management Practices:** Powdery mildew is usually only a problem in shady locations. Several cultivars of fine fescue are better adapted for shade and are more resistant to powdery mildew than Kentucky bluegrass. Turf-type tall fescue and perennial ryegrass are also better adapted for shade than Kentucky bluegrass. Avoid excessive nitrogen. Prune trees to improve light and provide better air flow. **Resistant Varieties:** *Kentucky bluegrass:* A 34, Bristol, Eclipse, Glade, Nugget, Touchdown
Pythium blight (Greasy spot) *Pythium aphanidermatum* Host: All turfgrasses. Bentgrasses are especially susceptible.	This disease appears first as an area of water-soaked tissue which turns light brown as the leaf tissue dies. The disease pattern seen in the turf reflects the presence of poor surface drainage. Under conditions of high humidity, diseased leaves may be covered with white cob-webby mycelial strands.	**Management Practices:** Avoid excess nitrogen and water, especially in hot weather on perennial ryegrass fescue and bentgrass. Provide adequate water and air drainage. Encourage leaf surface drying on a daily basis when conditions are favorable for the disease to occur. Dew or guttation fluids can be removed from turf canopies by a light sprinkling (less than 5 minutes) shortly after day light. Removing free moisture in this manner will cause quicker canopy drying that reduces the mobility and activity of Pythium. Where dew or guttation are not present, light daily watering may favor development of the disease problem. **Resistant Varieties:** None.

Turfgrass Diseases, Resistant Varieties, and Cultural Controls

Diseases *Scientific Name* Host	Symptoms	Cultural disease control
Red thread *Laetisaria fuciformis* **Pink patch** *Limonomyces roseipellis* Host: Bentgrasses, perennial ryegrass, bermudagrasses, fine fescues, bluegrasses.	Red thread can be distinguished in the advanced stages by the presence of bright red to pink fungal structures at the tips of the affected leaves. These two diseases present similar symptoms of irregularly shaped patches of blighted grass. From a distance, the patch may have a pinkish or reddish cast. On individual leaves, the initial blighted areas can enlarge causing leaf death. During prolonged moist weather, the leaves may become covered with pink gelatinous growth.	**Management Practices:** Increase nitrogen. Collect and destroy clippings during periods of infection. **Resistant Varieties :** *Kentucky bluegrass:* A 34, Adelphia, Birka, Bonnieblue, Touchdown. *Perennial ryegrass:* Citation, NK100, NK200, Score. *Fine Fescue:* Atlantic, Biljart, Centurion, Highlight, Pennlawn, Ruby, Scaldis.
Rust *Puccinia graminis* Host: Kentucky bluegrass, perennial ryegrass, zoysiagrass.	Early infections appear as light flecking on the leaves. Epidermal tissues rupture as the disease progresses giving a rusty appearance to the leaves. A rusty orange powder can be rubbed off the leaves when the spores are mature.	**Management Practices:** Usually a problem after turf growth has been slowed by drought. Avoid nitrogen and moisture stress. Light nitrogen application at first sign of infection may help. **Resistant Varieties:** *Kentucky bluegrass:* Fylking, Park, Sydsport.
Seedling blight *Pythium , Rhizoctonia and Fusarium* species Host: All turfgrasses.	In newly seeded areas, seedlings thin or die forming irregular patches. Affected plants may become slimy and form a crust over the soil surface.	**Management Practices:** Avoid spring or summer seeding of cool season grasses. Prepare a well drained seed bed that prevents water from standing on the soil surface. **Resistant Varieties:** None.
Snow molds *Typhula:* rare in Missouri *Gerlachia miralis* Host: Bluegrass, bentgrass, fescues, ryegrass.	Typical symptoms are dead areas of grass during cool wet weather from October to April. A pink cast may be seen on the area due to accummulation of pink fungal spores. No small brown sclerotia are found associated with the dead grass.	**Management Practices:** Avoid early fall nitrogen that leads to lush growth. **Resistant Varieties:** None.
Slime molds *Myxomycetes* Host: All turfgrasses.	Small white-gray or yellowish slimy masses spread on the leaf blade. Fruiting structures develop releasing masses of powdery black spores. Essentially nonparasitic, but unsightly.	**Management Practices:** Use spray from a garden hose or a broom to remove slime mold from leaf surface. **Resistant Varieties:** None -- not needed.
Spring dead spot *Leptosphaeria korrae* Host: Bermudagrass.	**Bermudagrass** -- Circular patches of bleached dead grass in spring when dormant bermudagrass resumes growth. Rough circular, bleached, dead spots appear with regrowth in the spring, varying in size from 2 inches to more than 3 feet in diameter. Sometimes the centers may survive after several years, resulting in "doughnuts." The spots often develop into rings and tend to reappear and enlarge in the same area for several years before disappearing.	**Management Practices:** Remove excess thatch when it reaches 1/2 inch. Aerify to relieve compaction and promote deep root development. Maintain balanced fertility. Promote good soil drainage. **Resistant Varieties:** Cultivars with a high level of winter hardiness are less affected by spring dead spot.

Turfgrass Diseases, Resistant Varieties, and Cultural Controls

Disease *Scientific Name* Host	Symptoms	Cultural disease control
Summer patch *Magnaporthe poae* **Necrotic Ring Spot** *Leptosphaeria korrae* (Fusarium Blight Syndrome) Host: Bluegrasses, bentgrasses, fine fescues, bermudagrasses.	**Kentucky bluegrass** -- Dead turf patches are seen in midsummer ranging from a few inches to a foot or more across. Often an area of dead grass will have areas of green grass in the center (frog-eye). Disease usually seen in fourth or fifth year after lawn initiation and generally most severe on sunny, exposed areas that tend to dry out.	**Management Practices:** Perrennial ryegrass is not affected by this disease. Overseed Kentucky bluegrass with perennial ryegrass to reduce the noticeable damage caused by summer patch or necrotic ringspot. Avoid excess nitrogen, drought, excess water, and very close mowing. Irrigate to maintain even growth with little turf wilting. Alternate wetting and drying cycles may increase infection. **Resistant Varieties:** *Kentucky bluegrass:* Adelphi, Enmundi, Glade, Parade, Sydsport, Touchdown, Vantage, Windsor.
Take-all patch diseases *Gaeumannomyces grammis* Host: Most cool season grasses. Bentgrasses are especially susceptible.	**Bentgrass** -- Typically appears in late spring or early summer as mahogany-brown areas of dead grass. A doughnutlike dead infection center usually occurs in pure bentgrass stands. In mixed stands, the bentgrass is killed and other grasses remain.	**Management Practices:** Use acidifying fertilizers or sulfur to lower thatch and soil pH. Avoid applications of lime. **Resistant Varieties:** None.
Yellow patch *Rhizoctonia cerealis* Host: Kentucky bluegrass, creeping bentgrass, fescue, zoysia.	Light green to yellow green, yellow, tan, straw, or bronze-colored rings and crescent-shaped patches, ranging from a few inches to about 3 feet in diameter, often with green grass in the center of the circles. Smaller yellow patches usually result from infections that occur under cold, wet conditions. The patches are often sunken as a result of rapid decomposition of the thatch. The symptoms appear in cool to cold weather (optimum 40 to 60 F) in the spring, fall, and winter and resemble summer patch and necrotic ring spot.	**Management Practices:** Nitrogen to promote recovery. Resistant Varieties: None.
Zoysia patch *Gaeumannomyces incrustans* (suspected pathogen) Host: Zoysiagrasses	Circular patches may be 2 to 10 feet in diameter and are usually only visible in the spring and fall but have also been observed during short periods of cool, wet and cloudy weather in the summer. In the spring symptoms will usually appear within 60 days of zoysia green-up. As zoysia greens up in the spring those areas affected by the disease will have a greater number of dead leaves, weakened roots, and a slightly sunken appearance, however, no more than 80 percent of the plants will die within the patch. Leaves at the outer edge of the patch will have a distinct yellow/orange color that results in a ring	**Management Practices:** Not yet established. **Resistant Varieties:** None.

Disease *Scientific Name* Host	Symptoms	Cultural disease control
	appearance on the perifery of the patch. As soil temperature increases the symptoms disappear and turf may sometimes fully recover in the affected areas during the summer. The patch and yellow/orange ring will usually occur in the same exact area as zoysia growth slows in the late summer and fall. The patches seem to increase one to two feet in diameter each year.	

Missouri Turfgrass Disease Calendar

Season	Anthracnose	Brown Patch	Dollar spot	Fairy ring	Leaf smuts	Leaf spot & blight	Red thread/pink patch	Powdery mildew	Pythium blight	Rust	Seedling blight	Snow mold	Slime molds	Spring dead spot	Summer patch and necrotic ringspot	Yellow patch	Zoysia patch
Spring				X	•	•	•			X		X	X	•		X	•
Late Spring/ Early Summer	X	X	•	•		X	X		X		X		X		X		X
Summer	•	•	•	X		X	X	X	•		X		X		•		
Late Summer/ Early Fall	•	•	•	X		X	X	•	•	X	X		X		X		X
Fall			X	•	X	X		X		•		X	X				•
Winter												X		X		X	

X -- Means disease *may* occur.

• -- Means disease *most likely* to occur.

Visual Symptoms

Lawn diseases generally show two types of visual symptoms – those affecting indi idual leaves and those that cause larger patches of damaged turf. Use the appropriate table to match the visual symptoms to the disease.

Leaf Symptoms	Dollar spot	Leaf spot	Ascochyta	Septoria	Nigrospora	Red thread
• Defined Lesion Margin	X	X			X	
• Pycnidia			X	X		
• Cobwebby mycelium under high humidity	X				X	
• Dies from leaf tip (sometimes)		X	X		X	X
• Straw colored or bleached	X		X			
• Yellow				X		
• Brown		X		X	X	
• Tan	X				X	X
• Purple/brown				X	X	
• Pink/tan						X

General Patch Symptoms	Pythium blight	Zoysia patch	Brown patch	Yellow patch	Summer patch and necrotic ring spot	Spring dead spot	Red thread
Cotton-like mycelium forms under high humidity							
•White	X		X				
•Pink-red							X
Patch color							
•Purple wilting at first notice	X				X		
•Pink							X
•Tan		X		X		X	
•Greasy brown/tan	X						
Ring forms							
•Yellow					X		
•Purple	X		X				
•Orange		X					
•Tan					X		
Patch type							
•Frog eye (center remains green)					X		
•Patch thinned but not entirely killed		X	X				
•Patch entirely dies	X						
•Sunken patch	X					X	

ture. Extended periods of free moisture in the turfgrass environment can be caused by dew, guttation, and frequent irrigation or rain. (*Guttation* – water droplets that form at the tips of grass leaves.) Remove dew and guttation from grass leaves by dragging a hose across the surface, using a whipping pole, or briefly irrigating it with large droplets — only long enough to wash the dew from the surface.

The objective of all three methods is to spread the concentrated dew or guttation droplets over a larger surface area so the turf canopy will dry faster in the morning.

Improper irrigation alone may create a disease problem that normally would not exist. Avoid frequent irrigation that results in extended periods of free moisture, especially when you expect pythium, brown patch and dollar spot. In general, deep and infrequent irrigation is recommended to allow some soil and leaf drying and a gaseous exchange between soil and atmospheric air. Use drying periods to disrupt the growth cycle of some of the water loving pathogens.

However, some exception to the rule is provided by summer patch and necrotic ringspot.

Alternate wetting and drying cycles usually accentuate these diseases. Avoiding severe drying is especially important because it also may cause additional heat stress. Where summer patch and necrotic ringspot are a problem, apply water frequently enough to prevent even mild turfgrass wilt.

Fertility: From a disease standpoint, supply enough nitrogen so that a proper mowing is required on a weekly basis. Dollar spot, rust, red thread, and pink patch are favored when nitrogen is deficient and turfgrass leaf growth is slow. Sometimes a light application of nitrogen will produce enough active leaf growth so disease symptoms are no longer visible, and you can avoid a fungicide application. If summer patch and necrotic ringspot have caused dead turf, apply nitrogen to speed recovery of the damaged areas. Other disease problems, such as brown patch and pythium blight, can be accentuated by nitrogen application, especially from the soluble nitrogen sources applied in the summer. Heavy spring applications of nitrogen will also encourage leafspot diseases.

Potassium has been shown to suppress disease development. Apply potassium at the same time and in the same amounts as nitrogen.

Drainage and Air Movement: A good exchange of air between the soil and atmosphere is necessary for vigorous turfgrass growth. Turf areas that stay constantly wet because of poor soil water drainage are prime targets for such water-loving, soil-borne diseases as pythium blight and brown patch. Surface contouring and subsurface drainage are costly but permanent solutions to wet soils.

Coring and slicing are turf management practices that can be repeated during the year to temporarily increase air exchange and soil drying. (To let in needed air, you can use a core aeration machine to punch holes into the ground and/or a slicing machine to cut grooves into it.)

You also can increase light penetration and air movement by selectively pruning your trees and shrubs. This will speed the drying of poorly drained areas and also reduce the humidity in localized turf areas on a daily basis.

Thatch: Essentially all turfgrass diseases are reduced by thatch control.

Thatch is a decayed layer of dead grass plants that is located between the soil surface and green portions of grass. It harbors active and resting stages of disease causing organisms. When environmental conditions are optimum, fungi can rapidly grow and attack living turf.

Remove excess thatch when turf is actively growing, so it will quickly recover from power-raking or verticutting — usually spring or fall on cool season grasses and mid summer on warm season grasses. Coring is a slower process of thatch removal but will cause less direct stress on the turf. Remove excess thatch when it accumulates to 1/2 inch in taller mowed turf (1.5 to 3.0 inches) and 1/8 inch in lower cut fine turf (below 0.5 inch).

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Cooperative Extension Service

report on

PLANT DISEASES

DEPARTMENT OF PLANT PATHOLOGY
UNIVERSITY OF ILLINOIS AT URBANA-CHAMPAIGN

RPD No. 102
(Revised 10/91)

SOILBORNE MOSAIC AND YELLOW MOSAIC (SPINDLE STREAK MOSAIC) OF WINTER WHEAT

M.C. Shurtleff, P.T. Himmel, and W.L. Pedersen

SOILBORNE WHEAT MOSAIC

Soilborne wheat mosaic, caused by a soilborne virus, was first discovered in Madison County, Illinois, in 1919. At present, this disease is known to occur in over 50 counties in Illinois. It has been found as far north as Grundy, Kendall, Mercer, and Putnam counties and as far south as Monroe, Randolph, and Wabash counties. The disease occurs throughout the eastern and central United States as well as in Argentina, Brazil, China, Egypt, France, Italy, and Japan.

Fig. 1. A part of the soilborne wheat mosaic test area at the Agronomy-Plant Pathology south farm in Urbana where many of the experimental wheat lines in the United States are tested against soilborne mosaic. Note the dramatic differences in the resistance and susceptibility of wheat cultivars.

Losses in wheat yields vary from year to year due to the cultivars being grown, continuous cropping to wheat, strains of the virus, and environmental conditions favoring disease development. Soilborne mosaic is one of the few diseases of winter wheat that can practically destroy an entire crop of a susceptible cultivar (Figure 1). In Illinois, the widespread use of resistant or tolerant cultivars has kept losses from this disease to a minimum in recent years.

VIRUS

Soilborne wheat mosaic virus particles (called virions) are rigid hollow rods 20 nanometers (nm) wide of two principal lengths, 90 to 160 nm and 300 nm. (Virus particles can only be seen with an electron microscope). Particles of both sizes are necessary for infection. The virus is highly variable and the length of the predomi-

M.C. Shurtleff is Extension Plant Pathologist, P.T. Himmel is Research Microbiologist, U.S. Department of Agriculture and Agriculture Research Service, Crop Protection Research Unit, and W.L. Pedersen is Associate Professor of Plant Pathology, all Department of Plant Pathology, University of Illinois at Urbana-Champaign.

nant short rods often changes as the wheat plants grow. Symptoms change as the predominant rod length changes, but the relation of rod length to yield loss is unknown. Infected cells often contain amorphous and crystalline inclusion bodies that contain virions in a paracrystalline array.

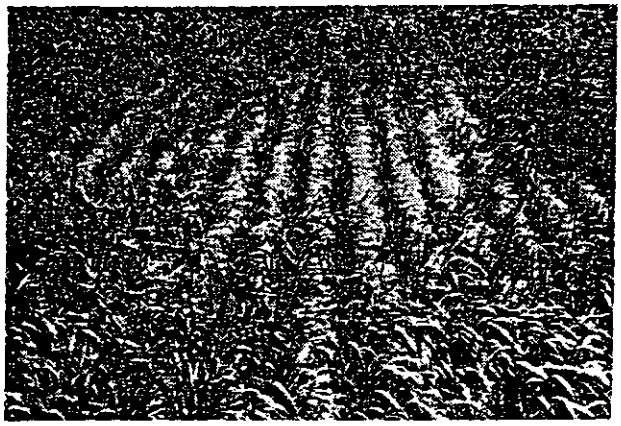

Fig. 2. Area in a wheat field of a highly susceptible cultivar infected with soilborne wheat mosaic virus transmitted by the fungus *Polymyxa graminis*.

The virus also infects certain cultivars of fall-sown rye, barley, emmer, and spelt, wild annual bromegrass (***Bromus commutatus***), sorghum, and some species of ***Chenopodium*** have been inoculated experimentally. Spring-sown wheats and most wild grasses appear to escape infection. The virus can remain infectious in dried leaves for several years or more. It is NOT transmitted through the seed or by insects, but it is transmitted by a soil-inhabiting fungus or mechanically at low rates.

SYMPTOMS

Two types of symptoms have been reported, chlorotic leaf mottling or mosaic and rosetting or stunting. Plants infected with the soilborne wheat mosaic virus usually appear in early spring as irregular patches of light green to bronze-yellow or light purple wheat within a field--depending on the cultivar, strain of the virus, and seasonal growing conditions. Severely diseased fields have an uneven appearance. The size and shape of infested areas will vary. The disease often occurs in poorly drained low areas and waterways in fields. A mosaic-infected area does not increase in size during the growing season. The infected area may increase over time due to tillage, land leveling, or flooding of fields. Wheat in the diseased area may be moderately to severely stunted in the early spring, but may recover later (Figure 2). Under conditions unfavorable for growth, however, infected plants remain dwarfed to maturity. Roots may be more severely stunted than shoots in some cultivars. Some plants may die, while others will produce fewer stems (culms) and heads. Maturity is often delayed. The heads on diseased plants may be shorter than normal heads and have shriveled, lightweight kernels.

The severity of this disease varies greatly, depending on the resistance of the wheat cultivar, concentration and virulence of the virus strain(s) in the soil, weather conditions, and planting date. A prolonged, cool growing period with a mean temperature below 60°F (16°C) appears necessary before susceptible wheat cultivars are appreciably damaged by this disease.

Soilborne wheat mosaic is sometimes mistaken for winter injury, wet spots in fields, an uneven distribution of fertilizers, or a nutrient deficiency. Field ice becomes a problem when wheat yellow mosaic is in the area.

LEAF MOTTLING

Soilborne wheat mosaic is best identified by an irregular mottling, streaking, and blotching of the leaves when plants are growing rapidly in very early spring before heading (Figure 3). The color of the mottling varies from a hard-to-see pale green to a conspicuous lemon yellow that may involve most of the leaf blade as well as the

leaf sheaths and glumes. The mottling may persist into late spring as long as the leaves are green--especially if the temperatures remain abnormally cool. When temperatures are above 64°F (18°C), new growth is symptom-free except for stunting. No yield loss occurs on resistant cultivars.

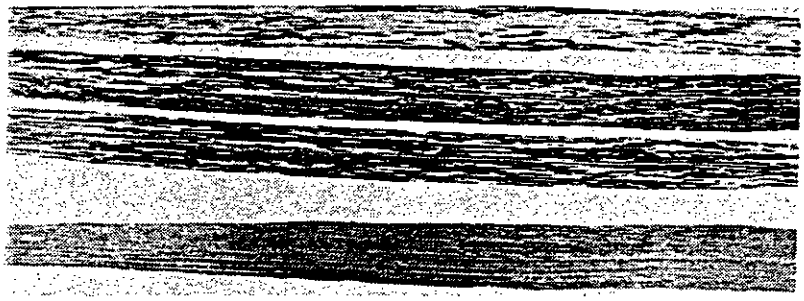

Fig. 3. A healthy wheat leaf (bottom) and three leaves showing symptoms of soilborne wheat mosaic—mottling, parallel dashes and streaks. Mosaic mottling is usually less conspicuous than is pictured here, and young unfolding leaves must be inspected closely before the disease can be identified.

ROSETTING

A few strains of the virus cause rosetting in highly susceptible wheat cultivars. The leaves and tillers remain short, growth is bunchy or compact, and tillering is excessive (Figure 4). The leaves of such plants are usually bluish green and may retain this color throughout the growing season. At other times, rosetted plants die early without developing much green color. Leaf mottling is usually not as pronounced in rosetted plants.

DISEASE CYCLE

The virus causing soilborne wheat mosaic survives in the soil and crop residues from season to season protected by its fungal vector or agent, ***Polymyxa graminis***, an obligate parasite in the roots of many grasses and a few higher plants. During cool wet periods motile spores (zoospores) are released by the fungus and infect roots of wheat plants. The virus particles are either inside the spores of the fungus (both zoospores and thick-walled resting spores) or tightly bound to the surface of these spores. The fungus normally infects wheat roots shortly after planting in the fall. The symptoms, however, do not normally appear until early spring.

Fig. 4. Rosette stage of soilborne wheat mosaic in the foreground and wheat plants that have escaped infection in the background.

The virus is transmitted from plant to plant by its fungal vector. The zoospores are produced in virus-infected roots or debris and carry the virus. The zoospores swim through the soil solution to healthy root hairs and epidermal cells, penetrate them, and thus inoculate the plant. Once inside the plant, ***P. graminis*** replaces plant cell contents with plasmodial bodies that either segment into additional zoospores or develop into resting spores two to four weeks after infection.

The ***Polymyxa*** fungus survives unfavorable periods in the form of resting spores clustered in the cortical and epidermal cells within plant debris (Figures 5 and 6). These spores can survive in soil for 10 years or more in the absence of wheat or other host plants. The virus and its transmitting fungus are capable of spreading with any movement of infested soil, even wind-blown dust. Because the swimming zoospores of

the fungus transmit the virus, soilborne wheat mosaic is most common and severe in low, wet areas of fields in years when fall rainfall is ample.

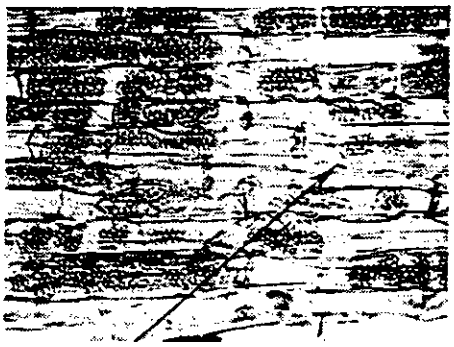

Fig. 5. Clusters of round *Polymyxa graminis* spores in wheat roots. (Courtesy J.E. Watkins, University of Nebraska).

WHEAT YELLOW MOSAIC (WHEAT SPINDLE STREAK MOSAIC)

Wheat yellow mosaic (usually called wheat spindle streak mosaic) is caused by a soilborne virus which also is transmitted by the soilborne fungus, ***Polymyxa graminis***. The virus can survive for 10 years or more in soil in close association with the fungus. Wheat yellow mosaic apparently makes plants resistant to soilborne mosaic virus. The ratio of soilborne wheat mosaic virus to wheat yellow mosaic virus in plants infected with both viruses is about 20:1.

Wheat yellow mosaic was first described in Japan in the early 1960's. At about the same time, the same disease was reported from the eastern United States-Canada border and described as wheat spindle streak mosaic (Ontario soilborne wheat mosaic in Canada). Wheat spindle streak mosaic is now considered to be wheat yellow mosaic virus.

In North America, wheat yellow mosaic is most prevalent near the Great Lakes but occurs over much of southern Ontario and the east-central United States. The disease is found in Illinois in the general area where soilborne wheat mosaic is prevalent. In southwestern Ontario, yield losses occur each year and may reach 40 percent in some fields where very susceptible cultivars are grown. The disease is now known to occur in China, France, Germany, and India.

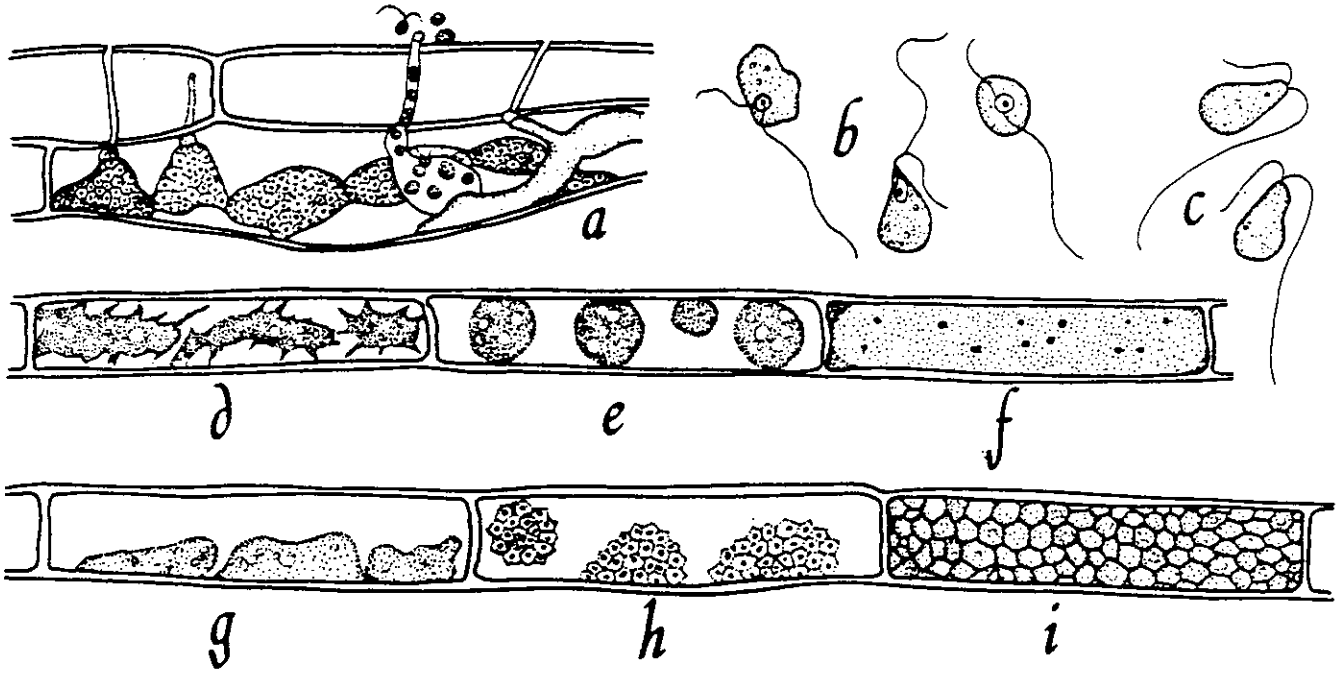

Fig. 6. Life cycle of *Polymyxa graminis*. *(a)* mature sporangia with exit tubes passing through adjacent cells. Motile zoospores are emerging fully formed from one sporangium; *(b)* biflagellate zoospores; *(c)* living amoeboid zoospores; *(d)* amoeboid spores during a period of active growth; *(e)* individual plasmodia (meronts); *(f)* same cell as *d* after the plasmodia have coalesced; *(g)* plasmodia just prior to cleavage into incipient cystosori; *(h)* cleavage of plasmodia into polygonal resting spores (cystosori); *(i)* wheat cell completely filled with resting spores. (Adapted from Ledingham)

The only host for wheat yellow mosaic is wheat, except in Germany, where it has been reported on barley and rye. Like soilborne wheat mosaic, both winter and spring wheats are susceptible to the virus but spring wheats rarely develop symptoms.

VIRUS

Wheat yellow mosaic virus particles are slightly narrower and much longer than those of soilborne wheat mosaic, being 14 to 18 nm wide and normally 200 to 2,000 nm long. The infectious flexuous rods are about 600 nm long and apparently aggregate to form particles up to three microns long. These particles are sparse in host cells of wheat and are difficult to observe in leaf-dip preparations in the electron microscope. They are found scattered and in loose to tight bundles in most epidermal and parenchyma tissues in leaves showing symptoms of wheat yellow mosaic. Infected host cells also contain prominent inclusion bodies which appear as pinwheels and membrane proliferations.

SYMPTOMS

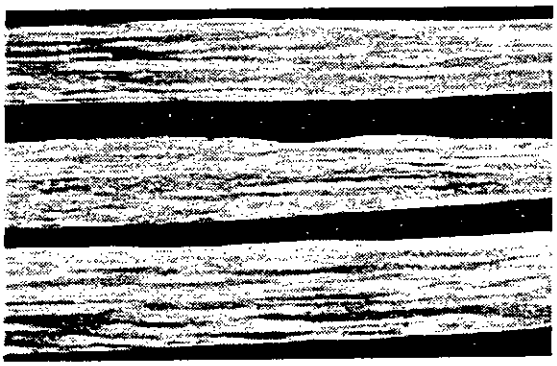

Fig. 7. Three leaves infected with wheat yellow mosaic (wheat spindle streak mosaic). Note the discontinuous steaks and "spindles" oriented parallel with the leaf veins. (Courtesy R.E. Stuckey)

The first leaves produced in early spring develop yellow-green mottling, dashes, and streaks. The discontinuous streaks are oriented parallel with the leaf veins and taper at each end to form yellowish "spindles" (Figure 7). Symptoms are most prominent on the lower leaves because warmer spring temperatures present their development on younger leaves. As the leaves mature and when temperatures remain cool, the center of the spindle may turn brown, streaking may progress to the flag leaf, and the yellow-green areas tend to merge. Reddish streaking and dieback of leaf tips or entire leaves sometimes occurs. Infected wheat plants remain slightly stunted and produce fewer tillers than healthy plants. When warm weather arrives, new symptomless leaves hide the lower leaves showing symptoms. Fewer heads and kernels are produced on infected plants, but kernel weight is not appreciably affected. Cold hardiness is reduced by infection with the virus. The disease tends to be more uniformly distributed throughout fields than soilborne wheat mosaic.

DISEASE CYCLE

There are several similarities between wheat yellow mosaic virus and soilborne wheat mosaic virus. Both viruses are transmitted in nature by the same soilborne fungus, ***Polymyxa graminis***. The two viruses also survive in soil for years in the absence of wheat, apparently in a stable, close association with their fungal vector. Fall infections are most important and account for symptoms produced in early spring. Spring infections may occur but cause no reduction in yield. The two viruses and their vector also are spread by any agency that relocates infested soil.

Infections of wheat yellow mosaic do not occur above 68°F or 20°C, and disease development is checked above 64°F (18°C). The optimal temperature for symptom development is between 41° and 57°F (5° to 13°C). The optimum temperature for virus transmission in the soil is 59°F (15°C). Without prolonged cool temperatures in spring, wheat yellow mosaic is of little importance in Illinois.

CONTROL

1. The planting of highly resistant or tolerant cultivars offers the only practical method of control for both diseases. Some wheats are resistant to one or both viruses, others to the ***Polymyxa*** vector. Most of the soft red winter wheat cultivars recommended for growing in Illinois, as well as a number of hard red winter wheats, are resistant to common strains of the soilborne wheat mosaic virus. Only a few of the cultivars presently recommended are resistant to wheat yellow mosaic (spindle streak mosaic). A listing of cultivars resistant to these two diseases is given in the Illinois Pest Control Handbook which is updated annually and available at your local Cooperative Extension office. There are no known commercial wheat cultivars that are immune to either virus.

2. Late autumn planting, after the Hessian-fly free date, is strongly suggested to reduce losses to these and other wheat diseases. Continuous wheat culture should be avoided.

3. Mosaic-susceptible cultivars may be grown in soil where soilborne wheat mosaic and wheat yellow mosaic viruses with their fungal vector do not occur.

4. Liberal use of fertilizers, based on a soil test, tends to decrease the incidence of these diseases.

Since the two viruses and the fungus that transmits them persist in the soil and crop debris for 10 years or more, crop rotation is of little value in control.

Malcolm C. Shurtleff

Malcolm C. Shurtleff
Extension Plant Pathologist

are easily pulled up. The roots are often brown or black, with water-soaked areas. Later, the roots die and slough-off. Infected crown tissue is bleached, brown or black, and rotted. Following wind and rain, lodging is common and may be severe. The heads are often white and "blasted" or filled with a few shriveled seeds. Disease development and symptoms of the important pathogenic fungi in Illinois are outlined below.

1. COMMON ROOT OR FOOT ROT AND SEEDLING BLIGHT

Common root rot or dryland root rot is caused by one or more unspecialized, widespread soilborne fungi. The more common pathogens include ***Bipolaris sorokiniana*** (synonyms ***Helminthosporium sativum*** and ***H. sorokinianum***, Figure 3), ***Fusarium culmorum***, and ***F. graminearum*** (Figure 4), whose teleomorph or sexual state is ***Gibberella zeae***. Less virulent root and crown rot pathogens include ***Fusarium avenaceum, F. acuminatum, F. oxysporum***, and ***F. equiseti***. Besides causing root rot (Figures 2 and 5), these fungi can attack seedlings, foliage, heads, and developing grain.

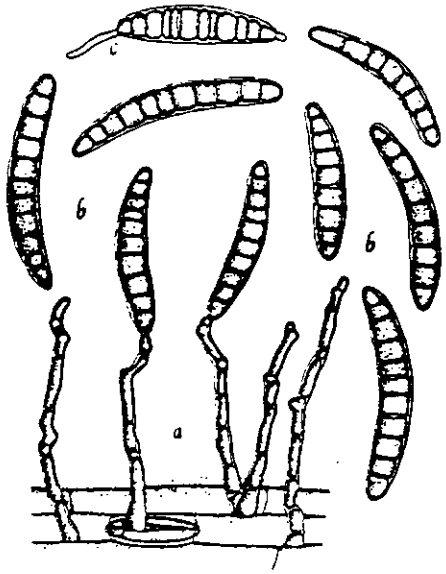

Fig. 3. *Bipolaris sorokiniana* (synonyms *Helminthosporium sativum* and *H. sorokinianum*), the cause of seedling blight, common root or foot rot and spot blotch of cereals and grasses, as it would appear under a high-power microscope: *(a)* conidiophores emerging from a cereal leaf, two bearing conidia at their tips; *(b)* conidia; *(c)* conidium germinating from both end cells. (Drawing by Lenore Gray)

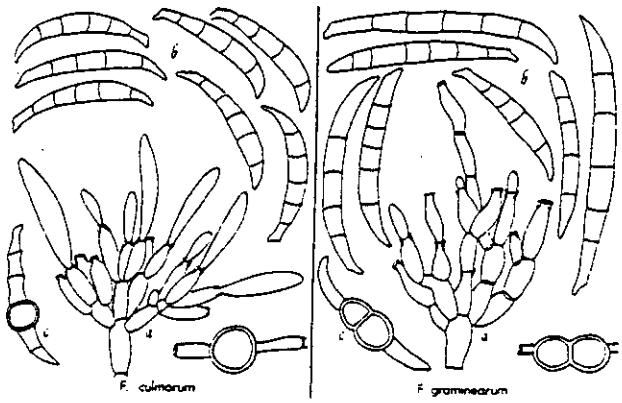

Fig. 4. Two species of *Fusarium (F. culmorum* and *F. graminearum)* that cause common root or foot rot and seedling blight of cereals, as they might appear under a high-power microscope: *(a)* branched monophialides bearing immature macroconidia; *(b)* macroconidia; *(c) left*, intercalary chlamydospore in a macroconidium, *right*, intercalary chlamydospore (or paired) in a hypha. (Drawing by Lenore Gray)

SYMPTOMS

If the fungus (or fungi) is seedborne, seedlings may be killed before or soon after emergence (damping-off) or they may be stunted and off-color with brown lesions on the roots, crown and coleoptile (Figure 1). If infection comes from infested plant debris or the fungi are soilborne, initial symptoms appear as small, brown lesions on the primary or secondary roots, coleoptile, subcrown internode, lower leaf sheaths, and culms. The lesions may elongate, causing severe constriction of the subcrown internode which turns brown to dark brown or nearly black. Infected tissue near the crown may become yellowish (chlorot-

ic) with brown lesions near the leaf base. Diseased plants, which often occur in random patches in a field, are stunted, often have reduced tillering and may prematurely ripen (Figure 2). For Fusarium foot rot, discolored culm tissue may extend two or three internodes (3 to 10 cm) above the soil line (Figure 5). Severely infected plants mature early, produce shriveled grain, appear bronzed or bleached, and form whiteheads. Diagnostic cottony pink mycelium frequently develops within the hollow culms and between the culm and lower leaf sheaths.

Fig. 5. *Left*, wheat culms discolored by Fusarium foot rot; *right*, healthy culms. (Courtesy American Phytopathological Society)

DISEASE CYCLE

The fungi that cause common root and foot rot occur on cereals and numerous wild and cultivated grasses, contaminate seed, and persist indefinitely in soil and plant debris. The ***Fusarium*** fungi overwinter as fruiting bodies (perithecia) and chlamydospores in infested debris. ***F. culmorum*** can survive in soil up to 8 or 9 years without a cereal or grass host. The mycelium, conidia, chlamydospores, and ascospores of ***Fusarium*** spp. are all infectious (Figure 4). ***Bipolaris sorokiniana*** persists between growing seasons as mycelium in crop debris and as thick-walled conidia in soil (Figure 3).

Primary infections from conidia or chlamydospores occur on coleoptiles, subcrown internodes, and less frequently on primary and secondary roots. Moisture stress and warm-to-hot (68°to 86°F or 20 to 30°C) soils, lack of nutrition, deep planting, freezing and insect injuries all predispose cereals to infection by common root rot pathogens. Secondary conidia of ***B. sorokiniana*** form on infected tissue above the soil level and are dispersed by wind and splashing water, causing lesions on the leaves and culms later in the season. Movement of soil by wind, water, and equipment will spread these fungi. Infested seeds also serve to transmit the pathogens over long distances.

2. PYTHIUM ROOT ROT (ROOT NECROSIS OR BROWNING), DAMPING-OFF, AND SEEDLING BLIGHT

There are numerous species of ***Pythium*** which can be found in practically all agricultural soils, parasitizing the roots of cereals, grasses, and higher plants--particularly where soils remain wet for long periods. The more common species, which act singly or in combination, causing Pythium root rot include: ***Pythium aphanidermatum, P. arisosporum, P. arrhenomanes, P. debaryanun, P. graminicola, P. hypogynum, P. irregulare, P. monospermum, P. myriotylum, P. rostratumn, P. splendens, P. tardicrescens,*** and ***P. ultimum***. Infection is difficult to diagnose, as cereals and grasses can be thinned and stunted without obvious symptoms of disease. The diseased plants tend to be more uniformly distributed in a field than with other root and crown rots (Figure 6).

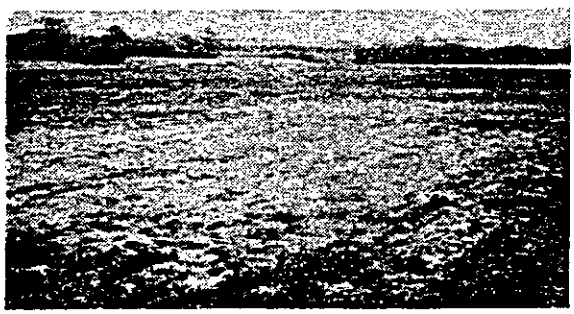

Fig. 6. A wheat field severely infected with Pythium root rot. (Courtesy British Ministry of Agriculture)

SYMPTOMS

Severe damage by ***Pythium*** species results in missing, stunted, off-color or poorly tillered plants. The first true leaf of seedlings is often stunted. Adult plants may appear stunted, light green to chlorotic, and nitrogen-deficient. Heading and maturity are often delayed with heads small and poorly filled. Infected roots have soft and wet yellow-brown to brown root tips, which lack root hairs, and tan to light brown lateral roots.

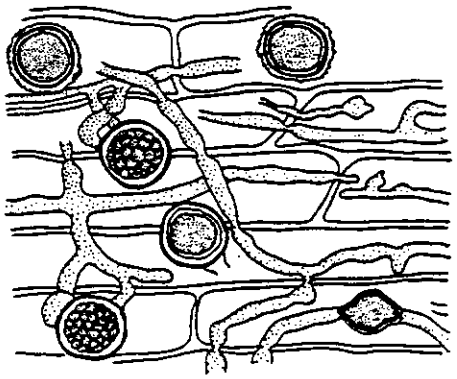

Fig. 7. A species of *Pythium* within wheat cells, as it might appear under a high-power microscope, showing the nonseptate branching hyphae, round oospores and an intercalary chlamydospore. (Drawing by Lenore Gray)

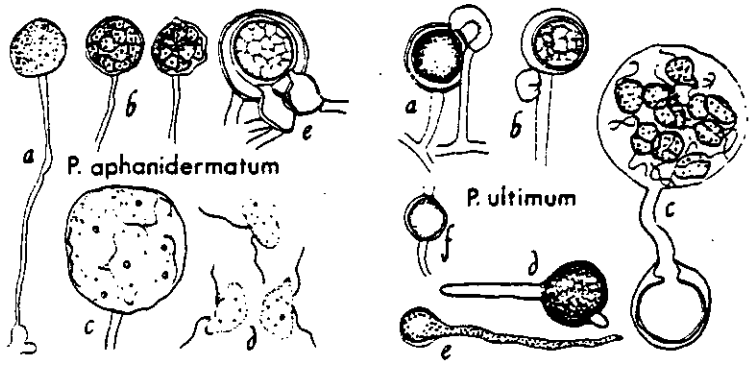

Fig. 8. Two *Pythium* species which commonly cause damping-off, seedling blight and root rot of small grains and many other plants, as they would appear under a high-power microscope. *Left, P. aphanidermatum*: *(a)* immature sporangium, *(b)* two mature sporangia prior to release of zoospores, *(c)* mature sporangium with several zoospores which are released in a mass from the sporangium, *(d)* motile zoospores, *(e)* oospore with two paragynous antheridia attached. *Right, P. ultimum*: *(a)* oogonium with a single paragynous antheridium attached from a nearby hypha, *(b)* oogonium with a single paragynous antheridium attached originating from the antheridial stalk, *(c)* oospore germinating by a germ tube which has formed a sporangium containing zoospores, *(d)* sporangium germinating with two germ tubes, *(e)* oospore germinating with a germ tube, *(f)* intercalary sporangium. (Drawing by Lenore Gray)

DISEASE CYCLE

Pythium species are mainly found in the top 5 to 7 inches of soil where crop residues serve as a source of nutrients and moisture. The fungi survive as thick-walled oospores and chlamydospores (Figures 7 and 8) in infested plant residue and can persist for 5 years or more in soil or embedded in plant refuse. In moist soil, oospores germinate directly or indirectly by forming 10 to 40 motile zoospores, which swim to root tips where infection occurs within 3 to 5 hours after the oospores germinate (Figure 8). Initial infections often begin in the embryos of germinating cereal seeds within 48 hours after planting. New roots, especially the root tips of fine lateral roots and root hairs, become infected. New oospores form in parasitized roots, and the fungi also grow on clean straw, chaff, and debris not colonized by other fungi and left on or close to the soil surface. Infection and growth of ***Pythium*** species are optimal between 59° and 86°F (15-30°C) in wet, compacted soils deficient in phosphorus and cropped previously to cereals and grasses.

3. RHIZOCTONIA ROOT ROT, SEEDLING BLIGHT, AND SHARP EYESPOT

Rhizoctonia root rot, caused by ***R. solani*** and ***R. cerealis***, occurs any time during the growing season on all cereals and a wide range of higher plants throughout the world. Like many other root diseases, it often goes unnoticed unless the roots, and especially the root tips, are carefully washed and examined under a microscope. The fungus is most active in the top 5 to 7 inches of soil where

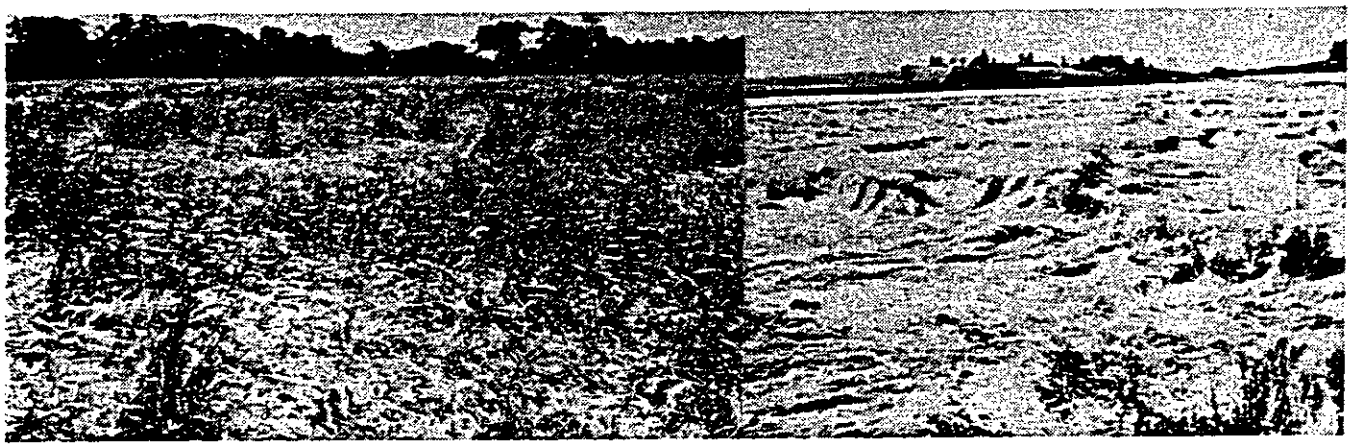

Fig. 12. Severe foot rot or strawbreaker in two wheat fields. It is typical for cereal plants to lodge in all directions. (*Left*, courtesy W.G. Willis)

are distributed principally by splashing rain. Symptoms commonly do not appear for several weeks after infection has occurred and autumn infections may not be evident until spring. Crops of conidia form on new lesions within 1 to 3 months and result in secondary lesions that may provide inoculum for succeeding small grain or grass crops. Mild winters and cool, wet springs prolong the production of conidia and infection periods. The fungus persists in soil or crop residue for several years in the absence of small grain crops.

5. TAKE-ALL ROOT AND CROWN (FOOT) ROT

Take-all is a widespread root, crown and foot rot of winter cereals and numerous grasses caused by the soilborne fungus ***Gaeumannomyces graminis*** (synonym ***Ophiobolus graminis***). The disease is most important in the Midwest where one cereal crop is grown continuously for 3 to 6 years, the soil pH is neutral or alkaline, the soil is sandy or infertile (especially deficient in nitrogen and phosphorus), compacted and poorly drained, and moisture is plentiful. The loss from take-all depends on when and how severely the roots and culm bases are colonized. The earliest infections are the most severe. When symptoms become obvious, yields may be reduced by 50 percent. Take-all is somewhat more damaging to winter cereals than to spring cereals, and where reduced tillage is practiced. Early, wet springs favor disease infection and development.

SYMPTOMS

When the weather is relatively moist, symptoms of take-all appear about heading time. In localized areas of a field, severely infected plants are stunted, turn pale green, ripen and die prematurely, and develop sterile whiteheads or heads containing a few shriveled kernels (Figure 13). When the weather is wet before harvest, the leaves, culms, and heads of affected plants become "sooty" from the growth and spore production of secondary molds (such as species of

Fig. 13. An area of wheat in the foreground severely affected with take-all disease.

Fig. 14. Culms of wheat *(left)* and rye *(right)* with shiny plates of coal-black mycelium under the lowest leaf sheath, a characteristic sign of the take-all disease fungus. (*Left*, courtesy T.M. Sjulin; *right*, courtesy Clemson University and the U.S. Department of Agriculture)

Alternaria, Cladosporium, Epicoccum, Stemphylium, and ***Sporobolomyces***). Plants at an earlier stage are stunted to severely dwarfed, somewhat yellow and have fewer tillers. Diseased plants are easily pulled up or break off near the soil line. The roots are sparse, blackened and brittle. The black-brown rot commonly extends into the subcrown internode, crown and culm bases where a diagnostic, superficial, shiny plate of coal-black mycelium covers the culms UNDER the lowest leaf sheath (Figure 14). Coarse "runner" hyphae of the fungus often form black strands several milimeters long on the surface of the roots (Figure 15). Under prolonged moist conditions the mycelial plate may be speckled with black fruiting bodies (perithecia) which erupt through leaf sheaths. Diseased culms, weakened at the base, lean or lodge in all directions.

DISEASE CYCLE

The causal fungus overseasons on infected roots of living cereal or grass plants and in or on host debris. Roots become infected as they grow through soil near infested debris. The roots first become colonized by runner hyphae (Figure 15). Infection by hyphae, and to a lesser degree by ascospores produced in perithecia (Figure 16), occurs throughout the growing season, with an optimum between 50° and 68°F (10 to 20°C). Root infections and colonization in autumn or early spring are most likely to produce a crown (foot) rot.

Most plant-to-plant spread occurs through black runner hyphae advancing across "root bridges." The fungus is widely dispersed by any agency that moves infested soil and host debris. Ascospores are spread by splashing rain and to some extent by wind.

Take-all is favored by alkaline, nitrogen- and phosphorus-deficient soils that remain cool and wet for prolonged periods.

6. ANTHRACNOSE

This worldwide disease of cereals and many grasses is caused by ***Colletotrichum graminicola***, teleomorph ***Glomerella graminicola***. It is only damaging on small grains that are nutritionally stressed, grown in alkaline or sandy soils, and grown under reduced tillage. Anthracnose is a much more serious disease on corn and sorghum than on small grains.

Fig. 15. Black "runner" hyphal strands on the surface of a wheat root, a characteristic sign of the take-all disease fungus. (Courtesy R.W. Smiley)

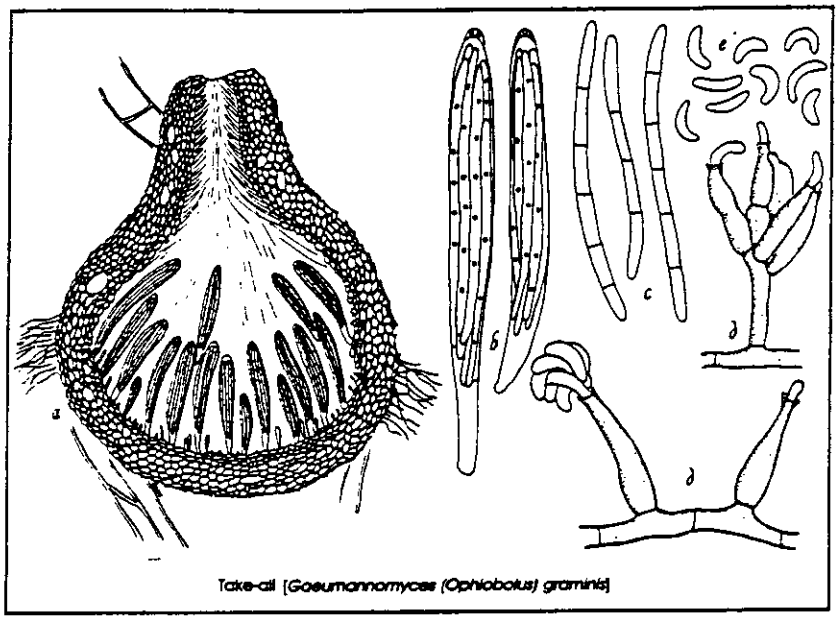

Fig. 16. *Gaeumannomyces (Ophiobolus) graminis*, anamorph *Phialophora* sp., the cause of take-all disease of cereals and grasses, as it might appear under a high-power microscope: *(a)* vertical section through an erumpent perithecium containing club-shaped asci (*Gaeumannomyces* state); *(b)* two asci, each containing eight elongate ascospores; *(c)* three septate ascospores; *(d)* conidiophores (phialides), some bearing immature conidia (*Phialophora* state); *(e)* conidia or phialospores. (Drawing by Lenore Gray)

Fig. 17. Wheat anthracnose caused by *Colletotrichum graminicola*. Discoloration of culm nodes is characteristic of the disease, as is the presence of black acervuli on the culms, sheaths, and glumes. Severely infected wheat plants ripen prematurely. (Courtesy Illinois Natural History Survey)

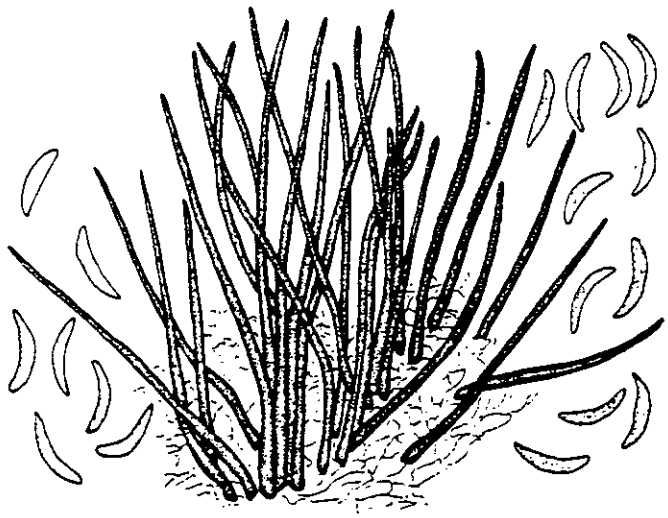

Fig. 18. An acervulus of the anthracnose fungus *Colletotrichum graminicola*, as it might look under a high-power microscope. The dark "spines" are setae, and curved "early moons" are one-celled conidia. (Drawing by Lenore Gray)

SYMPTOMS

Roughly elliptical lesions, 1 to 2 cm long, occur on roots, crowns and culms. The lesions are first water-soaked, then bleached, and later turn brown. At host maturity they are elongated and covered with black fruiting bodies (acervuli; Figure 17) containing clumps of dark spines (setae; Figure 18) visible with a hand lens or reading glass. On crowns and culms the lesions may resemble eyespot or sharp eyespot until the dark acervuli appear. Anthracnose normally is confined to the lower half of culms. When severe, the disease may cause a reduction in vigor, lodging, premature ripening, whitening of heads, and lightweight shriveled grain (Figure 17).

DISEASE CYCLE

The ***Colletotrichum*** fungus overseasons in living cereal or grass plants, on host residues, seed, and soil as mycelium and conidia produced in acervuli (Figure 18). The conidia are dispersed by wind and rain. They germinate in water films and penetrate root, crown and basal culm tissues directly. Infected leaf tissue and/or heads are the result of secondary inoculum. Weed grasses and continuous growing of small grains promote the development and overwintering of the anthracnose fungus. Prolonged, warm (optimum 17°F or 25°C) wet weather favors infection and the production of acervuli.

7. MINOR ROOT ROT FUNGI

There are a number of other fungi associated with darkened and rotted cereal and grass roots (root necrosis) in Illinois. They usually go unnoticed since they are not serious pathogens and a microscopic examination is usually needed to observe them. Most can be found in plants that appear healthy as well as in plants with symptoms of disease. These widespread, soilborne fungi include ***Microdochium bolleyi*** (synonyms ***Aureobasidium bolleyi*** and ***Gloeosporium bolleyi***), ***Naucoria cerealis***, ***Marasmius graminum*** (synonym ***M. tritici***), ***Periconia circinata***, ***Agrocybe molesta*** (synonyms ***A. dura*** and Pholiota dura), ***Polymyxa graminis*** (vector of soilborne wheat mosaic and wheat yellow mosaic [spindle streak mosaic] viruses), ***Phialophora graminicola*** (synonym ***P. radicicola*** var. ***graminicola***), ***Olphidium brassicae***, and ***Phaeosphaeria herpotrichoides***.

CONTROL

No single practice will control root and crown rots. When carried out collectively, though, the measures outlined below will keep losses to a minimum.

1. SOW THOROUGHLY CLEANED, CERTIFIED, PLUMP, DISEASE- AND CRACK-FREE SEED OF ADAPTED CULTIVARS. Use the ones recommended for your area by University of Illinois Extension Agronomists and your local Cooperative Extension Service Adviser. The seed should be treated with a recommended fungicide before planting. For details see the latest revision of Report on Plant Diseases No. 1001, "Seed Treatments for Field Crops," and the current edition of the Illinois Pest Control Manual.

2. WHERE PRACTICAL, PLANT RESISTANT OR TOLERANT CULTIVARS. A number of small grain cultivars are resistant or tolerant to one or more root- and crown-rotting diseases. See the current editions of the Agronomy handbook and the Illinois Pest Control Manual for additional information.

3. PLANT IN A FERTILE, WELL-PREPARED, WELL-DRAINED SEEDBED AFTER THE HESSIAN FLY-FREE DATE AT THE TIME RECOMMENDED FOR YOUR AREA. Plant no deeper than 2 inches.

4. MAINTAIN AN ADEQUATE, BALANCED SOIL FERTILITY. An optimum supply of available phosphorous, potassium, and nitrogen--based on a soil test--must be provided. Avoid high rates of nitrogen. Research has shown that ammonium and slow-release forms of nitrogen (anhydrous ammonia or urea) better suppresses take-all root rot, compared to the nitrate forms of nitrogen.

5. ROTATE SMALL GRAINS WITH NONGRASS CROPS, preferably legumes (soybeans and forage legumes), corn, sorghum, or canola for 3 or 4 years.

6. WHERE FEASIBLE, IF THERE IS NO LEGUME UNDERSEEDING, CHISEL PLOW OR PLOW DOWN CEREAL STUBBLE DEEP AND CLEAN BEFORE PLANTING. In cultivated fields, keep down volunteer small grains and grassy weeds, such as foxtails, quackgrass, cheat, wild rye and barley, bottle-brush grass, windmill grass, and fescues.

7. DO NOT SPREAD MANURE CONTAINING INFESTED STRAW AND CORN STALKS ON FIELDS THAT WILL BE PLANTED IN SMALL GRAINS OR GRASSES WITHIN THE NEXT YEAR OR TWO.

Malcolm C. Shurtleff

Malcolm C. Shurtleff
Extension Plant Pathologist

CONTROL

1. Propagate only with cuttings taken from disease-free stock plants or purchase only certified, disease-free cuttings and plants from a reputable commercial propagator.

2. Plant only in pasteurized soil. For details and precautions refer to University of Illinois Cooperative Extension Circular 1213, Soil Disinfestation: Methods and Materials.

3. Keep cuttings and potted plants widely spaced in a greenhouse free of plants infected with bacterial leaf spot.

4. Practice only surface or bottom watering. Avoid splashing water on the foliage. Avoid syringing the plants lightly with water, especially at high temperatures. If overhead watering is necessary, water in the morning on a rising temperature so the foliage will dry quickly.

5. Keep the humidity in the greenhouse at 85 percent or lower by increasing air circulation and adding heat as temperatures drop to prevent moisture condensing on the foliage. Keep the temperature as low as practical for good plant growth.

6. Spotted leaves should be carefully removed, placed in a plastic bag, and burned. Disinfect cutting knives with 70 percent rubbing alcohol before using on other plants. Remove and destroy severely infected plants together with the soil attached to the roots.

7. If disease is seen, carefully remove slightly infected plants and quarantine them in another greenhouse.

8. The spread of the disease can be prevented provided the cultural practices outlined above (1-7) are routinely practiced. Copper fungicides have given some control when plants are sprayed thoroughly and repeatedly. For details refer to University of Illinois Cooperative Extension Circular 1259, Plant Disease Control Guide: Flowers and Nonwoody Ornamentals (revised annually). Avoid unnecessary pesticide applications which may spread the bacteria from plant to plant.

9. There are no known highly resistant begonia cultivars.

The publications mentioned above should be available at your county Extension office.

Malcolm C. Shurtleff

Malcolm C. Shurtleff
Extension Plant Pathologist

Cooperative Extension Service

PLANT DISEASES

UNIVERSITY OF ILLINOIS AT URBANA-CHAMPAIGN

RPD No. 653
4/88

PEPEROMIA DISEASES

Malcolm C. Shurtleff and Jeanne M. Scheller

Peperomias (*Peperomia* spp.) are popular foliage pot plants that are subject to only a few diseases. Fortunately, these problems can be fairly easily controlled. The most common diseases are ring spot, oedema, Phytophthora rot, and cutting rot.

1. Ring spot

Peperomia ring spot is caused by a virus that is commonly transmitted by taking cuttings from apparently healthy but infected plants. The disease affects *Peperomia obtusifolia* and *P. obtusifolia* var. *variegata*.

Symptoms

Peperomia ring spot appears as concentric, brown, necrotic ring markings that disfigure the leaves (Figure 1). The young leaves on certain plants may be markedly cupped, curled, or twisted. On some plants only the older leaves are affected. Severely diseased plants may be stunted. Each ring spot starts as a small, translucent spot that enlarges by the outward addition of a number of narrow bands or lines. Some lines are light and translucent while others are brown and opaque. On the upper leaf surface the tissue over the brown lines often becomes sunken, forming narrow furrows or grooves. The area covered by the ring spot is lighter colored than the healthy portion of the leaf. When one or a few ring spots occur on a leaf, the outlines of the spots and ring patterns are regular. If many spots occur close together, they merge with their outlines and the rings form irregular patterns (Figure 1).

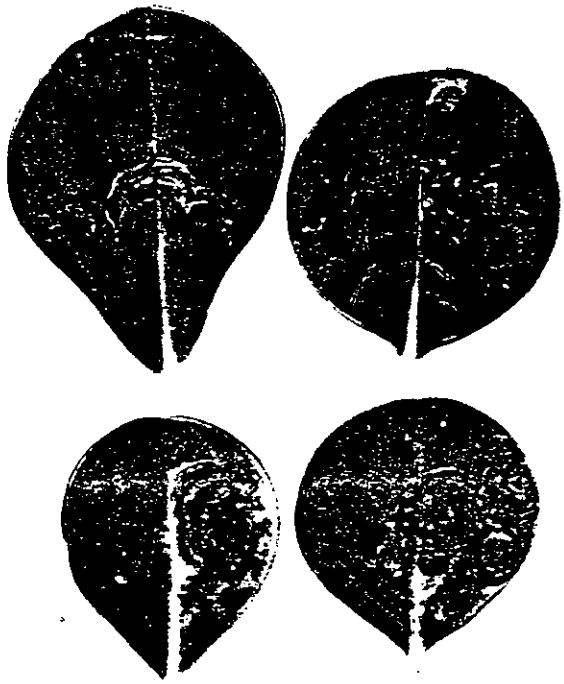

Figure 1. Ring spot of peperomia. Note the concentric, sunken dead areas (Illinois Natural History Survey photograph).

M.C. Shurtleff is Extension Plant Pathologist, Department of Plant Pathology, and J.M. Scheller is a former student in plant pathology, both University of Illinois at Urbana-Champaign.