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Diagnosis of Turfgrass Diseases: The Art and the Science

by Eric B. Nelson

Diseases are perhaps the most unusual and perplexing of the pest problems affecting highly managed turf. As a result, diagnosing problems that may be caused by disease represents one of the more challenging and frustrating exercises in turfgrass management.

Both weed and insect pests can be readily seen with the unaided eye. Insects or weeds, regardless of their stage of development, look much the same in any environment. In addition, being readily visible, they can be matched in appearance with the diagrams and photographs presented in books and other diagnostic reference aids.

Diseases, on the other hand, are caused by a wide variety of microscopic organisms, none of which is observable with the naked eye. And the activities of these pathogens can be seen only indirectly, by observing the responses of the turfgrass plants they have infected.

Diagnosis is complicated further by differences in the symptoms of infection by a particular pathogen, depending on factors such as the species of grass involved, the height of cut, local environmental conditions, or the presence of other pests and pathogens. Chemical, physical and biological stresses also affect the expression of symptoms.

Disease diagnosis can be thought of as a process of elimination, in which the range of potential causes for the observed problem is carefully reduced to one. The sequence of steps one follows in diagnosing turfgrass diseases is designed to assemble evidence for and against possible causes for the observed problem. It is important, therefore, that turfgrass managers maintain accurate and complete records of both site management activities and the season's weather. Combining these two data sets with careful observation of the turfgrass symptoms and examination of pathogen structures permits identifying associations between the disease and a causal agent.

Because they are perennial plants, turfgrasses develop long-term associations with pathogens. In fact, in nearly all mature turfgrass plantings, individual plants are continuously infected with many, if not all, of the pathogens capable of causing disease in that particular grass species. This is why symptoms of many turfgrass diseases are detected most often when turfgrass plants are under stress. Additionally, the presence of many turfgrass pathogens in a single sample complicates disease diagnosis by making it difficult to reduce the probable causes of the symptoms observed to a single agent.

*Photographs provided by Eric B. Nelson

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Considerable training and experience are required to overcome these difficulties and make competent disease diagnoses. Turfgrass managers therefore often turn to academic experts for assistance in the diagnostic process. This couples the manager's expertise in the field with the academic's expertise in the laboratory, facilitating both the determination of the cause of the problem and the selection and implementation of appropriate control measures.

Importance of correct disease diagnosis

Proper diagnosis is central to any successful turfgrass disease management program. There are several reasons for this. First, identifying the cause of any problem helps the turfgrass manager identify some of the conditions that may have fostered its development. In a sense, the diagnosis of disease itself helps the turfgrass manager understand more about the biology of the causal agents, and how to limit their impact.

Second, many contemporary disease control strategies are quite specific, being effective against one disease, ineffective against others, and potentially making still other diseases much worse. For example, the action of fungicides can be very narrow, affecting only certain groups of fungal pathogens. Inaccurate diagnoses could thus lead to the application of unneeded fungicides, which could have damaging side effects.

Third, the successful adaptation of Integrated Pest Management (IPM) scouting and monitoring protocols to the situation at hand depends on accurate identification of turfgrass pest problems. Identification is easily accomplished for weed and insect pests, but is problematic for diseases. Accurate diagnosis gives turfgrass managers the ability to map and measure specific disease problems so more effective control strategies can be developed and implemented. It also gives them the ability to gauge the effectiveness of control measures and aids in the prediction of future disease outbreaks.

Basic analytical processes in disease diagnosis

All disease diagnostic procedures follow a logical sequence of steps, designed to gather enough evidence to exclude potential causal agents from consideration. Both field and laboratory observations contribute to this process.

One of the first challenges to any turfgrass manager is to determine whether the observed problem in question is actually the result of disease. Disease symptoms often resemble damage from noninfectious agents (insects, for instance) or from a variety of abiotic problems (such as localized dry spots).

Sometimes, the characteristics of the damage can provide clues. The patch-like appearance of symptoms, usually easier to see on close-cut than on higher-cut turf, may be indicative of a disease problem. Most known turfgrass diseases are caused

Steps in Disease Diagnosis

In the field

1. Identify affected grass species and cultivars
2. Observe symptoms over the entire affected area
3. Observe specific plant symptoms
4. Make field observations of pathogen structures in turfgrass tissues
5. Record the cultural and environmental conditions
6. Attempt an initial diagnosis
7. Collect and submit samples for clinical diagnosis

In the lab

8. Laboratory examination of turfgrass samples
9. Pulling it all together into a final diagnosis

In the field

10. Select an appropriate management strategy

by fungi. Since fungi tend to grow outwards from the site of infection, many fungal pathogens cause circular, patch-like symptoms in turfgrass plantings. There are several turfgrass diseases, however, that do not typically induce patch symptoms. Symptoms of these diseases are commonly confused with signs of other turfgrass problems. The following are the steps one normally would go through in diagnosing these and other turfgrass diseases.

Step 1. Identify affected grass species and cultivars

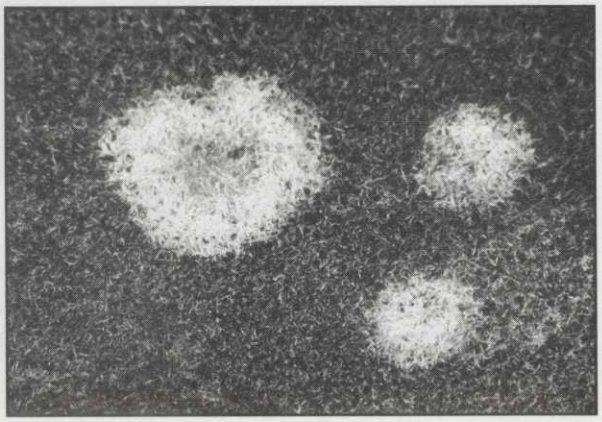
One of the first steps in any diagnosis is to determine which plants are affected. A number of turfgrass pathogens are relatively specific to particular turfgrass species. Some are even specific to individual turfgrass cultivars within a species. For example, take-all patch caused by *Gaeumannomyces graminis* var. *avenae* is generally found on creeping bentgrasses, but not on other turfgrass species. Similarly, summer patch disease caused by *Magnaporthe poae* is found on bluegrasses and fine fescues, but rarely, if ever, on perennial ryegrass varieties. Even within a turfgrass species, cultivars can vary in response to diseases. For example, varieties of Kentucky bluegrass such as Bristol, Eclipse and Glade are relatively tolerant of summer patch disease, to which varieties such as Chateau and Fylking are quite susceptible.

On golf courses where mixed stands of annual bluegrass and varieties of creeping bentgrass are common, the annual bluegrass tends to be affected more severely, or shows symptoms much earlier, than the creeping bentgrass variety. This can apply to root and crown diseases such as Pythium root rot caused by *Pythium graminicola,* anthracnose caused by *Colletotrichum graminicola,* and some nematode problems.

Turf affected in low-lying areas is suggestive of pathogens favored by wet conditions.

Symptoms occurring adjacent to concrete or pavement, which can raise soil temperatures, may indicate a disease favored by heat stress.

Areas experiencing heavy equipment traffic often show symptoms in regular patterns.



Patch-type symptoms are usually indicative of fungal diseases.

Step 2. Observe the entire affected area for symptoms

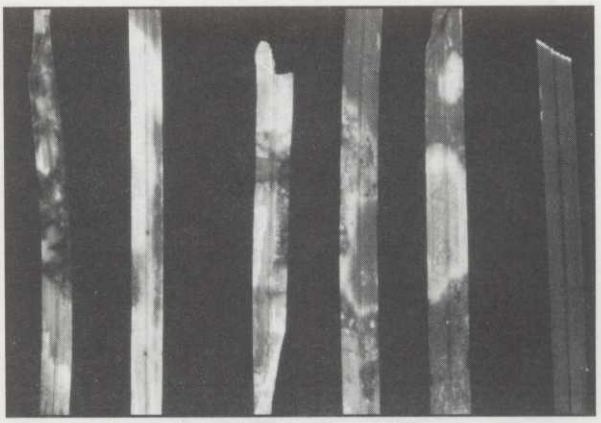
The location of symptoms in the affected area can reveal important information about the nature and distribution of the disease, since certain pathogens are usually associated with certain distribution patterns. For example, it is useful to know whether symptoms are restricted to wet, low-lying areas, or to high, dry areas. It would also be noteworthy if symptoms were limited to areas of intense foot or equipment traffic, or to areas of extreme soil compaction. Other important factors affecting symptom distribution are soil characteristics (such as texture and pH), the degree of shade, and the proximity of structures such as buildings, roads and sidewalks that may alter soil temperatures.

How symptoms appear can also reveal important information about diseases. For example, it is important to note whether the symptoms appear at random throughout the affected area, or are localized in discernible structures. Rather than appearing diffused throughout susceptible turfgrasses, some diseases usually appear as rings or patches. Root and crown diseases, for instance, generally give rise to more patch- or ring-like symptoms; foliar diseases, on the other hand, tend to result in more diffuse symptoms.

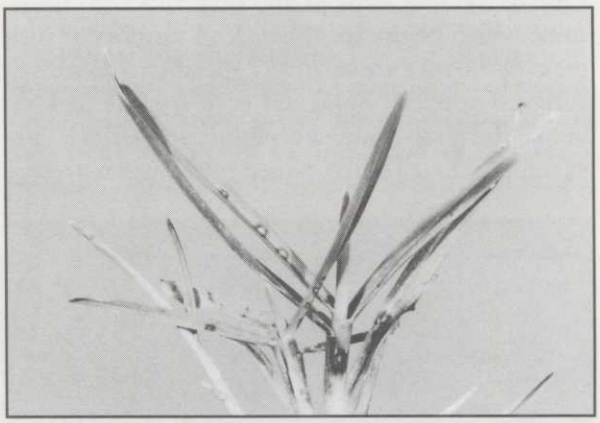
Disease symptoms that appear patch-like on close-cut turf may seem diffuse on higher-cut turf. Conversely, foliar diseases such as dollar spot and red thread may actually appear patch-like on both high-cut and close-cut turf.

Examining the whole set of symptoms at this stage of the diagnostic process will help to sort out whether the problem being observed is biotic or abiotic in origin. For example, if symptoms appear in a highly regular pattern, this may indicate a problem caused by maintenance equipment. An example would be the movement of equipment over heat-stressed turf. Overapplication of fertilizers or pesticides can also produce regular patterns. Examining turf at this stage might also reveal the presence of other noninfectious biotic factors such as insects, algae or moss that might be contributing to the observed problem.

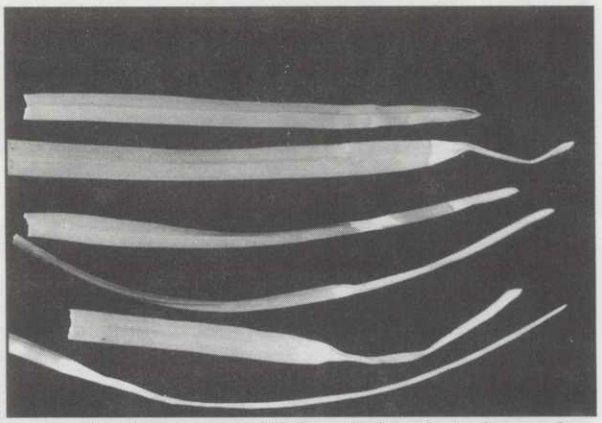
Often, when assessing symptoms, it is difficult to determine whether the problem under examination is currently active and worsening or has been inactive and stabilized for some time. This is particularly true of diseases such as red thread caused by *Laetisaria fuciformis*. On perennial ryegrasses and fine-leafed fescues, necrotic patches from red



Fungal pathogens often produce irregular leaf lesions.



Leaf-spotting pathogens tend to cause well defined, readily recognized lesions.



Some leaf pathogens cause leaf blades to die from the tip downward, producing tip blight.

thread can often be seen long after the pathogen has ceased to be active. Generally, the only way to tell whether the pathogen is still active is to get down on hands and knees and examine the turfgrass plants closely for the presence of progressive symptoms or pathogen structures. In the case of foliar diseases, the mycelium or other structures are sometimes visible when the pathogen is active or has recently been active. Where disease is concerned, fungal activity is difficult to assess.

Step 3. Observe specific plant symptoms

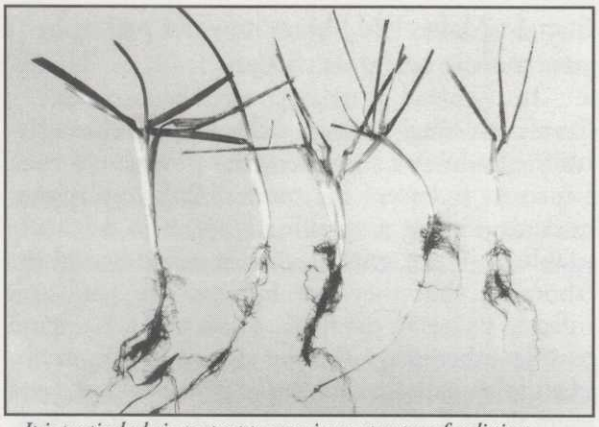
Specific symptoms on individual plants can provide still more information on the possible causes of disease problems. They are also one of the most important diagnostic features available for some diseases. The principal above-ground symptoms to look for are leaf spots. Pay particular attention to the appearance of the lesions. It is important, for example, to determine whether the lesions are irregular in shape or circular, and whether they have a yellow (i.e., chlorotic) halo or a purplish or brownish area on their borders.

Blighting, in which the plant, particularly the leaves, turns brown (i.e., necrotic), is another above-ground symptom to look for. With leaves, for example, it is important to note whether they appear to be blighting from the tip down, or from the basal stem upward.

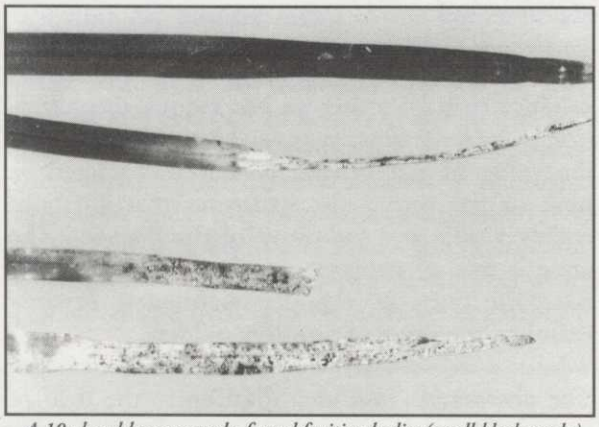
Other commonly observed above-ground symptoms include wilting, stunting and rotting. More specific observations about these symptoms should include such things as whether, during wilting or rotting, the plants appear dry or wet and greasy.

In addition to the character of specific plant symptoms, it is important to note which individual plant parts are affected. Blighting symptoms may appear on leaf blades or sheaths, for example. Rots may appear on sheaths, roots and rhizomes. Rotting symptoms are found most often on below-ground plant parts.

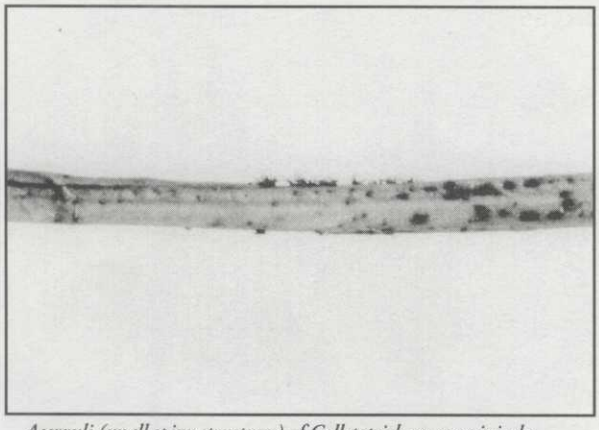
Examination of the root system deserves equal attention in disease diagnosis, noting abnormalities such as discolorations, deformations, distinct lesions and absence of root hairs. When removing individual plants for root inspections, it is extremely important to keep as much of the root system as possible intact. The article on root and crown disease diagnosis in this issue of *TurfGrass TRENDS* provides additional information on this important aspect of the diagnostic process.



It is particularly important to examine root systems for distinct lesions, the absence of root hairs, or non-specific root and crown rotting.



A 10x hand lens can make fungal fruiting bodies (small black specks) visible in diseased tissues.



Acervuli (small spiny structures) of Colletotrichum graminicola observed on partially living tissue are particularly diagnostic for Anthracnose.

Step 4. Make field observations of pathogen structures in turfgrass tissues

Since most fungi are identifiable by their characteristic reproductive structures, the presence of such structures is one of the more definitive pieces of evidence linking a specific pathogen to a specific problem. These structures are observed best in the laboratory, but they can be seen frequently on infected tissues in the field. The use of a 10x hand lens or other magnification device is a must for identifying pathogen structures in the field. A good example of a disease for which diagnostic reproductive structures may be seen under low magnification is anthracnose, caused by *Colletotrichum graminicola.*

During disease development, some pathogens produce structures that do not require magnification to be seen. For example, *Laetisaria fuciformis,* the cause of red thread, produces characteristic pink to red thread-like structures that are both readily visible and indicative of the disease. The same is true of pathogens such as *Typhula incarnata,* the cause of Typhula blight, and *Erysiphe graminis,* the cause of powdery mildew.

The observation and identification in the field of pathogen structures can accelerate the diagnostic process significantly. Since these structures tend to be short-lived, their presence can indicate how recently the pathogen has been active.

Step 5. Record the cultural and environmental conditions

Recording the cultural conditions before and during the onset of disease symptoms is an important part of the evidence-gathering process. The same holds true for the environmental and weather conditions immediately preceding the onset of symptoms. Both can quickly eliminate certain possible pathogens from consideration as causal agents.

Among the cultural conditions it is important to record are the age of the turfgrass planting and the specific fertilization, irrigation and pest control practices employed (including materials and amounts applied). Grooming and growth management practices should be noted as well; so should any peculiarities such as increased traffic, excessive thatch, unusual soil odors and the like. Where appropriate, unusual features of the landscape should be noted. These might include the presence of large trees or roots in and around the affected site, shading, air and water drainage and soil pH.

The important items of weather information to record are: maximum and minimum daily temperatures, relative humidity, rainfall, degree of cloud cover and wind speed. Obviously, the most appropriate weather data would be those collected at the affected site. National Weather Service data can also be used if a recording station is located close enough to the site to provide representative readings.

Step 6. Attempt an initial diagnosis

Once all the pertinent field information has been gathered, a tentative diagnosis is in order. Numerous guides have been written to aid in the diagnosis of turfgrass diseases. Disease identification manuals may be available from the state's land grant universities. Similar manuals may be available from pesticide and fertilizer manufacturers, the federal government, private consultants, professional turfgrass associations and scientific societies. There are also textbooks devoted exclusively to turfgrass diseases (a list of 10 accompanies this article). If no clear diagnosis can be reached after making observations, examining the cultural and environmental data and consulting the manuals, then the next step in the process is to enlist the help of a competent laboratory diagnostician.

Step 7. Collect and submit samples for clinical diagnosis

To ensure that the laboratory diagnostician has all the information required to make an accurate diagnosis, it is important to collect a proper sample and send it along with the appropriate field observations. Turfgrass samples with apparent aboveground or below-ground symptoms should be collected as early as possible after the onset of the disease, preferably as the problem is on the increase. Samples collected long after the problem was first noted can be difficult to diagnose accurately.

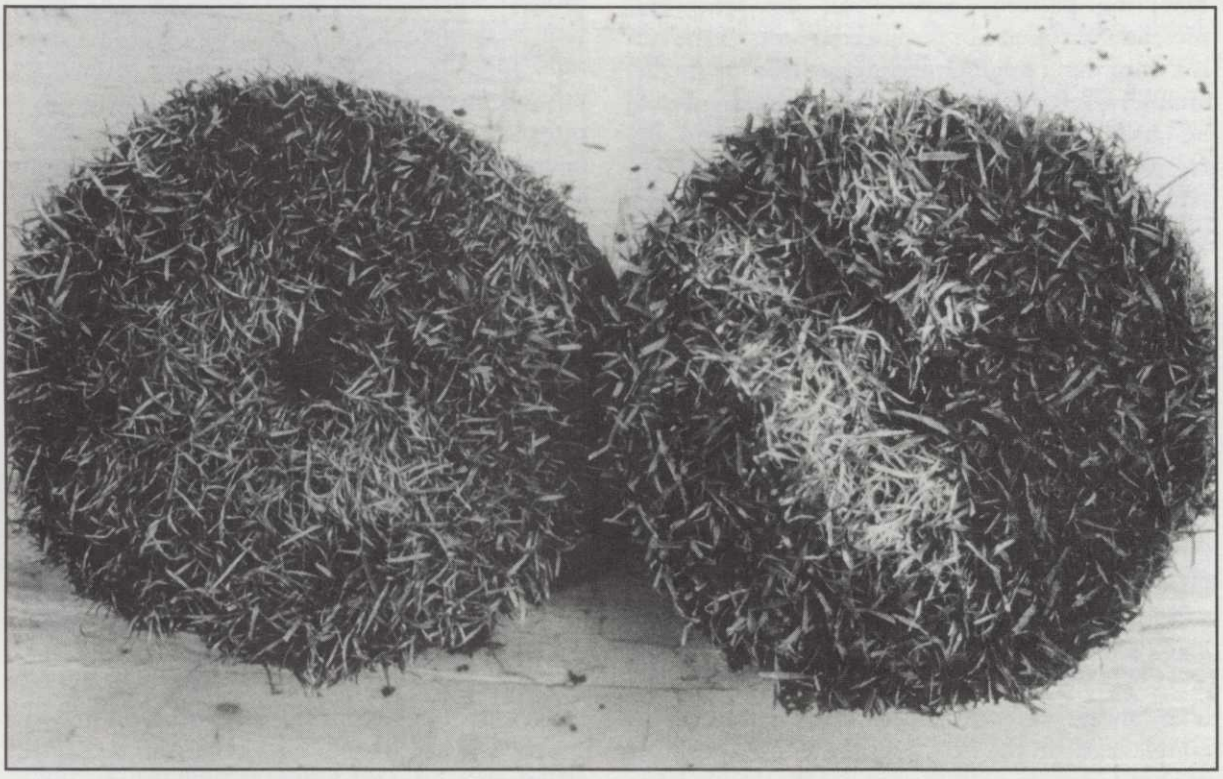
The samples collected should be representative of the symptoms observed over the entire affected area. Since the clinical diagnostician does not have

the luxury of observing the problem first hand in the field, it is critical that the sample be accompanied by an adequate description of the problem, a record of the cultural practices and a description of the environmental and weather conditions that were present at the time the problem was first observed. The following critical information should be included with the sample:

- The grass species. If known, the precise cultivars.
- A description of the overall symptoms, the date they first appeared and the extent of the affected area. Be specific about symptom location and appearance.
- A description of the cultural conditions before and during the onset of symptoms.
- A description of the weather conditions before and during the onset of symptoms.
- If possible, a photograph of the affected area (Polaroid is fine).

To facilitate comparison in the laboratory, samples should be collected both from the turf showing symptoms and from apparently healthy turf. In addition, samples should not be collected shortly after a fungicide has been applied. Generally, if the fungicide is effective against the suspected pathogen, it will have done its work before the sample can be analyzed, making meaningful diagnosis impossible.

If symptoms are patch-like, take the sample from the edge of the patch, making sure it contains both healthy and diseased turf. This allows the diagnostician to watch the disease progress in the laboratory. If symptoms are diffuse, take two samples: one from the diseased area and one from a nearby area that appears healthy. Even though many turfgrass pathogens are readily identified in both healthy and diseased turf, having samples of both helps eliminate some pathogens as the primary disease-causing agents, since the relative abundance of a causal agent may be greater in a diseased specimen than in a healthy turfgrass specimen. Turf collected from golf courses may be sampled with a cup cutter and need only be removed to a depth of



Golf course cup cutter plugs make ideal samples for diagnostic laboratories. Both healthy and symptomatic turf should be included.

two to three inches. If a cup cutter is not available, use a knife to cut a 6″x 6″ piece of sod. Sample from both symptomatic and apparently healthy areas as described above.

Packaging the sample for shipment to a diagnostic laboratory is critical. If the sample is relatively moist, wrap it in newspaper or aluminum foil, and place it in a cardboard box for mailing. If the sample is dry, moisten it slightly, wrap, pack and mail as described above. Avoid wrapping samples in plastic or plastic bags since these materials retain moisture in the sample and encourage many different organisms to grow, possibly masking important symptoms. Avoid exposure to heat or direct sunlight.

Sometimes, nematodes and the problems they cause must be taken into consideration in disease diagnosis. If nematodes are suspected as the cause of a problem, it is best to sample from both healthy and symptomatic areas. The most appropriate times to obtain such samples are in the spring, about a month after the turf greens up, and in the autumn, when turf may be more symptomatic.

Sampling patterns depend on the symptoms present and the size of the affected area. If the turf is exhibiting a gradual decline, a series of smaller samples—referred to here as subsamples—should be taken randomly throughout the area (in a zigzag pattern, for example). A minimum of six subsamples should be taken from an area that is one half-acre (~21,000 ft.2) in size. If symptoms appear in patches, subsamples should be taken just inside the periphery of the patch.

All sampling should be done to a depth of approximately four inches. Subsamples may be taken with a cup cutter, a 1″ soil sampling probe, or a trowel. Subsamples should be mixed together, placed in a plastic bag and shipped immediately. Avoid exposure to heat or direct sunlight. It is best NOT to moisten samples believed to contain nematodes. Further details about specific nematode problems and their diagnosis will be published in future issues of *TurfGrass TRENDS.*

It is always best to collect and mail turfgrass samples early in the week, so they do not spend the weekend in a post office or at the diagnostic laboratory. It is always helpful to telephone the diagnostic lab before sending the sample to make sure that the diagnostician is prepared to receive and process the sample quickly. This is particularly important during the busiest months of the season (June, July and August). Whenever possible, send samples to the diagnostic laboratory using an overnight delivery service (Federal Express or Airborne Express, for example). See the list in this issue of *TurfGrass TRENDS* for the addresses of diagnostic laboratories in the United States and Canada.

Step 8. Laboratory examination of turfgrass samples

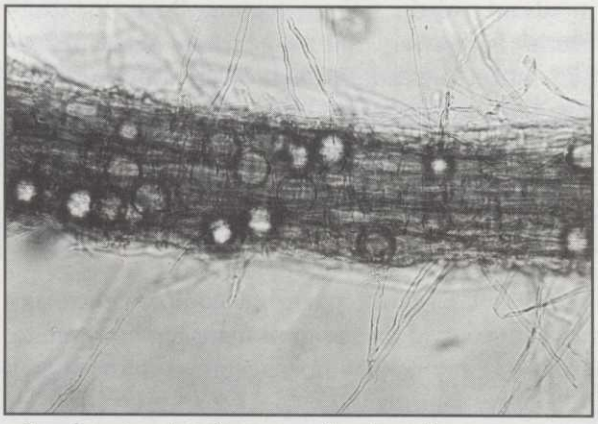
Closely examining turfgrass samples, whether in the field or in the laboratory, is a critical part of nearly all disease diagnosis. It serves as a means of verifying initial diagnoses based on field observations. In addition, in the case of some diseases, it provides the only means of definitively identifying the cause of the problem. The purpose of close laboratory examination is to find physical evidence for the presence of the causal agent(s).

Nearly all fungi causing diseases in turfgrasses produce characteristic structures, reproductive as well as nonreproductive, in affected plants. Since most fungal structures are not visible without significant magnification, the clinical diagnostician must use a microscope to examine them. Generally, two types of microscopes are used in the analysis of turfgrass specimens: a dissecting microscope for examining whole plants and plant organs, and a compound microscope for observing tissues and cells. Observation and classification of the fungal reproductive structures allow the diagnostician to more accurately identify the active pathogens.

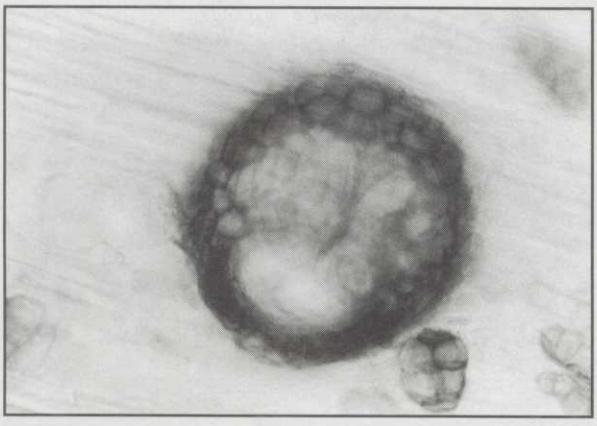
The structures for which diagnosticians look include: characteristic spore shapes, sizes and colors; unique mycelial shapes and structures; sclerotia; and fruiting bodies such as pycnidia, acervuli, and perithecia. The diagnostician must also check for the presence of other causal agents such as bacteria, viruses, algae and nematodes.

In some cases, no fungal structures may be apparent when tissues are observed under the microscope. In this case, leaf, sheath or crown tissues are incubated in a high-humidity chamber to encourage whatever fungal pathogens may be

After incubating a sample affected by brown patch in a high humidity chamber, the mycelium of the pathogen is readily visible.



Fungal structures in turfgrass organs (here is a turfgrass root containing oospores of Pythium spp., the cause of Pythium root rots) are visible under the microscope.



Microscopic examination of turfgrass tissues (this is a perithecium of a Leptosphaerulina species causing a foliar blight of Kentucky bluegrass) reveals additional fungal structures.

present to grow in a mycelial form or sporulate, revealing their reproductive structures for identification. These fungal tissues may then be transferred to laboratory culture media for further observation or examined under the compound microscope. However, since many different microbes on or in affected turfgrass tissues can grow and reproduce in this environment, the diagnostician may find evidence of several different fungal pathogens, along with a myriad of nonpathogenic or saprophytic fungi and other microorganisms.

If physical evidence of a pathogen cannot be found in turfgrass tissue, other methods for detecting and identifying pathogens may be used. The most common backup method for pathogen detection and visualization is isolation of the potential pathogen from the turfgrass tissue. Most fungal turfgrass pathogens can be readily grown on laboratory culture media. A few turfgrass pathogens are obligate parasites—in other words, constrained to living in a certain manner—which makes them difficult to culture. The latter include: *Puccinia* and *Uromyces* spp. causing rust diseases; *Erysiphe graminis* causing powdery mildew; and *Sclerophthora macrospora* causing yellow tuft disease. Once a pathogen has been cultured successfully in the laboratory, its growth and reproductive habits can be observed in detail and its physical appearance compared with what was observed in the diseased tissue.

Over the past few years, even more sophisticated methods of pathogen detection have been developed. These include immunological techniques that use pathogen-specific antibodies to detect the presence of specific pathogens in turfgrass tissues. More recent developments include methods for the analysis of pathogen DNA in the host tissue. These are similar to the blood DNA analysis currently used in criminal trials. Such techniques represent some of the most sensitive and accurate methods available for identifying particular pathogens and may prove to be the only means of confirming that the suspected pathogen is, in fact, the primary disease-causing agent.

In the event that no evidence of fungal pathogens can be found, the problem is either not a result of disease, or if it is the result of a disease it can only be the product of a nonfungal pathogen. The latter would include viruses, bacteria, and nematodes. The problem could also be caused by abiotic agents, or by noninfectious biotic agents such as algae, mosses, insects or rodents.

Step 9. Pulling it all together into a final diagnosis

Once all the pertinent information from field and laboratory has been assembled, the clinician faces the difficult task of interpreting all of the evidence and coming up with an accurate diagnosis. It should be noted that, while the process of assembling diagnostic evidence is rigorous, converting that evidence into a diagnosis is more art than science. Making the actual diagnosis is the most critical step in the educated guessing that goes on in this process.

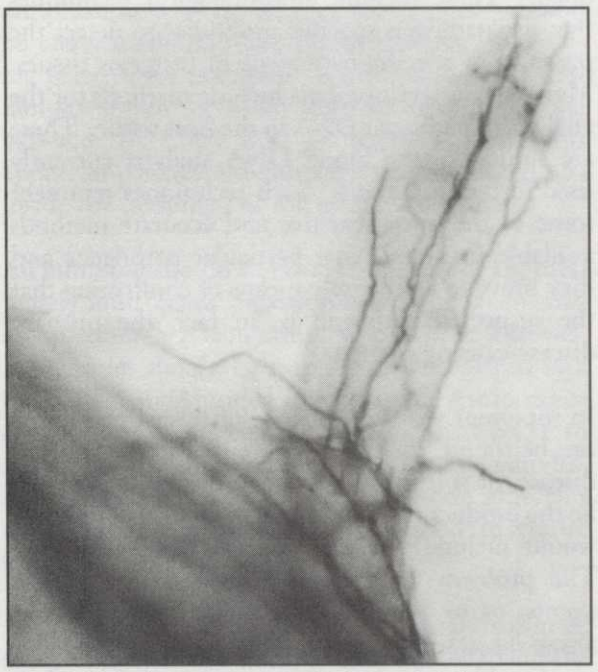
Sometimes the evidence available is either incomplete or inconclusive. In this case, further field observations, followed by another round of clinical examinations, may be warranted. In most cases, however, a diagnosis will prove possible.

Particularly in difficult cases, the diagnosis may hinge on the results of the laboratory examination. As is frequently the case, however, the clinical diagnostician may find evidence of two or more pathogens in the diseased specimen. Here is where the cultural and environmental information accompanying the sample becomes critical and must be evaluated together with the clinical observations to further narrow the range of possible causes. A diagnostician's ability to make accurate diagnoses is based primarily on his or her knowledge of specific diseases, the factors affecting their causal agents, and the pathogens themselves. Numerous written resources, in addition to those noted previously, are available to aid the diagnostician.

Diagnoses may be reported to the turfgrass manager in a variety of ways. Most often, a written report identifying the pathogen(s) believed to be the primary cause(s) of disease(s) is sent directly to the person who submitted the sample. Sometimes, to permit timely intervention to control a problem, the diagnosis will also be transmitted by telephone in advance of the written report. Prices for diagnostic services vary, but are generally in the $25 to $75 range, depending on the laboratory and the detail of the diagnosis.

Step 10. Select an appropriate management strategy

Recommendations frequently accompany the diagnosis. It is up to the turfgrass manager, however, to select an appropriate management strategy and implement it properly.



Ectotrophic hyphae growing on root surfaces can be indicative of a patch disease.

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Errata

On page 6 of the June 1995 issue of *TurfGrass TRENDS*, the term "Hyphae" is misspelled "Nypae."

Turfgrass Diagnostic Laboratories in the United States and Canada

Many turfgrass managers turn to the services of a clinical diagnostic laboratory for identifying turfgrass diseases. There are a large number of these laboratories located throughout the United States and Canada; most are affiliated with state and provincial agricultural universities. Many of the university laboratories also maintain branches in the offices of local or regional cooperative extension services.

A list of state-supported diagnostic laboratories, current as of July 1995, is presented below. All handle turfgrass problems. Since some laboratories are better able than others to diagnose a particular problem, it is not uncommon for turfgrass managers to send specimens to diagnostic laboratories in other states or provinces. Refer to the "Laboratory Services" key at the bottom of each listing.

In most cases, the first place to turn in search of a diagnosis is the nearest extension service office, even if your problem will be handled eventually by the laboratory. Please refer to the "Contact" remark at the bottom of each listing for guidance.

TGT Note: In talking with the laboratories, we heard one request again and again: "Please! Tell them to send in their specimens with documentation!" We are giving you phone, fax, and e-mail addresses to request the necessary forms and instructions.

Key ➢

A	Disease Diagnosis	D	Insect Identification	G	Screening for Resistance of Fungi to Fungicides
B	Pest Diagnosis	E	Nematode Detection		
C	Plant and Weed Identification	F	Screening for Turfgrass Endophytes	H	Other

Information compiled by Dr. Eric B. Nelson and Erin Kennedy

United States

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University of Alaska - Fairbanks
Agric. Forestry Experiment Station
Fairbanks, AK 99775-7200
Phone: (907) 474-7431
Fax: (907) 474-7439
E-mail: ffjhm@aurora.alaska.edu
Contact Ext. Service before Laboratory
Laboratory Services: A, B, E

Plant Diagnostic Laboratory
101 Extension Hall
Department of Plant Pathology
Auburn University
Auburn, AL 36849-5624
Phone: (334) 844-5508
Fax: (334) 844-4072
E-mail: jmullen@acenet.auburn.edu
Contact Ext. Service before Laboratory
Laboratory Services: A, B, C, D, E

Plant Disease Clinic
Lonoke Agricultural Center
P.O. Drawer D
Hwy. 70 East
Lonoke, AR
Phone: (501) 676-3124
Fax: (501) 676-7847
E-mail: fungus@uaexsun.uaex.arknet.edu
Contact Ext. Services
Laboratory Services: A, B, C, D

Department of Plant Pathology
University of Arizona
Forbes 204
Tucson, AZ 85721
Phone: (520) 621-1828
Fax: (520) 621-9290
Contact Ext. Services
Laboratory Services: A, C, E, G

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Fax: (303) 271-6644
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Colorado State University
Fort Collins, CO 80523-1174
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Newark, DE 19717-1303
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Fax: (302) 831-3651
E-mail: robert.mulrooney@mvs.udel.edu
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Gainesville, FL 32611
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Florida Extension Services
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Quincy, FL 32351
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401 Brooks Hall
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Regional Crop Laboratory
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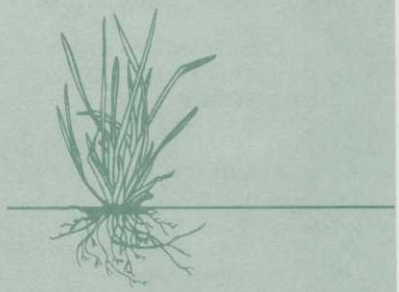

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Shurtleff, M.C., T.W. Fermanian, and R. Randell. Controlling Turfgrass Pests. Englewood Cliffs, NJ: Prentice-Hall, 1987 (ISBN: 0835910172).

Smiley, R.W., P.H. Dernoeden, and B.B. Clarke. Compendium of Turfgrass Diseases. 2nd ed. St. Paul, MN: The American Phytopathological Society/APS Press, 1992 (ISBN: 0890541248).

Smith, J.D., N. Jackson, and A.R. Woolhouse. Fungal Diseases of Amenity Turfgrasses. 3rd ed. London/New York: E.& F.N. Spon, 1989 (ISBN: 0419115307).

Vargas, J.M. Managing Turfgrass Diseases. Boca Raton, FL: Lewis Publishers, 1994 (ISBN: 1566700469).

Identification of Unknown Turfgrass Pathogens: Koch's Postulates

by Eric B. Nelson

History

In 1876, a country doctor in Germany by the name of Robert Koch developed the first conclusive empirical evidence that a particular microbe causes a particular disease. Although he was working on anthrax, a deadly disease of warm-blooded animals (caused by the bacterium *Bacillus anthracis*), the procedures he laid out for determining the pathogenicity of a microorganism (in other words, its ability to cause disease) are still in use today. These procedures are commonly referred to as "Koch's postulates." Although the theoretical criteria for establishing pathogenicity in microbes had been laid down as early as 1840, Koch was the first to successfully apply these criteria experimentally. He went on to isolate the tuberculosis bacillus (for which he was eventually awarded a Nobel Prize) and the bacillus causing cholera.

What Koch's postulates say

Basically, Koch's postulates identify the steps necessary to establish the causal relationship between a particular microorganism and a specific disease. These steps are:

1) The microorganism must be present in every case of the disease.
2) The microorganism must be isolated from the diseased host and grown in pure culture.
3) The specific disease must be reproduced when the pure culture containing this microorganism is injected into a healthy, susceptible host.
4) The microorganism must again be recovered from the experimentally infected host and grown in pure culture.

The postulates in action

These principles are routinely applied in the diagnosis of previously undescribed turfgrass diseases. Over the past several years, a number of previously unknown diseases or previously unknown pathogens on particular hosts have been described, based primarily on the successful completion of Koch's postulates. And all the diseases that we now recognize on turfgrasses were once examined in accordance with Koch's postulates in order to demonstrate that causal relationship.

Problems in implementing the postulates

Turfgrasses present a number of unique problems in fulfilling Koch's postulates, however. This is due primarily to the constant presence of pathogens on turfgrass plants.

First, because both turfgrasses and their pathogens are perennial, almost any pathogen can be associated with symptomatic turf. In other words, the first step of Koch's postulates is almost always satisfied, even without a direct causal relationship between a given pathogen and a given disease symptom, precisely because of the pervasiveness of pathogens in a persistent turfgrass ecosystem.

Second, Koch's postulates state that "the specific disease must be reproduced when the pure culture is injected into a healthy susceptible host." One of the problems of inoculating healthy susceptible turfgrass hosts is that this is typically performed using greenhouse-grown turfgrass plants. Symptoms on immature greenhouse-grown turf rarely, if ever, match the symptoms observed on mature stands of turfgrasses in the field. This makes it difficult to satisfy completely the third step of Koch's postulates.

Finally, if one were to inoculate mature turfgrasses in the field in an attempt to reproduce accurately and completely the symptoms initially observed with the unknown disease, it would be impossible to eliminate other potential pathogens, making the successful completion of Koch's postulates problematic.

In diagnosing unknown turfgrass diseases, there is always doubt whether the right pathogen has been found. This is particularly true in the diagnosis of diseases of root systems, where many different pathogens may reside (see the accompanying article in this issue of *TurfGrass TRENDS* on the diagnosis of root and crown diseases).

One of the basic assumptions of Koch's postulates is that a single organism, isolated from a diseased turfgrass plant and inoculated into a healthy turfgrass plant, will reproduce the symptoms observed in the field. We now know, however, that many diseases in turfgrasses do not act alone. Often, the infection of a plant by one pathogen will facilitate its infection by yet another pathogen. This is particularly true of root diseases, where some pathogens create wounds that allow the penetration by other, lesser pathogens. Disease complexes like this are more commonly the rule than the exception.

We also now know that plant stresses influence significantly the nature and degree of expression of symptoms. Numerous potential pathogens of turfgrass become problems only when the plants are under stress. This, too, is particularly common with root-infecting pathogens.

No substitutes for these procedures

The foregoing discussion has been critical of the procedures used to establish the pathogenicity of an unknown microorganism to turfgrasses. That process, however, is currently the most effective and widely accepted technique available to us for establishing those relationships. We still don't understand fully how pathogenic organisms induce disease symptoms in individual turfgrass plants; nor do we understand fully how such symptoms are expressed in mature stands of turfgrass in the field. Until we have such understanding, will have to rely on Koch's postulates for establishing the disease-causing potential of unknown turfgrass microbes—even knowing what we now know about the tenuous applicability of these procedures to perennial plants like turfgrasses.

Terms to Know

Biotic/Abiotic - of or relating to living organisms/non-living things

Disease - a destructive process in an organism; has (1) a specific cause and (2) characteristic symptoms

Lesion - function-imparing injury or other change in an organism's tissue

Necrotic - dead; describing a zone of dead tissue in an organism

Pathogen - an agent of any kind able to cause disease

Postulate - as in Koch's Postulates: prerequisites or basic principles

Saprophytic - living on dead or decaying organic matter

Senescence - aging; in plants, the growth phase between maturity and death

Symptom - a condition accompanying a disease and aiding in its diagnosis

Diagnosis of Root and Crown Diseases of Turfgrasses

by Eric B. Nelson

Root and crown diseases present unusually challenging diagnostic problems to turfgrass managers and diagnosticians. These challenges arise from a number of factors.

Problems bedeviling diagnosis

One of the greatest obstacles to the accurate diagnosis of root and crown diseases is the perennial nature of turfgrass plants. Roots of nearly all mature turfgrass plants are continuously infected with many, if not all, of the pathogens capable of causing disease on a particular turfgrass species. As a result, microscopic examination of roots and crowns usually fails to identify a single pathogen as the cause of an affliction.

Another factor complicating diagnosis is the presence of a large number of saprophytic fungi, which prefer to live on dead and decaying organic matter. Few of these fungi cause infection or disease in turfgrass plants, but they are readily observed on and in roots, rhizomes, stolons and crowns. Some of them may even penetrate the roots of some turfgrasses, but they rarely, if ever, cause direct plant damage.

The natural senescence of many turfgrass roots also presents diagnostic challenges. Roots of turfgrasses naturally age and wither at a rapid rate. In the process, they are often colonized by a vast array of microorganisms, both pathogenic and nonpathogenic. Since many of the pathogens causing root and crown problems prefer to live in a saprophytic mode, it is often not clear, when examining roots microscopically, whether a pathogen found in root tissue was the cause of its decline, or had colonized the root after that decline had begun. This dilemma is further complicated by the fact that, when examining roots microscopically, one can never be certain if the roots showing symptoms are from the current or the previous year, and if the latter, whether their decline was natural or disease induced.

One of the more aggravating problems in diagnosing root diseases is the difficulty of completing Koch's postulates satisfactorily (see the accompanying article for a discussion of these). The ability of root-infecting pathogens to cause significant damage depends heavily on environmental conditions and plant stresses. Turfgrass plants that are not under significant stress often fail to show foliar or root disease symptoms after infection of their roots. However, plants that are heavily stressed by excessively low mowing heights, excessively low or high fertilization regimes, excessively low or high soil moisture, excessive temperatures, high traffic, soil compaction and certain pesticide applications are more likely to decline as a result of root infection. In addition, those pathogens infecting root tips, root hairs or the epidermal layers of the root may not cause any significant necrosis, but may debilitate nutrient and water uptake through the root system. These types of pathogens may be par-

ticularly difficult to isolate from turfgrass roots, making it impossible to link them to the observed disease.

Obtaining a good sample for analysis

One of the critical aspects of diagnosing root diseases is proper sampling and recovery of root systems from soil, enabling their thorough examination. Roots are typically covered with soil and other organic debris. They may also be difficult to free from thatch. All of these factors make root observation difficult.

That difficulty is often compounded by a too-casual approach to examining the root systems of plants: clumps of turf are ripped from the ground, teased apart some, then examined superficially with the naked eye. This approach has serious deficiencies.

First, when roots have been infected with pathogens, the root tissues tend to be fragile and subject to easy breakage. When a clump of turf is pulled up, many of the diseased turfgrass roots are left behind in the soil, while healthy roots remain attached to the shoots. With just a casual inspection of such a sample, one might conclude that the roots were healthy, when in fact they might be seriously diseased.

Second, soil and thatch in the specimen can make suitable observations of roots nearly impossible. A more effective method of removing roots from soil is to cut a section of affected sod with a knife to a depth of approximately two inches, then place the turfgrass specimen under a stream of water and gently pull the specimen apart. The goal of this manipulation is to tease out individual plants with root systems intact and free of interfering thatch and soil debris. If special care is taken at this stage, more accurate diagnoses will be possible.

Under the microscope

Once individual plants with more or less intact root systems are in hand, it is relatively easy to determine whether their roots show any disease symptoms. It may be useful to examine roots of apparently healthy turfgrass plants for comparison. Things to look for (and record) are:

- the absence of root hairs
- discolorations of the root system, particularly at the root tips
- noticeable lesions or other deformities and their specific appearance
- the condition of the crown area
- whether discolorations or rotting appear to be progressing from the crown to the roots or from the roots to the crown
- visible fungal structures on or in the root and crown area (this usually requires a 10x or better hand lens)
- the nature of the rotting on the root system (For example, do the roots exhibit a wet, gooey rot or a dry rot? The former is more characteristic of pathogens such as *Pythium* species, while the latter is more representative of other patch disease root pathogens such as *Magnaporthe* and *Leptosphaeria.*)
- the presence of dark fungal mycelium growing on the surfaces of root and crown tissues (These structures are often observable with a 10x hand lens or a dissecting microscope and are indicative of problems associated with patch diseases.)

In nearly all cases, however, definitive diagnosis of root diseases requires a microscopic examination. This is necessary to actually observe the presence of the pathogen in infected and rotting roots and crowns. Small sections of symptomatic root, crown, rhizome or stolon tissue are placed on a microscope slide and stained with chemicals designed to color the pathogen but not the plant tissues. In some cases, diagnosticians may remove the contents of root cells with special chemicals, making detection of fungal structures in root tissues easier.

Occasionally, pathogen structures are not apparent. In these cases, the laboratory diagnostician may attempt to isolate and culture the pathogens from root or crown tissues. This is usually accomplished by placing pieces of fresh root tissue on sterile synthetic culture media that foster the growth of microorganisms. Sometimes, if a specific group of pathogens is suspected, turfgrass roots may be placed on media containing chemicals that will only allow that group of organisms to grow. If pathogens are present, they will usually emerge

from the infected roots and grow on the culture medium, enabling a more detailed study of the organism. Once potential pathogens have been recovered from infected roots, attempts can be made to complete Koch's postulates.

Most of the techniques used to diagnose root and crown diseases require specialized equipment and considerable expertise. Even experienced turfgrass pathologists have difficulties diagnosing some root and crown diseases on turfgrasses. As we learn more about the biology of root-infecting turfgrass pathogens, however, and as more sophisticated techniques for their detection and identification are developed, root disease diagnosis will become more accurate.

Not to be overlooked

Abiotic factors can also contribute to root dysfunction and decline, in particular high concentrations of soluble salts, root zone oxygen depletion and excessive soil temperatures. The natural senescence of turfgrass roots, particularly during the summer months, further complicates the picture. These factors must always be taken into account when contemplating, or conducting, root disease diagnosis, and should be included in any routine diagnostic procedure.

Terms to Know

Rhizome - a plant stem, usually horizontal, usually under the soil surface, with leaves or shoots above and roots below the nodes

Pre-/Post-emergent - before/after the emergence (of a weedy plant, for instance)

Surfactant - surface active agent; when added to liquids, surface active agents reduce surface tension, increasing the liquid's spreading and wetting properties

Tuber - a short, thickened, fleshy part of an underground stem; contains nodes and buds

Yellow Nutsedge: Biology and Control In Cool-Season Turf

by Joseph C. Neal

About the weed

Yellow nutsedge (*Cyperus esculentus*), often referred to as "nutgrass," is a tough-to-control perennial weed that infests most crops and turfgrass areas throughout most of the United States. Although grasslike in many ways, yellow nutsedge is not a grass; it is a sedge.

Sedges are easily distinguished from grasses by their leafy shoots, which are triangular in cross section. Shoots of grasses, on the other hand, are either flat or round. Distinguishing between grasses and sedges is very important, as most herbicides for grass control do not control sedges.

Yellow nutsedge emerges between late spring and midsummer, producing leafy clumps of long, narrow, light green and glossy, grasslike foliage. Yellow nutsedge spreads by rhizomes (underground stems), which produce "daughter" plants. Starting in late June, when days begin to get shorter, small, egg-shaped tubers begin to form at the tips of the rhizomes. Tubers mature in late July to mid-August. Under optimum conditions, a single plant can produce up to 7,000 tubers!

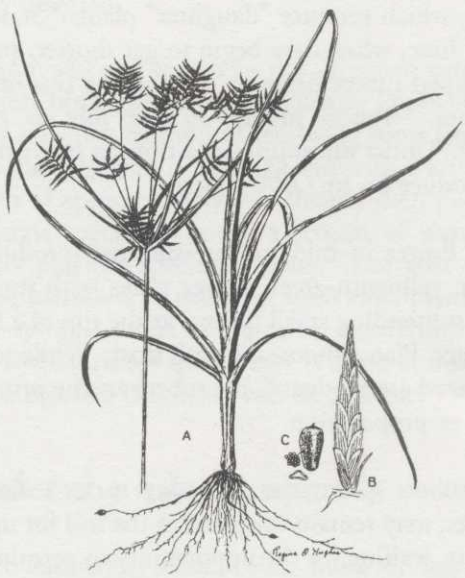
Plants flower in mid- to late-summer, producing slender, yellowish-green flower stalks with leaflike bracts subtending small flowers at the top of a leafless stem. Plants shoots die with frost. While some viable seed are produced, the tubers are the primary means of propagation.

Most tubers sprout the following spring. Some, however, may remain dormant in the soil for up to 10 years, waiting for the opportunity to germinate. Consequently, nutsedge control strategies must include a long-term commitment to preventing new tuber formation.

Where did it come from?

Yellow nutsedge is a native of North America. Although originally found primarily in poorly drained or wet areas, it now infests millions of acres of cultivated land and turf. Mindful of its past, many still consider the presence of yellow nutsedge a suggestion of drainage problems. On the contrary, yellow nutsedge is well adapted to growing in wet soils; dry, sandy soils; and everything in between. It tolerates close mowing, high or infrequent mowing and most herbicides labeled for use in turfgrass weed management. The primary reason for its spread appears to be the elimination of competition from other weeds. As we controlled other weed species, we eliminated the competition that previously restricted the distribution of nutsedge; in effect, we "released" nutsedge to become the major weed pest it is today.

Another factor contributing to the spread of yellow nutsedge is the resiliency of the tubers. Nutsedge tubers can remain dormant in the soil for up to 10 years. Control procedures may be effective within a season, but dormant tubers will remain to reinfest in following years. Additionally, tubers that have not yet sprouted are almost impervious to herbicides or cultivation. In conventionally tilled crops, tubers are spread by cultivation. As urban sprawl reaches farming communities, the top soil, much of it containing nutsedge tubers, is often sold and used for landscaping. Nutsedge tubers are also often introduced in root balls of field-grown trees and shrubs, from which rhizomes spread into adjacent turf.



Yellow nutsedge. Line drawing by Regina O. Hughes, from Selected Weeds of the United States, Washington, DC: USDA, 1970.

Why is yellow nutsedge so difficult to control?

Several herbicides are registered for yellow nutsedge control in turf; so, why is it considered to be so hard to manage? The answer requires a consideration of the biology of nutsedge growth and reproduction, as well as the situations where it is a pest. Yellow nutsedge reproduces primarily by tubers, then spreads by rhizomes. The tubers begin sprouting in the spring when soil temperatures reach about 65° F, but continue to emerge through late June. Since no pre-emergent herbicide labeled for use in cool-season turf is effective against nutsedge, post-emergent strategies are the only option available. Post-emergent herbicides are most effective on young nutsedge plants; but with no residual activity, and new plants emerging over an extended period, multiple applications are often necessary to achieve adequate control. Additionally, the extended dormancy of some tubers results in plant emergence and reinfestation of the site for years to come, even if effective control is obtained in a single season.

How to control yellow nutsedge

Herbicides registered for the post-emergent control of yellow nutsedge include the methane arsenates, MSMA and DSMA, and bentazon (Basagran T/O). With each product, multiple applications and treatments during warm weather are often necessary to obtain satisfactory control, but these same conditions can result in unacceptable turfgrass injury.

MSMA and DSMA are organic arsenicals that can be used for post-emergent control of crabgrass and nutsedge. Both are essentially contact-type herbicides; that is, they provide top kill and do not translocate to the rhizomes.

MSMA, available under numerous trade names, is used more commonly. It is generally available in

two formulations: 6 lb. ai per gal. or 6.6 lb. ai per gal. Besides the difference in ai per gal., the two formulations differ in surfactant recommendations. With the 6 lb. ai per gal. formulation, the surfactant is included in the product, and no additional surfactant is recommended. The 6.6 lb. ai per gal. formulation, on the other hand, contains no surfactant, and the addition of a nonionic surfactant at 2 to 3 oz. per 1000 ft.^{2} is recommended. The recommended rate of application for both formulations is 1 oz. per 1000 ft.^{2}. This is equivalent to 2 to 2.2 lb. ai/A (depending on the formulation).

Complete control is usually not achieved with a single application. Two to three applications at 14-day intervals are suggested. Since it is a contact-type herbicide, thorough spray coverage is essential for good control. Use a calibrated boom sprayer, not a hose-end applicator. The label suggests a spray volume of 2.5 gal. per 1000 ft.^{2} (about 110 gal. per acre); however, with flat fan nozzles operating at about 40 psi, excellent results can be obtained with as little as 0.7 gal. per 1000 ft.^{2} (30 gal. per acre).

Most turfgrass species are injured to some extent by MSMA; the extent of injury depends on the species, rate of application and environmental conditions at the time of treatment. Established Kentucky bluegrass, perennial ryegrass and tall fescue are tolerant, but may exhibit undesirable injury when treated during hot, dry weather. Bentgrasses and fine-leaved fescues are sensitive. Injury symptoms, which include yellowing, foliar burn and increased leaf spot disease, can persist for three to five weeks. Reducing the application rate limits the turfgrass injury but also results in diminished control, necessitating several applications.

The label suggests treating weeds during warm weather, when daytime temperatures are between 80° and 90° F. While this will provide rapid control of the weeds, high temperatures increase the severity of turf injury. In my research, I have found MSMA to work very well under cooler conditions. In fact, under cool, moist conditions, I have obtained better nutsedge and crabgrass control than when applications were made during hot, dry weather.

Bentazon also controls yellow nutsedge and annual sedges after they emerge. Unlike MSMA, bentazon does not control crabgrass. Bentazon is translocated to some extent in treated plants, but is still considered to have contact-type action on yellow nutsedge. Consequently, like MSMA, thorough coverage of the weeds is essential for control. Applications in water at a minimum of 1 gal. per

New Help Against an Old Pest

by Joseph C. Neal

Monsanto recently introduced a new product for nutsedge control with the trade name "Manage," the proposed common name halosulfuron and the research code number MON12000.

In trials across the country, this compound has controlled yellow and purple nutsedge as well as or better than industry standards, with reduced turfgrass injury. The active ingredient is a member of the sulfonylurea class of herbicides and, like other members of this herbicide class, will be used at low application rates—probably between 0.03 and 0.06 lb. ai per acre. Manage has been released for distribution and is already available from some distributors and suppliers in some states.

From our research at Cornell University, it appears that sequential applications will provide more consistent control than single treatments. In 1993, treatments applied at the three-to-five leaf stage controlled nutsedge at 0.03 lb. ai per acre. However, in 1994, the same treatment only suppressed nutsedge for about six weeks in our Long Island experiments and provided about 70% control in an up-state New York trial. In the up-state trial, 0.03 lb. ai per acre applied at the three-to-five leaf stage, followed by a second application six weeks later, increased control to 85%. These results mirror those obtained by other researchers in other regions. The variability between years may be attributed to drier weather during the 1994 trials. Research continues to refine the best rates of application and intervals for sequential treatments.

TGT view—Dr. Neal's promising results with Manage notwithstanding, turfgrass managers should keep in mind that nutsedge is one of the most difficult turfgrass weeds to control. Prudence suggests managers remain flexible when deciding on a control strategy based on new product. If experience is any guide, different strategies will be required to deal with nutsedge at different sites.

1000 ft.^{2} (about 40 gal. per acre) are recommended, using a calibrated boom sprayer.

Bentazon is available in a 4 lb. ai per gal. formulation. The recommended application rate for yellow nutsedge control is 2 to 4 pt. per acre (0.75 to 1.5 oz. per 1000 ft.^{2}, or 1 to 2 lb. ai per acre). The higher rate provides more effective nutsedge control, yet repeat applications at 10- to 14-day intervals are often necessary. With the lower rates, a 10-day period between applications (rather than 14 days) is recommended. The addition of a non-phytotoxic crop oil concentrate at a rate of 2 pt. per acre (0.75 oz. per 1000 ft.^{2}) greatly improves yellow nutsedge control but may increase the potential for turf discoloration.

Tolerant turfgrasses include established bluegrass, ryegrass, fescue and bentgrass (not collars or greens). While I have personally seen no turfgrass injury from bentazon, many turfgrass managers in warmer climates have observed injury on perennial ryegrass when applications were made during warm weather. Consequently, to avoid unacceptable turf injury, many superintendents with perennial ryegrass fairways reduce the application rate to 0.75 lb. ai per acre and/or omit the crop oil concentrate. Also, a longer interval between applications, 21 days, is suggested on the label. As you might expect, these actions will reduce nutsedge control.

Maximizing yellow nutsedge control while minimizing turf injury

While bentazon and MSMA differ in many respects, the following guidelines are useful for both herbicides.

- Know where the nutsedge is located. Map the infestations in late summer and scout these areas the following spring. This knowledge will enable you to be ready with the proper herbicides in adequate amounts and to treat the nutsedge when it is young and more easily controlled.

- Begin treatments when weeds are young. Young plants are more easily controlled, and cooler, moister conditions in the early season will reduce the potential for turfgrass injury. With early treatment, lower rates will be effective, but follow-up applications 10 to 14 days later will be necessary to control later-emerging weeds and plants that survive the first treatments.

- Continue these treatments at the appropriate intervals until control is achieved and no more yellow nutsedge emergence is observed.

- Avoid applications during hot, dry weather. Weed control is reduced and the likelihood of turfgrass injury is increased. Irrigation the day before treatment will help, but is no substitute for natural rainfall and cooler weather.

- Calibrate the sprayer. Both herbicides require thorough coverage for maximum control. Overdosing will increase turf injury; underdosing will decrease effectiveness. Also, watch your overlaps; too much overlap effectively doubles your application rate!

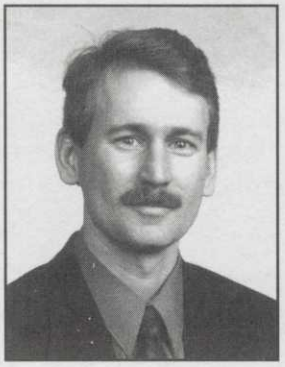
- Keep after it. Due to long-term tuber viability, it may take five years or more to get this weed under control.

- Remember that tubers may be brought to the surface or introduced in top soil when you do repair work.

With careful attention to the timing, dose, application method and retreatment intervals of herbicide applications, and with a long-term commitment, this weed can be controlled in cool-season turf. But then, no one ever said this job would be easy!

***Dr. Joseph C. Neal** is an Associate Professor of Weed Science in the Department of Floriculture and Ornamental Horticulture at Cornell University. He has degrees in Horticulture from the University of Georgia and Clemson University and in Horticulture Weed Science from North Carolina State University. Dr. Neal is currently researching the biological control of weeds; he also conducts research and extension programs in weed management for nursery and floriculture crops, turfgrass and landscape horticulture. His most recent contribution to TurfGrass TRENDS appeared in the May 1995 issue.*

Ask the Expert: Common questions, misconceptions and mistakes regarding nutsedge



Professor Joseph C. Neal
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Q1. ***It looks like grass, so why didn't pendimethalin control it?*** This is a sedge, not a grass. Most grass control herbicides are not effective on nutsedge.

Q2. ***The area looked so bad that I did a Roundup renovation last September, but the nutsedge is back as thick as ever.*** Roundup will control nutsedge if it is applied in mid to late June. Applying Roundup or any other herbicide for nutsedge control in late summer or early fall has no impact on tuber formation, however, and as a result won't affect nutsedge populations in subsequent years.

Q3. ***I bought clean topsoil, so where did the nutsedge come from?*** From a weed perspective, there is no such thing as "clean" topsoil. Whenever bringing in topsoil, be prepared to contend with a new complex of weeds, few of which will be as troublesome as nutsedge. In New York, at least, it seems the one thing you get for free with any load of topsoil is nutsedge!

Q4. ***I never had it before I put in sod there. It must have come in with the sod!*** Sod farms, both mineral and muck, may be infested with it, but nutsedge is highly unlikely to be transported in sod. Properly cut sod has so little soil attached that it would be virtually impossible for it to contain nutsedge tubers. It is more likely that during preparation of the site for sodding, nutsedge tubers were introduced or brought to the surface.

Q5. ***Early fall applications of Basagran really toasted it.*** My reply to this often heard suggestion: So does frost! See my comments to Q2.

Q6. [From a turfgrass manager on the Delmarva Peninsula]: ***I tried Basagran at the full labeled rate three years in a row, but couldn't control the nutsedge!*** Check with your local cooperative extension service to confirm the identity of your sedge. Purple nutsedge (*Cyperus rotundus*) and kyllinga (*Kyllinga* spp.) are also in this region (and further south). These species are not controlled by Basagran, and MSMA is only partially effective.

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