Chemical control of annual bluegrass, *Poa annua*, in turf has not been satisfactorily achieved despite the availability of a number of herbicides for the purpose. The problem is not ineffectiveness of the herbicides but the result of an interaction or conflict between turf cultural practices and weed control practices. The difficulty is compounded by growth patterns and characteristics of certain dominant ecotypes or natural strains well adapted to turf conditions.

Early observations and studies showed that in California annual bluegrass seed begins to germinate with the first cool fall weather reaching a peak germination by mid to late fall. Seeds continue to germinate through the winter and early spring but at a much lower rate. Little germination is likely during summer. Perennial strains were shown to make up a large part of the annual bluegrass population in many older golf greens and similar turf areas. A single strain frequently occupied many square feet of area crowding out all other grasses.

Other studies have shown that disturbing a turf during the period of annual bluegrass seed germination by such practices as vertical mowing or aerification will increase the annual bluegrass population greatly. These practices provide the light and soil atmosphere conditions favoring annual bluegrass seed germination.

As these practices, necessary for good quality turf, frequently must be performed during the annual bluegrass germination period, they should be integrated with a chemical control program to reduce the annual bluegrass population. An experiment to determine the interaction of vertical mowing and preemergence herbicide applications on annual bluegrass populations was conducted in 1967-68. Results may serve as a guide for better greens management programs.

The site of the experiment was at the Victoria Country Club in Riverside on an old Seaside bentgrass nursery. The turf had contained a large population of annual bluegrass, but a limited irrigation program had eliminated practically all the established plants. Numerous seeds were expected to be in the soil, however. A moderate thatch had developed over most of the area.

Bensulide was selected for the preemergence herbicide as its toxicity to germinating annual bluegrass seed had been well established. The application rate on all plots was 9 fluid oz. of a 45.2% active material in 5 gal. of water per 1,000 sq. ft.; the rate recommended by the manufacturer. Eight treatments were given the turf as follows:

1. No bensulide, no vertical mowing
2. No bensulide, one vertical mowing, fall
3. One bensulide application and one vertical mowing, fall
4. One bensulide application, fall, no vertical mowing
5. Two bensulide applications, two vertical mowings, fall and winter
6. Two bensulide applications, fall and winter, no vertical mowing
7. Three bensulide applications, three vertical mowings, fall, winter and spring
8. Three bensulide applications, fall, winter and spring, no vertical mowing.

The bensulide was applied immediately after vertical mowing and watered into the turf for 10 minutes. Treatment dates were September 26, December 27, and March 29. Vertical mowing was performed so as to cut through the thatch but not into the soil. Grass material cut out was removed before the chemical was applied. Each treatment
was replicated four times and individual blocks were 5 feet by 15 feet. Annual bluegrass populations were determined in late spring. Estimates were made by considering the plot with highest annual bluegrass population as no control and evaluating all other plots in respect to it.

Results are shown in Table 1. Highest annual bluegrass populations were observed in blocks which were vertical mowed in the fall but given no herbicide treatment. The population in these plots was higher than in those which were untouched (Treatment 1). The population was significantly reduced if bensulide was applied after the fall vertical mowing (Treatment 3). However, reduction of the annual bluegrass population was even greater from a single application of bensulide if not preceded by a vertical mowing (Treatment 4). Similar results were obtained from the treatments given two and three bensulide applications. Reduction of the annual bluegrass population was always greater in plots not vertical mowed.

This study confirms results from earlier experiments which showed that vertical mowing during its germination season increases the annual bluegrass population. It also shows that annual bluegrass populations can be reduced if a herbicide is applied immediately after vertical mowing. A fall and a winter application gave better control than a single fall application with this herbicide. The additional spring application was beneficial following vertical mowing but had no effect on the untouched plots.

This study emphasizes the importance of using a good preemergence herbicide following vertical mowing or renovation of any turf subject to annual bluegrass invasion. However, fall vertical mowing will increase the annual bluegrass population even when followed by herbicide treatments so this practice should still be avoided, if possible, during this season. It seems reasonable to conclude that similar results may occur from aerification during this period of annual bluegrass seed germination.

Table 1. Estimated percent annual bluegrass control by bensulide and vertical mowing treatments.

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Recognizing Turfgrass Diseases *

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WHAT IS A TURF DISEASE?

When we prepare to talk about turf disease identification, we should know first of all the answer to the question – “What is a turf disease?” Broadly speaking, it is the operation of any factor which continuously interferes with the normal structure or function of the turf plant. Generally speaking, there are two broad causes of turf diseases. One of these consists of unfavorable growing conditions, and with this we are not concerned at the moment, although we must recognize that such unfavorable conditions can literally create “disease.” The second broad cause is parasites, and this may include bacteria, fungi, viruses, nematodes, etc. Of this group we will not concern ourselves with bacteria, as their effect in turf disease is considered insignificant. As to virus diseases, we seldom hear of these, although more attention is being focused on the virus as a cause of turf damage. Nematodes, we know, create disease problems, but they represent a separate and distinct classification, and need not be considered here.

Therefore, we will concern ourselves here primarily with fungi and with fungus diseases.

a. What is a Turf Fungus? - It is a simple microscopically visible plant, which lacks chlorophyl, and therefore lacks the power to generate its own food. It must get its food from other organic matter. Saprophytic fungi are those which draw their food from dead or decaying organic matter. There are hundreds of varieties of these, but they don’t bother us since they do not attack living grass. Parasitic fungi are those which attack living grass, and these constitute our problem of disease and our problem of identification.

b. How Does a Parasitic Fungus Function? - Fungi usually start as “seeds” or spores, which simply remain dormant, often for a number of years, if conditions for their development are not ideal. In contact with a susceptible host, in this case a susceptible grass, the spore “germinates” producing “branches” called hyphae. These grow and multiply into masses, or bodies, called mycelia, and it is these which can occasionally be seen with the naked eye. The hyphae grow, penetrate into the plant tissues through natural openings or through wounds such as those made by mowing, and proceed to grow within the plant tissues, drawing their subsistence from the host plant, breaking down its tissues, and otherwise destroying the normal function of the grass plant. At maturity the fungus gives off more spores, which then proceed to start the life cycle all over again. In a few cases, spores are not created, and perpetuation of the life of the fungus is carried on by parts the mycelium scattered about and growth being generated from these parts. Actually, it is similar to growing new turf from stolons.

VISUAL EVIDENCE OF FUNGUS DISEASE

Diseases caused by parasitic fungi usually are evidenced by characteristic outward signs – the effects of...
Another pathologist, asking for an identification. "And for the Snow Mold organism to be active. The questioner explained that the presence of snow itself is not necessary said it sounded to him like Snow Mold. "But there was completely dead, under cold, wet conditions. Dr. Howard appeared as a panel member. One of his questioners described a disease on his greens, with circular areas turning off color, I recalled that helminthosporium had been widespread during the previous week, causing leaf spots like Joe was describing. I suggested that it might be helminth - and since our broad-spectrum fungicide is designed to control this disease, among others, I suggested that Joe apply some of it. However, I told him to get confirmation first, from someone who could possibly speak from experience. I referred him to our local distributor, who had been a golf superintendent for 36 years, and who works closely with a number of experiment stations and plant pathologists. Joe agreed this was a good idea, and in about ten minutes he called me back. Said he, "Your distributor certainly told me what my problem was." "What did he say it was," I asked? "SOD WEB-WORMS" he replied. Well, you can imagine my embarrassment, but you'll recognize that I simply had not had all the facts. Also, I had assumed Joe had subjected his lawn to reasonably good maintenance, including insect control, which, of course, he had not.

b. The second incident concerns a recent talk with a well-known plant pathologist in a Northeastern state. He said he often receives, by airmail, a plug of diseased turf, with a request that he identify the disease organism. He gets to work on it immediately, he says, unless he finds that similar samples have been sent to other pathologists. In such cases he respectfully declines to undertake the identification, telling his inquirer he's sure he'll get an accurate diagnosis from other pathologists he has contacted. Now this fellow has absolute confidence in his diagnoses, and I certainly do, also. But the travels of a turf plug through mail and other channels, over varying distances and under varying conditions, could well activate pathogens which were not active when the sample was mailed, or could inactivate the pathogen that originally caused the damage. There is no reason why a good pathologist should have his word questioned, under conditions like this.

c. The third incident concerns the G.C.S.A.A. International Conference several years ago at Long Beach, where the eminent pathologist, Dr. Frank Howard appeared as a panel member. One of his questioners described a disease on his greens, with circular areas completely dead, under cold, wet conditions. Dr. Howard said it sounded to him like Snow Mold. "But there was no snow," said the questioner. Dr. Howard patiently explained that the presence of snow itself is not necessary for the Snow Mold organism to be active. The questioner then said he had sent a sample of the diseased turf to another pathologist, asking for an identification. "And what did that pathologist say," asked Dr. Howard. "He said that it was Snow Mold," said the questioner. Here is another example where there was some doubt about the identification.

d. The fourth incident concerns a very well known golf superintendent in a southern state, who telephoned me about a year ago and said, "Stan, I have a terrific Pythium problem. I'd like to get confirmation that Pythium is the culprit. To whom can I send a plug of this diseased turf?" We recommended a pathologist whom we'll call Dr. Brown (mainly because that was not his name), and our friend sent him the plug. About two weeks later we received from this superintendent a carbon copy of a letter that Dr. Brown had written to him, stating that he could find no evidence of Pythium at all, but that there was plenty of Helminthosporium activity, and the plug contained a big nematode population. At the foot of the carbon copy, the superintendent had penned a note which said, "With all due respect to Dr. Brown, I know Pythium when I see it."

WHAT DO INCIDENTS LIKE THESE TELL US?

They point up a number of things to be considered in disease identification:

a. Accurate turf disease identification sometimes isn't easy. Under any circumstances, all facts are needed.

b. The complete facts may be difficult to get, especially when turf plugs are being dealt with, after being subjected to varying conditions during lengthy travel periods.

c. Under any conditions, there may be an honest difference of opinion as to which organism is doing the damage - and perhaps one or both are.

d. In the final analysis, accurate identification is often possible only through the scientific procedures and expert interpretations of plant pathologists.

WHY IS FUNGUS DISEASE IDENTIFICATION SOMETIMES DIFFICULT OR COMPLICATED?

For a number of reasons:

a. For instance, symptoms of one turf disease often are quite similar to those of other disease. For example, helminthosporium attacks often look like curvularia attacks. Curvularia attacks sometimes have the same appearance as a fusarium attack.

b. Sometimes symptoms of a turf disease are similar to those of another problem which isn't a disease at all. For instance, a discoloring of large grass areas that may look like helminth damage actually may be iron chlorosis, or, what is suspected to be leaf spot disease actually may be damage by insects such as webworms.

c. Even under the microscope, the observance of turf disease organisms is never a sure indication that the organism you see is the one doing the damage.

d. Even if there is an actual disease, and so diagnosed, this may not be the real problem at all, but possibly the result of a more basic seal problem. For example, while Pythium disease may be evident, the real problem may be poor drainage in the turf area, which permits the Pythium to develop and damage the turf.

COMMON DISEASES OF TURF

1. Rhizoctonia brown patch. Symptoms: Brown patch appears as irregularly shaped patches of blighted grass ranging from a few inches to several feet in diameter. There is usually no problem identifying it during warm, moist, periods because of the development of dark purplish "smoke rings" at the edge of the diseased areas. These are seen early in the morning but usually fade as day progresses and the relative humidity drops.
The broad-spectrum organic fungicides Maneb and Kromad avoid excessively high nitrogen levels. Once established in a grass blade, further hyphal production occurs, the hypha bridging across from blade to blade. Intense hyphal development will occur when there is free moisture on the blades and 100% relative humidity causing the cob-webby, smoke-ring effect.

The feeding of the fungus in the leaf blades results in the blighting of the blades. Control: Cultural practices - (1) Removal of free water following a heavy fog or dew by sweeping the green with large brushes, or dragging a hose or pole across them. (2) Increase aeration through proper spacing of ornamental plants. (3) Proper fertilizer program, particularly avoiding excessively high nitrogen levels.

Chemical control: Brown patch has been controlled for years by mercury-based fungicides, particularly the two inorganic mercuries, mercurous chloride and mercuric chloride. Organic mercury fungicides also control and so do the broad-spectrum organic fungicides Maneb and Kromad. PCNB is particularly toxic to the Rhizoctonia brown patch organism.

2. Sclerotinia Dollar Spot. - Symptoms: Under close clipping of putting green, at first there are small spots of blighted turfgrass which develop into circular, straw-colored areas 2 to 3 inches in diameter. When dew is present, a white, cob-webby growth of mycelium may be seen in the affected foliage. Where the mowing is high, the spots may be much larger, irregular in shape and can range from 6 to 12 feet in diameter.

Cause: The causal fungus Sclerotinia homoeocarpa forms small blighted areas on a broad leaf host. An example: bluegrass rust goes to a red stage where the rust evolves into a black spore stage. The black spores germinate the following spring producing still another type of spore (basidiospore) that does not infect grass but does cause infection on a broad leaf host. An example: bluegrass rust goes to certain barberry species. Spores produced on the barberry can cause infection on bluegrass but not barberry. Alternate hosts are not important under normal turf conditions because usually they are not present. The rusts are very effective. Inorganic and organic mercuries and inorganic mercuries, mercurous and mercuric chlorides. Organic mercury fungicides also control and so do the broad-spectrum organic fungicides Maneb and Kromad. PCNB is particularly toxic to the Rhizoctonia brown patch organism.

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Control: Cultural practices - A high nitrogen level coupled with maintaining soil moisture to near field capacity reduces the disease development. However, these practices do not control the disease and it is necessary to have a chemical control program.

Chemical Control: The cadmium containing fungicides are very effective. Inorganic and organic mercuries and Dyrene have also been effective. When this disease is known to be present a preventative spray schedule should be started in the spring when daytime air temperatures are 70°F repeating application every 2-3 weeks. Curative application must be made at 3 to 4 day intervals until plant recovery is accomplished.

3. Rust. - Symptoms: The turf becomes rusty in appearance. A close look reveals the blades have numerous, round, rusty spots. The rust readily rubs off onto one's fingers when the blade is rubbed. A rusty dust collects on one's shoes and other wearing apparel when walking across the turf.

Cause: There are a large number of species of rust fungi. They all have a similar life cycle and characteristic manner of parasitism.

The red stage is the summer stage of the rust fungus. The rusty material is actually thousands of spores each capable of causing a new infection. The length of time from germination in the infection to the production of a new pustule is about 7-10 days.

There are five distinctively different types of spores formed during the life of a grass rust. However, we are interested only in the red spore stage because it results in the infection and disease damage to the turf. As the infected grass blade dies, the rust evolves into a black spore stage. The black spores germinate the following spring producing still another type of spore (basidiospore) that cannot infect grass but does cause infection on a broad leaf host. An example: bluegrass rust goes to certain barberry species. Spores produced on the barberry can cause infection on bluegrass but not barberry. Alternate hosts are not important under normal turf conditions because usually they are not present. The rusts are very effective. Inorganic and organic mercuries and inorganic mercuries, mercurous and mercuric chlorides. Organic mercury fungicides also control and so do the broad-spectrum organic fungicides Maneb and Kromad. PCNB is particularly toxic to the Rhizoctonia brown patch organism.

Control: Cultural. Some people advocate applying nitrogen to stimulate rapid growth which would be dipped off before the rust has time to develop.

Chemical: The carbamates, Zineb and Maneb, and Cyloheximide give excellent control; sulfur will also give control.

4. Pythium Blight. - Symptoms: The turf becomes spotted with irregularly shaped, small blighted areas ranging from 1/2 to 4 inches in diameter. In the early stages the grass blades have a water-soaked greasy appearance. This is followed by shriveling of the leaves and the patches fade from green to light brown. The initial spots look like Dollar Spots, but are pinkish in color, with cottony mycelium visible in the spots. As the disease progresses, the patches meld together, causing large areas to be killed.

Cause: There are two species of pythium fungus which parasitize turfs. They are capable of living as soil saprophytes thus they are omnipresent. However, in a previously diseased turf, the organism may remain as dormant mycelium and overwinter in infected leaf blades and crowns.

Warm days followed by cool nights with long dew periods favor infection and reproduction of the rust fungus. Rust is more apt to become a problem in the late summer or fall because of favorable environment for infection and the growth rate of the grass blades may be reduced allowing for adequate time for pustule development before being clipped off.

Control: Cultural. Some people advocate applying nitrogen to stimulate rapid growth which would be dipped off before the rust has time to develop.

Chemical: The carbamates, Zineb and Maneb, and Cyloheximide give excellent control; sulfur will also give control.
Chemical: On the whole, fungicides have not provided adequate control of this disease. The product Dexon is an exception and has shown excellent promise.

5. Fairy Ring - Symptoms: Fairy ring disease causes a circular ring of fast growing, dark green grass often surrounding a ring of thin or dead grass. Sometimes the ring is not complete and gives the appearance of an arc or horseshoe. The rings vary in size from a few inches in diameter to many feet. The strip of thin or dead grass varies from three to six inches in width. After rains or watering many mushrooms or toadstools may appear in the area of dark green grass.

Cause: Fairy ring is caused by several soil-inhabiting mushroom types of fungi which grow very intensively in the area of the ring. Growth starts with a germinating spore at a central point and filaments of the fungus grow outward equally in all directions. The outward spread may occur at the rate of a few inches to two feet per year. The part of the fungus on the inner side of the circle dies off as the fungus grows outward. The darker green grass is caused by microorganisms in the soil breaking down organic matter, subsequently resulting in greater nitrogen availability.

Control: Mercurials will control Fairy Ring if they can be gotten into the soil to the depth where the organism exists. Perhaps adding wetting agents to the fungicide solution and drenching the area with acid will aid in better control.

6. Typhula Snow Mold - Symptoms: This disease is usually found in winter or early spring in wet shaded areas or where snow is slow to melt. It appears as more or less circular, dead, bleached areas from one inch to several feet in diameter. Several spots may run together forming large, irregular areas. Affected grass may be first covered by a dense, whitish-pink or dirty-gray to almost black moldy growth.

Cause: The fungus, Typhula Itoana, survives the summer as sclerotia. Under snow cover and over unfrozen ground, the sclerotia germinate to produce mycelia which infect the grass blades. In the spring, the cool temperatures and high moisture levels associated with the melting snow are ideal for further disease development.

Control: Only preventative measures will control gray snow mold. Applications of inorganic or organic mercury compounds in late fall before the first freeze or heavy snow fall will do the trick. The inorganic mercury's are usually considered to give better control, with longer residual action.

7. Melting-out or Fading-out - Symptoms: A variety of symptoms are caused by this disease. Thinning out of grass in scattered areas of the turf is one sign of melting out. A general brownish undercast caused by dead grass leaves accompanies the general thinning out. As the disease progresses, large irregular areas of the turf are killed.

Close examination of the leaf blade reveals a spotting of the leaves. The spots are usually brown or purple. The leaf spots stage of the disease commonly does not cause extreme damage to the lawn. The severe injury results when the leaf sheath area in the crown of the plant is infected.

Cause: There are several fungus species (Helminthosporium curliaria) involved in this disease complex. These particular fungi live as saprophytes and are commonly present. The fungus lives in the dead debris from where it can produce infective mycelia that infect leaf sheaths and crowns or produce spores that cause infection on the leaf blades resulting in leaf spots.

During cool moist weather in the spring and fall the disease is more common as a leaf spot. In the summer when the weather is relatively warm and dry, the fungus is restricted primarily to the crowns and roots.

Control: Cultural: High nitrogen levels favor the development of this disease. Therefore, do not overstimulate grass with inorganic nitrogen. Keep the grass growing well during midsummer because drought stress favors invasion of crown and roots. Close clipping also favors the disease.

Chemical: Inorganic mercuries, Maneb and Dyrene have given good control. If chemicals are to be used, a two-week spray program should be started in spring until dry, warm weather of summer.

8. Fusarium Patch - Symptoms: This disease is also sometimes called pink snow mold. It exhibits a variety of symptoms. It may begin as areas of pale-yellow grass, irregularly circular in outline and ranging from 2 inches to 1 foot or more in diameter. The affected areas change to a whitish-gray color. Under snow cover, aerial mycelium develops which is white at first but becomes purplish upon exposure to light. Usually, only leaves are attacked but under severe conditions the organism may penetrate the crowns causing complete destruction of the plants.

The disease can be distinguished from typhula blight by, 1) the production of pink mycelium and, 2) the absence of the c h a r a c t e r i s t i c brown, leaf-embedded sclerotia so typical of typhula blight.

Cause: The fungus Fusarium nivale survives adverse periods as dormant mycelium on plant debris. The disease may occur in the late fall, winter and early spring whenever weather conditions permit. Conditions favoring development are high humidity and an air temperature range from 32°F to 45°F. Although it may actively continue up to 65°F, above 70°F the fungus becomes dormant.

During favorable growing conditions for the fungus, it produces spores that are distributed to leaves by wind where they germinate and produce an infection tube that penetrates a stomate.

Control: Cultural: Late application of inorganic nitrogen favors the development of this disease. Applying a straw mulch to protect the plants or allowing a heavy thatch to develop results in the creation of “moist chambers” that are ideal environmental chambers for the pathogen.

Chemical: Inorganic and organic mercury's applied in the fall and winter have given good control.

GENERAL PRINCIPLES OF DISEASE CONTROL

Along with the general subject of turf disease identification, it's well to emphasize again the associated principles that affect disease activity and development.

1. Follow good maintenance practices: Make sure your water, fertilization, insect control, weed control and aerating programs are all in order to give the grass optimum growing conditions. Check soil structure and drainage - make sure neither standing water nor soaked soil conditions create an environment favorable to disease. Keep your mechanical equipment in tip-top order - mowers sharp - spray and broadcasting equipment properly calibrated for exact dosages.

KNOW YOUR TURF DISEASES AND HOW TO CONTROL THEM: Stock your library with good books on turf diseases. Dr. Malcolm Shultefl's book “How to Control Plant Diseases” is excellent for professionals and non-professionals alike. Dr. Houston Couch's fine new book
"Diseases of Turfgrass" is one of the most scholarly ever written on the subject. I commend it to you most highly. DuPont, Cleary, Upjohn and other manufacturers have published literature you should have in your library. And, of course, our own "Turf Disease Handbook," well illustrated, and with excellent keys to identifying diseases both at normal visibility and through the microscope should be always at your fingertips.

FOLLOW A STRONG PREVENTIVE FUNGICIDE PROGRAM: I wonder if many of us really - and I mean really - understand the need to prevent turf diseases, rather than to cry to "cure" them once they've started their damage. Let me give you some rather compelling reasons why you should be following a preventative program.

A. Damaged turfgrass never heals - the blades may grow, and the damaged part removed by mowing - but that damaged tissue is gone, it cannot be "cured."

B. On a good golf course or fine turf area it is always less expensive to prevent disease than to correct the damage by re-seeding or re-sodding.

C. Like life insurance, preventive disease control is a small price to pay for the tremendous protection it brings - including protection of the job of the superintendent or turf manager.

D. For some diseases there is no alternative to prevention. Spring Dead Spot, for instance, cannot be detected on bermuda throughout the winter 'while it is at work - only in the spring, when the diseased areas don't green up. And Grey Snow Mold, that originates under heavy snow cover, may already have done its damage when the snow thaws.

E. A superintendent or turf manager does not get paid to continually correct problems like disease problems. He gets paid not to have problems! His superiors expect him to maintain disease-free turf, not to be constantly treating unsightly diseased areas while still other areas are being attacked.

F. It's likely that more superintendents and turf managers are dismissed because of loss of fine turf to disease and other such results of mismanagement than for any other reason.

G. FINALLY - A good preventive program eliminates much of the need for the thing we're talking about today - turf disease identification. If your program prevents disease from ever attacking, you'll never have damage - never see the disease activity - and hence never have to identify the organism.

USE GOOD TURF FUNGICIDES

In your preventive programs use only fungicides that are effective. Cost should be only secondary, and when considered at all, it should include not only the per pound price, but also the rate and frequency of application. A $0.70 per pound fungicide is expensive if only one pound per 1,000 square feet must be applied weekly - the total cost is $2.80 per 1,000 square feet per month. A $9.00 per pound fungicide is economical - only $0.28 per 1,000 square feet per month - if only 1/2 ounce per 1,000 square feet is needed per month.

CONSULT THE EXPERTS

When all sound maintenance principles have been followed and your grass nevertheless sustains an attack from an unidentified disease, see an established plant pathologist for an identification and for control recommendations. Airmail him at least a four-inch plug, containing, if possible, both healthy and diseased turf. Include a letter with all facts, such as date plugged, kind of grass, temperature, chemicals used on the turf during the previous several weeks, and all other factors that could have an effect, making full allowances for possible changes in the plug in transit. Give your full respect and credence to the pathologist's diagnosis. In all likelihood it is the correct one. In the final analyses only the pathologist is really qualified to give a meaningful and fact supported turf disease identification.

The Effects Of Temperature And light On Vegetative Growth*

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Before we can begin a discussion of light and temperature effects on vegetative growth we must obtain a view of the relationships of a plant to its entire environment. The environment of a plant is dynamic and extremely complex. It involves the interrelationships of soil, climate, topography and other organisms including man. No aspect of light or temperature acts upon the plant alone but rather in concert with all other factors and as it is modified itself by the other factors.

A moment's thought will make this obvious. We may say that high temperature has injured a plant, but was it the high temperature alone? Was the soil moist or wet? Was the wind blowing? Was the atmosphere's humidity high or low? Was the plant in full sun or shade? What were the conditions to which the plant was exposed prior to the high temperature damage? Many similar questions could be asked but these will suffice to point out the complexity of what may at first appear to be a simple effect of environment. Not only do many factors interact to produce a given response but the history of a plant's growing conditions may have a direct bearing on how it responds to factors of the present.

Finally, of great importance in determining an environmental response, is the plant's heredity - its genetic makeup or genotype. Species and varieties within a species have evolved over many years through the process of natural selection. Natural selection is nothing more than the survival and reproduction of those individuals best adapted to a particular environment. Reproduction, of course, is the key as there can be no natural selection or evolution of an organism unless sexual reproduction with genetic recombination occurs generation after generation.

Now looking at the specific factors, light and temperature, which are the subjects of this discussion, we must recognize several important aspects of each as they effect plant growth.

Light intensity, duration of light period (daylength or

plant. When considering plants we must also distinguish between soil and air temperature as they affect growth. Experimentally, however, it is frequently difficult to separate their effects and we must speak of the two together.

Photosynthesis and respiration are the light and temperature regulated processes that are basic to all other growth functions of a plant. These two physiological processes provide the plant with the energy needed for survival and growth.

In photosynthesis, carbohydrates are synthesized in the green chloroplasts of the plant’s leaves from carbon dioxide and water using light as the source of energy. Thus, this is an energy storing process. Respiration on the other hand is a process of energy utilization through the break-down of the carbohydrates and release of carbon dioxide. The first steps in photosynthesis can occur only in the presence of light while respiration takes place during both light and dark periods.

Both processes increase with increasing temperature. At moderate temperatures photosynthesis occurs at a higher rate than respiration thus there is an accumulation of carbohydrates as sugars, fructosans and starch. As temperatures increase a point is reached at which the photosynthetic rate levels off and at higher temperatures begins to fall. However, the respiration rate continues to increase with increasing temperature to a much higher point. Eventually the plant is utilizing carbohydrates at a higher rate than they are being synthesized and the a higher rate than respiration thus there is an accumulation of carbohydrates as sugars, fructosans and starch. As temperatures increase a point is reached at which the photosynthetic rate levels off and at higher temperatures begins to fall. However, the respiration rate continues to increase with increasing temperature to a much higher point. Eventually the plant is utilizing carbohydrates at a higher rate than they are being synthesized and the plant begins to draw upon the reserve carbohydrates accumulated at the lower temperatures. The optimum temperature of carbohydrate accumulation is related to light intensity. Obviously, an overdraft on carbohydrates cannot continue indefinitely without harmful effects on the plant.

We have shown experimentally that maximum carbohydrate storage of both a warm season grass (bermuda) and a cool season grass (Kentucky bluegrass) occurs at temperatures near the minimum for measurable growth. A period of high temperature of only a few days will rapidly deplete carbohydrate reserves. Young Kentucky bluegrass plants grown at warm temperatures are unable to accumulate any reserve carbohydrates and even lose the little which may have been stored earlier at a cooler temperature. This may be what happens when Kentucky bluegrass turf is planted in late spring.

Varieties differ in their ability to store carbohydrates at any temperature. We have shown that Merion Kentucky bluegrass has a much superior ability to accumulate reserves than does Newport even at the most favorable temperatures. This differential becomes especially pronounced as marginal temperatures are approached.

The significance of these temperature and light reactions and plant responses to turf survival is a subject of debate, however, a relationship to root, top and stem growth will be noted in the following discussion of the growth of these tissues in respect to light and temperature.

TURF DENSITY

Turf density is maintained through either tillering or rhizome growth or both depending upon the grass species. Stolon growth is important to achieving density in an immature turf but is a secondary factor in an established turf except for healing of damaged areas. Tillering is the development of new shoots from buds arising in the leaf axils of existing shoots. The new tillers as they mature also produce tillers as well so the process is a continuing one as long as environmental conditions are favorable.

Initiation of tiller buds is not dependent upon climatic factors but the subsequent growth of the tillers is related to both temperature and light. Often under unfavorable conditions buds may be noted in almost all leaf axils but essentially no tillers develop from them.

Recent studies have shown that a young tiller is dependent upon the parent plant for food materials until such time that it has sufficient leaf surface to sustain itself. Hence, temperature and light conditions that are optimum for a high net photosynthetic rate will be favorable for tiller development. High light intensity is particularly important for most turf grasses.

Tillering of cool season grasses is most rapid at moderate temperatures. Either high or low extremes will prevent tiller development. The exact optimum, however, is related to the species. Ryegrass has its maximum tillering rate at about 60°F while the maximum for colonial bentsgrass is at approximately 70°F. Warm season grasses in general have a higher optimum temperature for tiller development than do the cool season grasses. Even different strains of a species may have different optimum temperatures. For example, we have shown that Merion and Fylking Kentucky bluegrasses have a higher optimum tillering temperature than Newport under identical light conditions. Hence, we might expect Merion and Fylking to be the better varieties for areas of high spring and summer temperatures.

Although vegetative growth in general increases as the daylength increases tiller initiation in grasses under turf conditions does not seem to be determined by length of day. The best evidence to date indicates that a high rate of tillering occurs throughout the range of moderate daylengths such as would exist during spring and fall. Rhizome and stolon growth on the other hand is favored by long days. This is true of both warm season and cool season grasses. Bermuda, Zoysia and bent grasses have little or no stolon growth at daylengths of 8 to 12 hours even though temperatures are optimum for the species. Scleron growth increases rapidly as daylengths increase from 13 to 17 hours. Although this may in part be the result of the plants receiving more total light energy we have obtained strong evidence that it is primarily due to the length of the light period per se. Several species were given hours of full sun light plus 2, 4, 6, 8 and 10 hours of low intensity artificial light in growth chambers. The long day relationship still held true even though the supplemental light was too low for significant photosynthesis to occur.

Factors causing rhizomes to emerge from the soil are not clearly understood and appear to vary considerably with different species. Temperature and daylength may be factors but there are conflicting reports for even a single species such as Kentucky bluegrass.
ROOT GROWTH

Before discussing the light and temperature relationships to root growth of turfgrasses it must be emphasized that frequency and height of clipping override the effects on the root system of all other factors. The reduction of root weight and length by clipping as practiced on turf cannot be fully compensated for by any aspect of temperature light or nutrition.

Moderate temperature and high light intensity stimulate root development of cool season grasses. As these are the same conditions necessary for a high photosynthetic rate, we may assume that rapid root growth under these conditions is the result of a readily available supply of carbohydrates. Root initiation and growth is practically non-existent under high temperatures but top growth and respiration rate may be high. Many workers have concluded from this that cessation of root growth results from the utilization of all available carbohydrates by respiration and top growth.

Soil temperature as distinct from air temperature also has a pronounced effect on root growth. In one experiment we grew bermuda, Kentucky bluegrass and Highland bentgrass at an air temperature of about 74°F and at root temperatures of 50°, 60°, 74° and 80°F. Root weight and length was least for all three grasses at 50°F and highest at 74°F with an obvious decrease from 74° to 80°. This decrease in root growth at the warm root temperature may be significant since the surface of the soil may frequently reach temperatures well above the air temperature. Many years ago it was demonstrated that the temperature of the surface layer of soil exerts the same effect upon the root as it does if applied to the entire root system. High soil temperatures may actually cause a degeneration of root tips.

No attempt has been made in this brief discussion of a complex subject to present cultural recommendations. However, this information is presented in the firm belief that only as we increase our knowledge of grass growth and the factors affecting growth can we intelligently revise our management practices to produce better quality turf.

REFERENCES