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Proceedings of Scotts Turfgrass Research Conference

Volume 2 - Turfgrass Diseases

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PREFACE

This volume contains the proceedings of the second Scotts Turfgrass Research Conference. The sessions of this conference were devoted to turfgrass disease--their nature, impact and control. Representatives from 31 states and Canada joined with the research staff of O. M. Scott & Sons Company at Marysville, Ohio, on June 16-17, 1969. Seven formal papers were presented; each followed by a question and answer period. In addition, a special session was devoted to a general discussion of problems related to turfgrass pathology.

By far the largest number of turfgrass diseases are incited by members of an insidious horde of pathogenic fungi. However, one of the papers presented here describes St. Augustine Decline, a new virus disease of lawngrass.

Keeping turf healthy is a challenging, major responsibility. The contents of this volume bear testimony of the complexity and variability encountered among turfgrass pathogen. An evaluation of the biology and control of turfgrass diseases is both timely and worthwhile. We trust the information presented here will prove interesting and useful to all those concerned with growing high-quality, disease-free turf.

Philip M. Halisky
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ENVIRONMENTAL FACTORS INFLUENCING TURFGRASS DISEASES

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Almost all turf-forming grasses are subject to serious diseases, particularly when maintained under close clipping. A knowledge of the characteristics of these diseases, and of the best methods for their prevention and control is important in successful turf management (Musser, 1950). Environmental factors that produce morbid conditions in growth and development of turf cause disorders referred to as physiogenic diseases. Such diseases are incited by environmental stress and include: extremes in soil moisture content; unfavorable atmospheric and soil microclimate, and chemical or mechanical injuries. Environmental factors that produce physiogenic disease also create conditions that predispose host plants to pathogenic microorganisms. In all instances an interrelationship exists between the environment, the pathogen, the host and disease development.

Environmental Conditions Favoring Fungus Diseases

Climate and weather have a great influence on disease. Climate determines whether a pathogen can flourish or persist under normal conditions in a given locality. Micro-climate determines whether a host-pathogen relationship will develop into disease. All stages in the life cycle of a pathogen take place within fixed temperature ranges.

Temperature appears to be the limiting factor in the behavior of some diseases. Humidity appropriate to the requirements of a pathogen is necessary for all its active stages. Low humidity ordinarily retards or prevents the development of a pathogen. Few plant diseases are damaging under conditions of consistently low humidity. Within limits, an organism's need for moisture varies with the temperature (Nat. Acad. Sci., 1968).

Moisture

The fungi that cause turfgrass diseases need liberal quantities of moisture to germinate spores and sclerotia, and to keep mycelial strands growing actively. The latter are very delicate and cannot withstand drying out. Saturated soils and high air humidity create ideal conditions for their rapid development. Poor drainage, heavy watering and excessive rains that keep soils waterlogged for long periods increase the chances of fungus infection. Humid air and heavy dews keep the foliage wet and also favor fungus growth. Pockets of stagnant air that occur where there is poor air drainage contribute to disease development. In this regard, landscape planning is important (Musser, 1950). Excessive moisture produces lush grass and a more favorable microclimate for disease development. An added factor in waterlogged soil is the inability of the grass to recover from injury because of low nutrient availability and shallow, restricted root-system development.

Soil Moisture

Low soil moisture increases accumulation of toxic ions, such as manganese and boron, causing tissue damage. It can also cause stomatal closure by creating a water stress from the soil system through and including the plant system.

High soil moisture leads to a lack of oxygen in soils. Over long periods, this soil moisture can make plants so succulent they become particularly sensitive to invasion by certain pathogens (Nat. Acad. Sci., 1968). Soil moisture affects both soil-borne and air-borne pathogens. High moisture can lead to root suffocation and injury through reduction in oxygen content. Drying of soil is generally accompanied by reduced pathogen activity, because most fungi go into resting stages as free water disappears from the soil. Temperatures drop further and faster in a dry soil during the winter; therefore, winter kill is prevalent in dry winters.

Temperature

Excessive moisture alone will not cause fungus attacks. It must be accompanied by temperatures that are favorable for development of the disease-producing organisms. Like other plants, each fungus has an optimum temperature for growth. The brown patch organism, Rhizoctonia, grows best at the relatively high temperatures of midsummer. Snow mold, Fusarium nivale, represents the other extreme. It causes the most severe damage in the late winter and early spring when temperatures are close to the freezing point. Diseases, such as dollar spot and leaf spot, develop over a much wider temperature range than brown patch or snow mold, but they are apt to be more severe during cool, wet periods in the late spring and early fall.

Soil Temperature

Temperature affects the rate of moisture absorption. High soil temperature can induce disorders such as basal lesions, soil-cracking and root damage. The freezing of soil can directly kill the roots of many tropical and subtropical plants. Persistent and unseasonably low soil temperatures usually stunt plants. Soil temperature may affect disease either by its effect on the host or on the soil-borne pathogen. Cold soils affect mobility of nutrient elements and may cause temporary chlorosis. Therefore, soil temperature is closely connected with abiotic diseases.

Soil Texture

Soil structure can also affect disease development. Turf grown in heavy, compact and poorly-drained soils usually shows greater losses from Pythium and Fusarium than does turf grown in well-drained soil (Altman, 1966). Soil type may influence amounts of CO_2 in the soil, a change in the balance of microorganisms, lack of available nutrients or other factors.

Light

The diurnal light rhythm affects the periodicity of spore release, partly because of its direct effect and partly because of changes in temperature, humidity and air movement that accompany changes in light intensity at dawn and dusk.

The shade microenvironment is also very important in disease development. Shade adversely alters the turfgrass microenvironment.

It is the more favorable microenvironment of shaded conditions plus the lack of disease resistance that results in the severe disease problem. The microenvironment of shade which encourages disease activity includes: (1) Higher relative humidity; (2) extended dew periods; (3) reduced light intensities that produce a more succulent growth, and (4) low light intensities resulting in lowered respiration rates and, consequently, lower energy levels in the host which restricts cell wall development and maturation. Thus, thinner cuticles and underdeveloped cell walls are produced. The juvenescent state of the host is prolonged and physical mechanical barriers to pathogen penetration are reduced.

Low light intensities do not affect stomatal opening. It seems reasonable to assume that 1,000 ft candles (10% of full sunlight) would be sufficient to open stomates since Zelitch (1961) has shown that only 250 ft candles of light intensity will achieve maximum stomatal opening in tobacco. Therefore, the shade microenvironment that affects the host, such as lower sugar transport under low light intensities, thinner cuticles and a more intense hydrosphere above and around the stomata, are more relevant in the disease syndrome than stomatal opening. Helminthosporium vagans is favored by low light intensities. Lukens (1968) refers to this pathogen as the low sugar disease pathogen since it is more severe when sugar levels in the host are curtailed.

Soil pH

No single characteristic of soil is more significant than its pH. Acidity is one reason for the thatching of turf, although not the sole cause (Musser, 1950). Grasses renew a large part of their root system each year. When soils are not too acid, the old roots slough off and decay due to indigenous soil bacteria and fungi. Such decay tends to reduce thatch.

However, in acid soils the dead roots, stems and leaves accumulate forming a thatch, due to lack of microbial activity. This thatch impedes the penetration of air and water and is largely responsible for the formation of localized dry spots in turf. It also creates an excellent medium

for pathogenic fungi. Helminthosporium thrives on dead organic matter (Altman, 1965).

The pH affects the number of earthworms, bacteria and fungi present in the soil. While fungi develop over a wide pH range, they are most prevalent under acid conditions (pH 4-5). Many of them are desirable and necessary because they are responsible for the initial decay of organic matter, but certain groups are disease-producing. The dollar spot fungus and brown patch fungus, for example, are stimulated by excessive acidity and are checked when acidity is controlled by proper liming in certain areas of the country.

Microbial competitors that keep the disease-producing fungi in check are more active when soil reaction is near neutral. Soil reactions below pH 6.0 tend to favor turf disease fungi. It is often practicable to reduce the severity of disease outbreaks in such turf by light dressings of hydrated lime every 3 or 4 weeks during the time when disease is likely to occur. This practice is safe when soil reaction stays below pH 7.0, but will cause iron chlorosis and trace-element deficiencies when the reaction is pH 7.5 and above. Lime should not be used in the semi-arid sections.

Thatch

A heavy mat of spongy turf provides ideal conditions for the growth of disease-producing organisms. It always contains large amounts of dead leaves and stems which absorb moisture readily and remain damp for long periods. This condition is favorable for the growth of fungi and increases the difficulty of obtaining good control with fungicides. Where turf is heavily thatched, it is often necessary to use excessive water to obtain adequate penetration with a fungicide. Normal fungicide treatments would be too dilute to be effective. To achieve effective disease control, heavier rates of chemicals are required, but these may discolor the turf.

Winter Injury

The most important types of winter injury to turf are desiccation (dehydration) and freezing-out. Desiccation is common in regions where there is limited rainfall and soil moisture is low during the winter months. It is aggravated

by dry, cold winds. The dry soil and dry air draw so much moisture out of the dormant or semi-dormant grass plants that they shrivel and die. Injury of this type occurs on both greens and fairways and is most severe on knolls and other exposed areas that are blown free of snow. The damaged grass first has a dull-brown color which may bleach to grayish-white by spring. The best method of avoiding winter injury by desiccation is to moisten the soil throughly to a depth of 5 to 6 inches late in the fall and again in winter if rain or snow fall are scarce. It is common practice in northern dry areas to place tree branches and brush on windswept greens to collect and hold snow during the winter.

In Colorado, particularly on the eastern slope of the Rockies, many golf courses are open for winter play. In addition to winter watering of greens, the use of various acrylic turf colorants is increasing. These colorants are composed of polyvinyl acrylics containing a green dye. Treated greens are less prone to desiccation and disease. Less snow mold occurred on 50 greens in continuous play during 1967 and 1968 that had been treated with turf colorant and fungicide as compared to greens treated only with fungicide. Greens treated with the combination were less brittle and had better ball retention qualities than non-treated greens. Non-treated aprons were infected with Fusarium snow mold in March and April of each year.

Winter injury of turf due to freezing-out, as distinct from desiccation, is caused primarily by poor surface or subsurface drainage. It is often aggravated by the use of poorly adapted grasses and by management practices that weaken the turf and make it less tolerant to adverse conditions. Poor surface drainage causes water to collect in depressions. The frozen soil prevents it from draining out, even when its physical condition is satisfactory. Acccumulations of snow and ice may produce the same result by damming back the water. The alternate freezing and thawing of such pools causes winter killing of the grass.

A good program of turf maintenance during the growing season often prevents or reduces winter killing. The use of grasses that are cold hardy or adapted to the conditions under which they must be grown in an important factor. For example, when Kentucky bluegrass is destroyed, because of saturated soils on spring seepage or ponded areas, Colonial or creeping bentgrasses should be used. The bents are more

tolerant of wet soils and will survive longer under such conditions. The various kinds of bents differ in cold tolerance. Some of the newer vegetative strains, such as Toronto and Old Orchard, are more resistant than other types. Seaside is very susceptible to freezing injury.

Sound fall fertilization will help reduce winter injury provided nitrogen is not applied after mid-September. One pound of nitrogen per 1,000 sq ft will provide a tough grass that is less susceptible to winter damage. Reduced watering also helps to harden the turf and put it in good condition for winter; however, many turf areas will require late fall and winter watering, particularly in arid areas similar to eastern Colorado.

Summer Injury

Turf is subject to many types of injury during the growing season that may be mistaken for disease attacks. These may be due to unfavorable weather and soil conditions or to inadequate maintenance. Scald is a common trouble of this kind. It occurs as irregular areas of discolored turf on poorly drained soils or in depressions on greens during periods of excessive rainfall or when the grass is watered heavily in hot weather. Thorough aeration of the damaged areas to hasten drying and permit air to get down to the roots is a temporary remedy and may save some of the turf. The only permanent remedy is to provide adequate surface and subsurface drainage. The use of lateral stone drains 4-6 in. wide and 12-18 in. deep will help.

Localized dry spots may develop on greens and fairways where the turf suffers from lack of moisture, even when irrigated regularly. This condition may be caused by excessive compaction or because of thatch accumulation. Localized dry spots should be thoroughly aerated and lime and fertilizer added where needed to hasten organic matter decay. They should be watered regularly until normal soil moisture has been restored.

In regions subject to high temperatures and hot dry winds, turf may be seriously injured because of wilting. This type of injury takes place when the grass roots cannot absorb moisture

from the soil as fast as it is lost from the leaves. The first indications are the development of a dull, bluish-green color and severe footprinting on the turf. Wilted turf recovers very slowly, and in serious cases the leaves may shrivel and die. Injury can be avoided by frequent, light sprinkling (syringing) of turf, two to three times daily, to provide readily available moisture to reduce turf and soil temperatures. This type of watering cannot replace normal irrigation to replenish the moisture supply throughout the root zone.

Turf may lose its vigor and thin out because of tree-root competition. This competition can be eliminated by ditching between the putting green and trees that are sources of trouble, or by the periodic use of a deep running single-blade root cutter.

Turf of low vigor, resulting from one or more factors that induce summer injuries, is usually predisposed to red thread and dollar spot. Rhizoctonia brown patch and Pythium grease spot also occur as high temperature diseases in the U. S.

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DISCUSSION PERIOD

Dr. Wadsworth: I was intrigued with your comments about the Pythium blight outbreak on a golf course that was 11,000 feet high. Do you happen to recall what the average temperatures were about this time, and whether or not this was on mature grass or newly-seeded grass?

Dr. Altman: It was on newly-seeded Penncross. The temperatures during the day were between 50-60oF and at night around 32oF.

Dr. Wadsworth: Would you normally expect this type of outbreak with those temperatures in that area?

Dr. Altman: No, not with Pythium blight. However, what is normal for disease in certain areas of the country is not normal with regard to Colorado. Brown patch normally occurs in warm, moist periods, but I have noticed it on our grass in Colorado in late September and October when everything is dry. It may be that there is some selecting out of the Pythium and Rhizoctonia strains that don't react the way they normally do under much warmer temperatures and higher humidities. However, the humidity in this area was pretty high, and the soil was constantly moist. The soil was a silt loam that came out of a lake bottom. I agree that the temperatures were unusual for Pythium development.

Dr. Wadsworth: We've seen some Pythium outbreaks at rather low temperatures in the Oklahoma area but this has been after we have had periods of high humidity and warm temperatures. Once the inoculum level was established, then all that was needed was a period of relatively high moisture for several days.

Dr. Altman: I know that the weather was not warm prior to this Pythium occurrence. We are in the process of undergoing a tremendous outbreak of Helminthosporium vagans and Fusarium roseum in Colorado. In the early part of May we had quite a bit of moisture and very cool temperatures. Grass began to go down rather rapidly toward the latter part of May. In the last two weeks we've been getting reports of disease from many areas of the eastern slope of Colorado. When cultured, almost all the samples are Fusarium roseum and it's one of the few times that I've noticed such a high

incidence of only Fusarium. In these instances, perhaps we had the right moisture and temperature in early May to build up inoculum levels of Fusarium. These conditions are the same as those that occurred prior to the Pythium outbreak.

Mr. Benedict: You commented on the various ferrous sulfates used on Merion under high temperatures, causing the turf to turn black. What was the cause of that? How long did this last and what was the final result of this treatment?

Dr. Altman: This black condition persisted for well over a month after application. All the leaf surfaces of the grass which had been treated, curled and seemed to be burned off; it just stood still. The grass recovered after several waterings but we were without any blades of grass in this area for at least a month, and in some spots up to six weeks.

Mr. Benedict: Do you expect any varietial differences to this effect?

Dr. Altman: I don't know. It just happened to be all Merion in this one instance.

Dr. Carlstrom: What were the symptoms of the Helminthosporium you are seeing this year?

Dr. Altman: We're getting some crown killing and also some long, purple lesions with dead centers. Also, for the first time, we're getting a tremendous amount of melting-out.

Dr. Carlstrom: Did you see any Pleospora in conjunction with Helminthosporium?

Dr. Altman: No.

Dr. Carlstrom: We saw this in Oregon two years ago.

Dr. Altman: In culture we are getting pure isolates of Helminthosporium. No perfect stage with the Fusarium, only Fusarium roseum and nothing else. You can find spores only on the tillers of the grass.

Dr. Carlstrom: Are you finding any Curvularia in conjunction with the Helminthosporium?

Dr. Altman: No, this is one that I have been considering for a long time. Some researchers suggest that Curvularia may be an immature Helminthosporium which occasionally produces a bent spore. I haven't seen any in our area.

Dr. Carlstrom: What were the conditions conducive to Helminthosporium coming on? What were the weather conditions?

Dr. Altman: Temperatures between 60 and 80 degrees and high humidity promotes the disease. For the past 2 to 3 weeks our weather has been moist and cloudy with very little sunshine.

Dr. Carlstrom: Was it following a dry period when you had the increasing moisture?

Dr. Altman: Yes, we had probably a month-long dry period and then we had moisture.

Dr. Weihing: You mentioned that you get Helminthosporium vagans in your isolations. Do you ever pick up Helminthosporium sativum? This is our common one and we rarely get H. vagans.

Dr. Altman: Yes, we get H. sativum but I would say approximately 80% of the turf samples are Helminthosporium vagans.

Dr. Weihing: Do any of those samples come from the more eastern locations?

Dr. Altman: They are coming from the Denver and Colorado Springs areas.

Dr. Worf: I am interested in knowing whether you would be aware of a definitive study that concerns the breakdown of thatch and how this might be related to associated organisms, particularly bacteria and actinomycetes.

Dr. Altman: I think they are important in the breakdown of the cellulose material. The bacteria can take over after the cellulose is broken down. You might build up some thatch, in the absence of fungi, particularly if you have a nitrogen-deficient turf. Furthermore, if you are using a fungicide for disease control, it's not only killing the pathogens, but also

the fungal saprophytes. Here again, there is the possibility of a thatch build-up. I've been comparing clipping removal, versus no removal, of Merion in soil that was in agricultural production for the last 30 years. After five years, there was absolutely no difference in the thatch accumulation. The unique thing about this soil is its high microbial activity. I have tried the same thing on home lawns where excavated material has been used as soil, and for the first four or five years you do get a tremendous build-up of thatch. In the older lawns when turf is being replaced, this excavated soil becomes modified. It has a little more microbial activity and the thatch doesn't seem to accumulate quite as rapidly. But on farm soil, where I have about 8,000 sq ft of Merion, I have not been able to see any difference.

Dr. Worf: In our observations, the removal of clippings has very little influence on the development of thatch. With respect to the thatch decomposition, would we be reasonably safe in assuming that there is a broad spectrum of fungi involved in the initial breakdown process? Many of our fungicides are rather specific in the spectrum within which they work.

Dr. Altman: Yes, there would be. For example, you would find Tricodermas that are not affected by some of the fungicides. Green and white Tricodermas, capable of breaking down this material, are present. Although I indicated that it's possible for the fungicide to restrict fungal activity, including saprophytic activity, I think there are ample numbers of fungi that are not inhibited and can continue to break down cellulose.

MODE OF ACTION OF THE DISEASE ORGANISM

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Recent contributions to the study of turf pathogens have been substantial. Four processes have been partly described: (a) arrival of the pathogen at the site of infection, (b) penetration of the grass, (c) growth in grass tissue, and (d) build-up of inoculum for an epidemic of disease. In addition to what is know, areas of little knowledge also will be emphasized. An understanding of the mechanisms by which pathogens cause diseases is necessary in order to devise more effective measures for control.

Arrival of the Pathogen at Infection Site

When disease breaks out in an area, the pathogen is usually not freshly introduced, but is already established. In a new lawn, the source of pathogen is infected soil, sod or seed. Once the disease breaks out, infected clippings can be distributed by foot, mower or other equipment to uninfected areas of turf. With rust diseases, the fungus is carried in the air.

Turf is a unique crop, and its uniqueness makes it easy for the pathogen to travel to the site of infection. The plants are arranged in a close compact carpet of thickness three-sixteenth of an inch in putting greens to two inches or more in lawns. The entire crop is wetted daily with dew because the vertical arrangement of leaves provides no canopy to prevent the formation of dew on other parts of the plant. Also, the vertical fibrous structure of the carpet is conducive to formation of large amounts of dew.

Air-borne Inoculum

The common air-borne pathogens are those that cause rust and powdery mildew. Others whose spores may travel by air are the leaf spotting and blighting fungi--Helminthosporium, Colletotrichum, Cercospora and Fusarium. Aeciospores of rust pathogens are carried long distances by air from the alternate host to leaves of grass. Urediospores produced on grass are air-borne also. Conidia of the powdery mildew pathogens are carried by breezes or propelled by rotary mowers and settle on leaves of grass. Stripe smut disease produces a black cloud of chlamdospores which, when propelled by rotary mowers, drifts into the house and is a problem for homeowners.

We have trapped conidia of Helminthosporium from air one foot above the turf. Because spores of Helminthosporium are large, compared with those of mildew and rust pathogens, the pathogen may travel short distances in the air down-wind from the disease site. Conidia of Fusarium nivale are blown by wind from the source of production to new infection centers (Couch, 1962).

Spores are discharged into the air from infected turf when it is dry. Thus, transport of air-borne spores is restricted to clear days from midmorning to early evening when the turf is free from dew. Moisture on diseased leaves retain the spores and prevents them from becoming air-borne. We do not know if spores borne in moisture can become air-borne upon drying. If only spores that are formed without free moisture become air-borne, spore production and transport will have to be accomplished during the same day. This information is critical in devising a proper schedule for applying foliar-protecting fungicides.

Hyphae of Fusarium, Sclerotinia, Helminthosporium, Pythium, Rhizoctonia and Colletotrichum grow out from diseased tissue when the tissue is moist. During epidemics, it is common to find hyphae growing from leaf to leaf on dew covered grass. The extensions of hyphae into the air between leaves may be considered a mode of air-borne inoculum over minute distances.

Water-borne Inoculum

Water contributes to the transport of inoculum in two ways: (1) spores, hyphae and sclerotia are carried to the host by flow and splashing rain drops; and (2) mycelium is encouraged to grow in water films, drops of dew and guttation droplets on the host to uninfected sites. Water-borne inoculum is a major means of transport for turf pathogens.

Spores of Helminthosporium, Colletotrichum, Cercospora and Fusarium may be splashed from the source to uninfected turf. The force of a rain drop, the distance of travel and the importance of inoculum traveling in splashing drops of water are not known. Such information may be important in choosing the size and speed of water droplets when irrigating turf.

Fungi grow and move in a water environment on sod. Zoospores of Pythium aphanidermatum swim in films of water to uninfected turf to cause grease spot disease (Kraft and Endo, 1966). Rhizoctonia solani grows saprophytically in moisture on turf. Drops of guttated water at the ends of cut blades are important in brown patch disease (Rowell, 1951). Wet turf is important for mycelium of Helminthosporium vagans to invade the crowns of bluegrass in spring (Couch, 1962). The requirement of soggy turf for snow mold diseases is indicative of the necessity of a water film for the pathogen to reach the grass.

Refuse-borne Inoculum

The distribution of contaminated clippings and soil over turf by foot or implement occurs when sanitation procedures become lax. Both Pythium blight and melting-out diseases have appeared in streaks on putting green turf where small amounts of clippings have been allowed to dribble from the grass catcher or mower when golf course greens were groomed. Mowers may carry inoculum from one green to another. On several occasions, melting-out disease has been restricted to greens that were mowed with the same mower.

Penetration of the Grass

Fungi are restricted to particular sites of entry and gain entrance by certain physical and chemical means. Rowell (1951) has shown that Rhizoctonia solani enters leaves of bentgrass through the cut ends when the grass is clipped. Unclipped grass inoculated with hyphal fragments did not succumb to disease. Couch and Bedford (1966) have reported that Fusarium roseum can invade turf grass through the cut ends of leaves. Disease has been observed to progress from the cut end of bluegrass leaves that were placed in a moist chamber and dusted with spores of Curvularia spp. The fact that wounds from mowing can serve as sites of entry for pathogens emphasizes the need for sharp mowers to avoid enlarged shredded wounds of grass leaves.

Fusarium nivale, Corticium fuciforme (the cause of red thread disease), Helminthosporium vagans and H. sativum can enter grass leaves through the stomata (Couch, 1962; Mower, 1961). Presumably, most fungi capable of growing appressoria can, by chance, penetrate the leaf by way of the stomata. In the process, the hyphae from germinating conidia or mycelium form a pad or appressorium which presses against a stomate and inserts a peg through the pore. Once inside of the stomatal cavity, the fungus grows hyphae of normal width. Activity of Helminthosporium is restricted to the stomatal cavity with only a few parenchyma cells being invaded (Mower, 1961). With Helminthosporium, entry into bluegrass through the stomata is a minor mechanism for disease initiation.

Direct penetration of cuticle and epidermal layer of cells with the aid of the appressorium and infection peg is a common mechanism of many turf pathogens. Notably among these are Helminthosporium, rust fungi, Erysiphe graminis and Rhynchosporium secalis (the cause of scald). In the process, appresoria formed at the terminals of germ tubes and hyphae, press against the cuticle and inserts pegs through the waxy cuticle and epidermal cells. With Helminthosporium, the pegs go between the epidermal cells, and with Erysiphe graminis the peg penetrates into the epider-

mal cells (Mower, 1961; Couch, 1962). Zoospores of Pythium aphanidermatum form appressoria and penetrate roots of bentgrass by means of penetration pegs (Kraft et. al, 1967).

The process of penetration requires moisture and nutrients and may be aided by extracellular enzymes of the pathogen. Conidia of Erysiphe graminis require humidity approaching saturation but not free water to germinate and make ingress. Nutrients from the host seem to be required because the presence of a grass leaf stimulates spore germination and appressorial formation. Humidity and free moisture are required for the penetration process of most pathogens. With Helminthosporium vagans, the process takes place in 18 hours and free moisture is required (Mower, 1961; Lukens, 1970). Nutrients from guttated water stimulate spore germination and appressorial development of H. sorokinianum and Curvularia spp. (Endo and Amacher, 1964; Healy and Britton, 1968). The active ingredient in the guttated water is glutamine, a transient nutrient produced by turf in large amounts following nitrogenous fertilization of the grass (Curtis, 1944). The amount of guttation water increases with increase in water content of the soil. Thus, both soil fertility and soil moisture can play a direct role in the effectiveness of pathogenic fungi attacking turf grasses. The addition of glycine to spore suspensions of Helminthosporium vagans increases the number of leaf spots produced on inoculated bluegrass leaves. Most fungi carry nutrients in spores and other dormant structures, however, a supplement of certain nitrogenous materials from the host may encourage these fungi to penetrate the host.

Pathogens that macerate host tissue do so through activity of extracellular enzymes which degrade the cement between cells of host tissue. Several pectinases and cellulases have been found in bentgrass blighted with Pythium ultimum (Moore, 1965). The appressorial peg may penetrate, in part, by means of enzymic degradation of substances between epidermal cells. Helminthosporium vagans produces pectinases in vitro in the absence of glucose (Patil, unpublished data). Glucose inhibits synthesis of the enzymes and protects bluegrass against invasion by H. vagans

(Lukens, 1968). Indeed, the chemical mechanism of host penetration by turf pathogens needs more investigation. The knowledge may be useful in devising measures for the control of disease.

Growth of Pathogen in Grass Tissue

Growth of fungi in host tissue is accomplished by hyphae which grow into and between cells. The fungus invades by haustoria within the cell into which host nutrients permeate. The obligate parasites, Erysiphe graminis and Puccinia spp., grow haustoria without disturbing the delicate integrity of the host cell until the late stages of disease (Couch, 1962). Ustilago striiformis grows systemically within the grass plant without symptoms of disease. Hyphae of Rhizoctonia solani can ramble through leaf tissue of bentgrass with no outward symptoms until the water stress becomes acute and the entire leaf suddenly collapses. Rapidly wilted leaves turn black and cause the smoke ring typical of brown patch. Helminthosporium species which grow haustoria within host cells cause the collapse of the host protoplast shortly thereafter (Mower, 1961). With Pythium, host cells collapse within an hour of penetration of the fungus (Kraft et al., 1967).

Hypha of Helminthosporium grow intercellularly through palisade and mesophyll of the leaf to the vascular system. Along the way, parenchyma and sclerenchyma cells are invaded by haustoria of the fungus. The presence of the pathogen causes collapse of host cells within a day of invasion. However, little necrosis proceeds beyond the space occupied by the fungus. This suggests that if toxins are involved in the death of the host cells, the toxins are not abundantly produced by the fungus and do not permeate beyond the immediate area of infection. On the other hand, with Victoria blight of oats, which is toxin-incited, yellowing extends to the terminals of leaves and stem from the point of infection (Luke and Wheeler, 1955).

The mechanism of invasion of host tissue determines, in part, the size and appearance of the lesion. Pinpoint lesions of Pythium on roots of bentgrass develop from the few cells that collapse immediately after invasion by the pathogen

(Kraft, et al. 1967). Necrotic spots of Helminthosporium and other leaf-spotting pathogens arise from the limited invasion of the pathogen (Mower, 1961). Apparently, the leaf-spotting pathogens require moisture for further infection and the host cells that are collapsed lose water quickly and dry out. Pathogens that macerate tissue are able to extend the size of lesions because moisture is conserved in the macerated diseased tissue.

Succulent growth of grass from high soil moisture and high nitrogen fertility is conducive to Rhizoctonia brown patch and Helminthosporium blights. Increase in disease by succulent growth may be caused, in part, by an increase in infectivity of the pathogen. The cuticle and walls of cells are thinner so that barriers to host infestion are weak. The pathogen can grow more rapidly in the enriched secretions of leaves of highly fertile turf. The production of simple carbohydrates from photosynthesis in succulent tissue is less than that from hardy tissue. The pathogens utilizing extracellular enzymes growing through host tissue are not hindered by sugars in synthesizing these enzymes. Melting-out disease of Kentucky bluegrass is a low sugar disease (Lukens, 1970). The area of foot-rot infection in turf is proportional to the percent of solar radiation shaded from turf (Fig. 1). The reduction in sugar content was consistent with the reduction in solar radiation. A short cutting height increases melting-out (Halisky et al., 1966). Leaves from turf of low cut contain less reducing sugar than leaves of turf of higher cut. Moreover, an analysis of data from disease and sugar content of leaves of five bluegrass varieties at two cutting heights gives a correlation coefficient of 0.96 (Fig. 2). An application of glucose to Kentucky bluegrass turf reduced the rate of disease development for about a month (Lukens, 1968). Hence, by altering the sugar content of grass leaves with shade, cutting height, variety and sugar spray, disease increased inversely with the content of sugar in the host.

Glucose sprayed to turf caused a delayed increase in disease. Apparently, the pathogen had grown saprophytically on the sugar in sod. Inoculum from this sugar-induced growth was sufficient to overcome the resistance of the turf conveyed by an increase in sugar content in the host. A

Fig. 1.

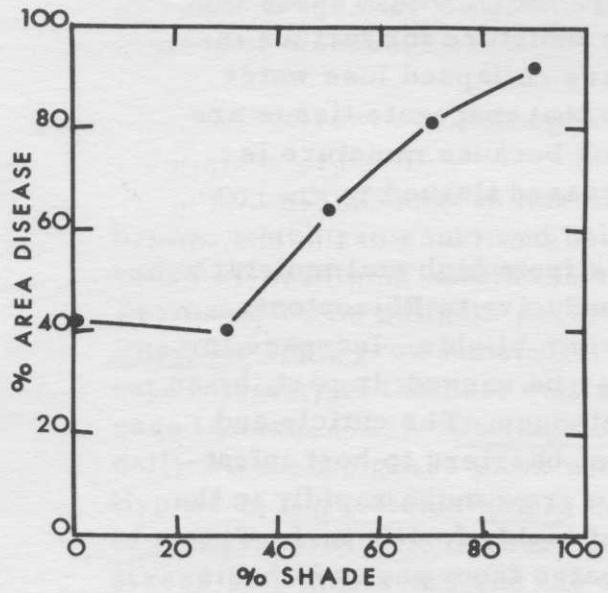

Fig. 1. Effect of shade on the incidence of infection of Kentucky bluegrass turf by Helminthosporium vagans after four weeks of treatment in May (Lukens, 1968).

Fig. 2.

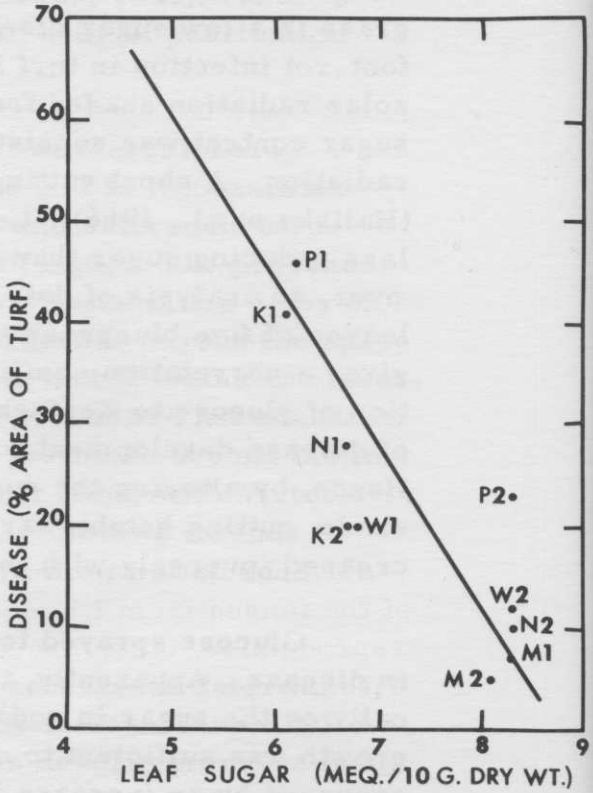

Fig. 2. Effect of leaf sugar on melting-out by Helminthosporium vagans. Variety of bluegrass: P = Park; K = Kentucky; N = Newport; W = Windsor; and M = Merion. Cutting height: 1 and 2 in. respectively. The curve is the regression line of leaf sugar on disease.
b = 160**.
(Lukens, 1970).

sugar which conveys resistance to the host but does not serve as a carbon source for the pathogen is needed to effectively control this disease.

Build-up of Inoculum

Large amounts of inoculum are required to sustain an epidemic of disease. The inoculum is produced by the pathogen from mycelium in diseased tissue of the host or in refuse in the sod. The build-up of inoculum in turf occurs from several cycles of disease; each cycle requires about three days. In the disease process, mycelium builds up in lesions of a certain size. With powdery mildews, infection is superficial and the mycelial mat or stroma is produced externally on the leaf. Conidiophores grow out from the stroma and, shortly thereafter, conidia are produced. Pustules of uridiospores of rust are born superficially on leaves of bluegrass. With Helminthosporium, the stroma is produced in the diseased tissue and conidiophores are borne on the outer surface of the host. With stripe smut, the fungus grows systemically from crown to leaves in which it produces a mass of black chlamydospores. The leaf ruptures and the spores are exposed to air currents.

Various pathogens that are able to live as saprophytes can produce inoculum on decayed clippings and organic matter in sod. Inoculum from overwintering material may be sexual spores, asexual spores, sclerotia or merely hyphal fragments. Certain of these inocula are also produced during the growing season. Hyphal filaments of Helminthosporium, Pythium, Fusarium and Rhizoctonia growing from refuse to the host in moist sod are an important source of inoculum for disease. The amounts of hyphal inoculum from these sources is dependent upon moisture, temperature and the nutrition of the refuse.

Sporulation in fungi accompanies a change in growth of the fungus from filamentous to a budding type. In sexual reproduction, other habits of the pathogen enter the picture. With change in growth habit there occur changes in metabolism and, in turf, changes in response of the fungus to environment. Helminthosporium vagans grows mycelium at its maximum rate of 25° C. (Horsfall, 1930) and sporulates at

15° C. (Lukens, 1968). Hence, spore inoculum is produced by H. vagans most abundantly in cool weather and pathogenesis advances at a faster rate in moderate weather. These opposing effects of temperature may explain, in part, the lack of a clear relationship between temperature and melting-out disease (Bean and Wilcoxon, 1964).

Conclusion

Mechanisms of fungal attacks on turf grasses have been described. The fungus, in the form of hyphae, sclerotia and spores, arrive at sites of new infections by air, water or debris. It infects a grass plant through cut ends of leaves through stomata, or directly through the cuticle. The fungus grows special structures – the appresorium and peg, for infecting grass. The development and functions of these structures require moisture, nutrients and possibly extracellular enzymes. Hyphae of the pathogen grow between cells of grass tissue and small branches of the hyphae penetrate into host cells. Haustoria, cells for absorbing nutrients from the host protoplasm, are produced inside of host cells by the fungus. Host cells tolerate haustoria of obligate parasites, but they collapse when infected by haustoria and hypha of other pathogens. Fungi may kill host cells some distance away from the invading hypha by excreting substances that are toxic to the host. Soon after establishment in the grass plant, the fungus grows a mat of mycelium from which more inoculum in the form of hypha, sclerotia and spores are produced. Usually, epidemics of disease break out following several rapid cycles of dispersal, penetration, infection and inoculium production of the pathogen.

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DISCUSSION PERIOD

Dr. Endo: I was interested in your correlation with low sugar content in relation to Helminthosporium. I was thinking of an alternative hypothesis. I have inoculated leaves of Kentucky bluegrass at different ages with Helminthosporium and found a difference in behavior of the germ tubes on senescence leaves, the germ tubes grow directly to the stomata. In 24 hours the epidermal cells are thoroughly ramified with mycelium. On very young leaves the germ tubes are very long, few appresoria are formed, penetration is rare, and colonization is usually limited to a single epidermal cell.

First of all, the germ tubes react differently according to the age of the leaf; penetration is different and colonization varies. When plants are shaded, senescence and cell membrane permeability may be increased, favoring the exudation of inorganic and organic compounds. I was wondering whether the leaching of nutrients from the shaded plants could have influenced Helminthosporium. Also, the behavior of proteins in senescent leaves might be different and, therefore, the host defense mechanisms might also be affected.

Dr. Lukens: In your work with the guttated nutrients, would you say that different age leaves from the same plant have different characteristics, and that nutrients exuded to the surface vary? Guttation has a great bearing on disease.

In reference to our shade work, the grass in 70% and 90% shade grew so fast in height that I complained to the farm manager, who had always taken good care of my turf, that he wasn't mowing the grass faithfully. After all, he never let the grass get six inches high. I went to get samples one Friday, when the turf was just mowed, and when I returned the next Monday, the grass in 90% shade had grown 6 to 8 inches. Turf in the open sun grew less than one-fourth of an inch. The turf was not neglected. Shading had caused etiolated and very succulent growth with little indication of senescence.

Of course, shading does change the protein structure. Dr. M. Zucker at our laboratory, working on proteins of tobacco and potatoes, found perhaps 90% of the protein in the leaf contained in the chloroplast. Since shading reduces chlorophyll formation, we are altering the chemical composition of the plant. I don't deny that. However, we found a good relationship between leaf sugar and disease. This relationship may just be an outward expression of something more subtle. At least it is a hypothesis that works. I'm trying to relate it to some kind of mechanism.

Dr. Altman: With regards to shade and Dr. Endo's comment on senescence, I think the shade grasses have a thinner cuticle and, also, inside the substomathal chamber there is a thinner cuticle lining. The barrier to entrance by the appressoral peg is reduced in shade so you may not have to have a variation in senescence. I'm inclined to think that you get this increased elongated type of growth, rather than senescence, and more of a juvenile stage. Maturity and hardening off processes are not as well-developed under shade as they are in full light. This may be part of your story. What I wanted to ask you about was glucose. I found that those plants that exude more glucose at the hypocotyl area are more susceptible to Rhizoctonia. I'm talking about broad leaf plants near the soil-hypocotyl interface. What is the relationship of this to your suggesting glucose as a means of control of disease?

Dr. Lukens: Glucose can reduce infection from Helminthosporium, but glucose outside of the plant appears to stimulate the pathogen to grow saprophytically. The high level of inoculum produced may overcome any effect one may induce in the plant by the sugar supplement. I rated the disease from week to week for eight weeks to calculate the increments of increase in disease per day. During the first few weeks the disease was reduced proportionally by the long concentration of the sugar in the spray. But after four weeks the whole thing fell apart. The organism grew saprophytically on sugar and this upset the whole balance. Subsequently, disease was increased by the sugar supplement.

Dr. Altman: Under the shade microenvironment, in the sphere that surrounds the stoma, there's usually a higher

oxygen content. Could this be involved in getting more direct or mechanical penetration?

Dr. Lukens: I'm relying mostly on Dr. Mower's work. In his thesis he studied H. vagans and H. sativum. I think roughly 80% of the infections in common Kentucky bluegrass were by direct penetrations and about 20% through stomata. Infections of the stomates were pretty much limited to the area around the stomatal cavity. With Merion bluegrass, most of the infections were through stomatal cavity; very few through the direct penetration of the cuticle. I didn't emphasize this in trying to explain the resistance of Merion to the disease. It's probably one means, among many, which makes Merion fairly resistant to the Helminthosporium melting-out disease. Apparently, the disease also is related to the nature of plant growth. Sugar may be one reason; thin cuticle may be another. They all play a part.

Dr. Pellett: Most of the discussion during the last few minutes has been centered around the physiological and anatomical relationships of the host. I'm wondering what the direct effect of the quality of radiation has on the development of these organisms in shade versus full light and at various elevational differences.

Dr. Lukens: Helminthosporium vagans is one of the fungi studied by Dr. C. M. Leach at Oregon State when he examined the influence of light on sporulating habits of fungi. We have done some work with two fungi. Light both increases and decreases asexual sporulation. The response depends upon the stage of sporulation being irradiated. Light of the near UV range causes hypha to grow conidiophores through inducing the synthesis of a sporogenic substance. Conidiophores, the stalks on which spores are borne are wider and have thicker walls than hypha. Light of visible wave lengths may prevent conidiophores from growing spores and the degree of photo-inhibition depends upon temperature. Alternaria solani sporulates in the dark only at 25°C and in the light or dark at 15°C. Helminthosporium vagans does not require a dark period. It requires a cool temperature to produce a spore. Sporulation decreases with departure from 15°C. So various components of the environment affect fungus sporulation.

I raised the question in the paper of inoculum release from a dry surface. If wind-blown spores are the main vehicle for spread of the fungus, spores on a wet surface will float off and would no longer be capable of becoming air-borne. This raises other questions that may relate to disease. I don't know how much inoculum of turf fungi is wind-borne. If appreciable, a mere manipulation of the environment can effectively control disease.

Mr. Stottlemyer: What do you think this sugar is doing? What is the mechanism?

Dr. Lukens: I suggested one mechanism. It was originally suggested by my colleague, Dr. Patel, who is now with the University of Hawaii. His idea is that these low sugar pathogens require extra cellular enzymes at pinpoint spots were the infection peg penetrates through the cuticle and through the cells. The enzymes may degrade pectin between cells and cellulose in the cell wall. These low-sugar pathogens, in vitro, will not produce pectinase in the presence of glucose or any simple sugar.

Mr. Stottlemyer: If you make a sugar analog, you should be able to inhibit this enzyme.

Dr. Lukens: This is what I was to examine this year. I made two criteria for the search of that carbon source. One, that the sugar had been reported to not serve as a carbon source for some plant pathogen. And two, that it's cheaper than a dollar or so a pound. I came up with about 6 or 7 and I planned to use them on my grass. Unfortunately, the turf was infested with so many weeds that I decided to delay this experiment until next year. Since I measure disease by visual estimates of the percent area brown from foot rot, a thoroughly uniform surface is required to grade disease accurately. Instead of experimenting, I used Scotts Plus-2 to get rid of the weeds.

Mr. Stottlemyer: Did you run any spectra in the shade to determine which wave lengths were coming through?

Dr. Lukens: No, but this can be readily accomplished. I assume it's just light scattering and light absorption of a general nature with little selective removal of a particular wave length.

Dr. Worf: From time to time we make the observation of a severe Helminthosporium leaf spot. On other occasions we see quite a bit of foot rot. Also, in some seasons we have the diseases and other seasons the diseases are almost absent. We have generalized that this is being influenced by the environment to a great extent. But in light of your sugar hypothesis, I wonder if there might also be some changes in sugar levels within the individual tissues of the plant. Particularly, let's say, in the crown area or in the root area at a certain stage of physical development of the plant. It might be influencing what we are seeing.

Dr. Lukens: I was measuring foot rot, and infection of crowns, and analyzing leaf sugar, which I should probably relate to the leaf-spotting stage. But we feel, with this disease, that the permanent damage is due to foot rot, and spores may or may not be the inoculum. We found foot rot infections immediately when the grass greened up in the spring. Leaf spotting, which is indicative of spore production, followed shortly. We believe that the mycelum or the hyphae penetrates directly from the thatch into the crown with little spore involvement. To say which is more prevalent would be merely an academic question, since mycelum and spores are both produced in the sod when the grass starts growing in the spring. I assume that sugars formed in the leaf by photosynthesis migrate to other parts of plants where they are needed.

Dr. Worf: Does the sugar content vary in the crown tissue from one stage of plant development to another? Let's say at the time of the year when flowering and fruiting is taking place. Is there a reduction in the sugar content at that time which would make the crown area more susceptible?

Dr. Lukens: During flowering, the sugar moves from leaves to flowers. Thus, sugar level of leaves and possibly the crowns will go down. All of our analyses were done on leaf tissue. I didn't vary the sugar by flowering. Sugar levels were varied by choice of variety, height of cut, shading and concentration of sugar in the spray.

DISEASES OF WARM SEASON TURFGRASSES

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The grasses and their disease problems in Florida and the southern part of the United States may not differ widely from other areas of the country, but I think they are a little unique in many respects.

Warm-Season Turfgrasses

First of all, the primary grass we grow for the home lawns is St. Augustine (Stenotaphrum secundatum). This is especially true in the peninsula part of Florida and along the coastal region of other southern states, including Texas. This grass has salt and shade tolerance and, hence, is well-adapted to our area. For those of you who are used to bluegrasses and bentgrasses, St. Augustine is rather coarse with leaf blades up to one-half inch in width. Bermudagrass (Cynodon dactylon) is our principal grass for fine turf areas and golf courses, including the tees, fairways and putting surfaces. Another popular grass is centipedegrass (Eremochloa ophiuroides), also a rather coarse grass. It is popular in the northern parts of Florida and along the southern portions of other southern states.

Bahiagrass (Paspalum notatum) is popular also. This is a very coarse, tall-growing grass that was first used as a pasture grass and still is one of the dominant pasture grasses in Florida. It grows well and, due to a deep root system, is drought-tolerant. A few years ago, people thought that this grass did not have any problems. About that time, chinch bugs were wiping out our St. Augustinegrass and many people converted to bahiagrass. Due to its rapid growth, they

soon became slaves to this grass and many ended up with what looked like a pasture for a lawn. Nevertheless, it is still a very popular lawn grass.

Japanese lawn grass (Zoysia spp.) is not extensively grown in Florida. We tried zoysia a few years ago and most people are converting back to other grasses. Zoysia seems to have too many problems in Florida. During the winter when our bermudagrasses go dormant and turn brown, we overseed with annual ryegrass (Lolium multiflorum). We're beginning to use some bents and other grasses, but primarily we use ryegrass for overseeding purposes in the winter.

Turfgrass Diseases

As far as the diseases are concerned, we have seven important fungus diseases that occur on grasses in Florida (Freeman, 1967). Brown patch (Rhizoctonia solani) is probably our most important problem. This disease affects all of our grasses, but St. Augustine and centipede seem to be more affected than some of the others. Dollar spot (Sclerotinia homoeocarpa) is of primary importance on our bermuda, bahia and zoysiagrasses. We are also troubled with the various Helminthosporium diseases. On bermudagrasses, we isolate six such species: cynodontis, stenospilum, rostratum, triseptatum, spiciferum and giganteum. In bahiagrass, we find H. micropus, which we have recently identified as a pathogen on this grass. The primary species affecting ryegrass are H. sativum and H. siccans.

Another important disease, Pythium blight, usually occurs during the winter in Florida on overseeded grasses. This is somewhat unusual because the common pathogen is P. aphanidermatum, which is a warm season Pythium. In fact, some of our work a few years ago established the optimum temperature for disease development somewhere around 95°F. This disease occurs because of a unique situation in Florida during the winter. The weather may be rather cool, but temperatures can go up into the 80's in the southern part of the state during the day. This disease kills the grass very rapidly. We have found that, once inoculum potential is built up, the temperature needs to rise into a conducive range for only a few hours during the day (on several

suscessive days) for the grass to become damaged. Therefore, we can have disease damage from Pythium even during periods that most people in Florida would consider cool. We almost lost the confidence of people in industry when we reported Pythium blight as a warm-weather disease in Florida. This didn't make sense because people were seeing it during the winter months. Incidentally, we see quite a number of diseases during the winter in Florida.

We consider brown patch a winter disease in southern Florida. It occurs predomiantly from about November through March and April. The summer temperatures seem to be too hot for brown patch, and we see very little of it in Florida during the hotter months. Gray leaf spot, or blast disease of St. Augustinegrass (caused by Piricularia grisea) is another disease which is of concern. The causal fungus is a very close relative of the Piricularia that occurs on rice. It attacks many other grasses but is of prime importance only on St. Augustine. During the summer months, practically no plantings of St. Augustine can be found free of this particular disease. It does not kill the grass but it causes plantings to look rather ragged in many instances. The fungus attacks young, vigorously-growing grass more severely than it does more mature grass. Consequently, it is of prime interest to the sod industry since they are concerned with rapid coverage by the grass plants.

We have rust diseases in Florida, although they are not of prime importance. Occasionally they occur in damaging proportions, but not with any degree of regularity. We have rust on bermudagrass (caused by Puccinia cynodontis), St. Augustinegrass (caused by P. stenotaphri), zcysiagrass (caused by P. zoysiae) and ryegrass (caused by P. coronata). On ryegrass, rust usually does not occur until spring. Rust on Zoysia appeared in Florida a couple of years ago and was so severe that the grass appeared orange. It was the most devasting rust outbreak that I had ever seen. However, if you would ask me to find rust today on zoysia in Florida, I would be hard pressed. Since that initial outbreak, the disease has almost disappeared. I don't think it can thrive in our warm temperatures. I wanted to do some temperature relationship studies a year ago, but couldn't get enough spores for inoculation purposes.

Another problem in Florida is nematode diseases. Bermudagrasses, centipede and zoysiagrasses are the three most severely affected by nematodes. Sting and lance nematodes appear to be the most damaging to these grasses. It has become a common practice to use nematicides on golf courses in Florida. Practically all golf courses use them on their greens and tees and many of the richer courses are beginning to use nematicides on their fairways. You can get a very dramatic response from their use.

Nitrogen Studies

We have sandy soils that are very low in nutrients. Nitrogen, especially, is leached from these sandy soils very readily and is lost. Therefore, we find that fertilization with soluble forms of nitrogen is necessary on nearly a two week-cycle in bermudagrass. With less soluble forms, the interval may be a little longer. Nevertheless, most golf courses use from 25-50 lbs of nitrogen per 1,000 sq ft yearly on greens and tees. We have been concerned with the effect of nitrogen fertilization on disease development.

Based on observations of fertility plots, in cooperation with Dr. G. C. Horn, we have found that high rates of soluble nitrogen increase the incidence of brown patch in St. Augustine and centipedegrass. We have seen brown patch kill turf right to the line between high and low level plots. In addition, we found that nitrogen influences the severity of infection by Piricularia on St. Augustinegrass (Freeman, 1964). Adding a high rate of soluble nitrogen dramatically increases the infection of this grass. Conversely, we found that giving bermudagrass a high rate of nitrogen fertilizer retards the development of dollar spot. In fact, this is becoming the accepted method of controlling dollar spot in bermudagrass in Florida. Recently, we have obtained indications that high rates of nitrogen also retard development of Pythium blight.

Piricularia Leaf Spot

We are also studying how nitrogen affects disease severity. We became interested in this aspect a few years ago while studying Piricularia in St. Augustinegrass. I was

studying the influence of rates of nitrogen on disease severity starting out at the zero rate and increasing to 1, 2, 4 and 8 lbs of nitrogen per 1,000 sq ft. Based on leaf-spot counts, there was a significant increase in disease severity with the addition of 1 lb of nitrogen per 1,000 sq ft. Then there was a leveling-off of the disease without a significant increase between 1-2 lbs. However, another significant increase occurred between 2-4 lbs. A slight, but not significant drop occurred between the 4-8 lbs rates. Therefore, we had a stair-step arrangement of increasing disease severity with increasing rates of nitrogen.

Since this disease was very similar to the rice blast disease in which nitrogen content of the plant was correlated with disease severity, we decided that we would check the nitrogen content of the plant. We found the content of total nitrogen followed very closely the curve which we had established for the number of leaf spots - 0.96. With amino acids, the correlation dropped to 0.92, but that is still a high positive correlation. Then we checked the content of individual amino acids and found that the bulk of the increase in amino acids came from aspartic acid, glutamic acid, alanine and the amidesasparagine and glutamine.

Glutamine immediately became of interest to us because it had been implicated in the rick blast disease as being a factor influencing infection. We checked for effects of glutamine and found it stimulates germination of Piricularia spores. We have not been able, however, to change the degree of disease severity using glutamine, either by spraying it on with spores or allowing the plant to take up excessive amounts of the amide. Consequently, we are not sure that we have established the exact reason for increased infection under increased nitrogen fertility. However, our data strongly suggest that nitrogen metabolism of the host affects its disease reaction.

Dollar Spot

Another disease we have been interested in is dollar spot on turfgrasses. Dollar spot is exactly the opposite to

the Piricularia leaf spot. High nitrogen retards the development of this disease. Nitrogen fertilization has a dramatic effect on the incidence of dollar spot in bermudagrass. For all practical purposes, the disease can be controlled with nitrogen fertilizer. In our studies, we used ammonium nitrate at 1 lb of N per 1,000 sq ft. There was a little dollar spot in nitrogen treated plots, but only in the tips of leaf blades and was of no practical concern. I really don't know what the nitrogen is doing; it may be making the grass more resistant, or simply stimulating the grass to outgrow the fungus.

Endo (1966) has postulated that disease reduction is due to the fact that the dollar spot organism requires a nitrogen base to establish a parasitic relationship. There is more senescent tissue in unfertilized grass to provide this nitrogen base. In other words, senescent tissue provides the nitrogen base for the dollar spot organism to attack the grass. With this in mind, we became interested in the effect of nitrogen nutrition on S. homoeocarpa. We checked various rates and sources of nitrogen for their effect on the growth of S. homoeocarpa in liquid cultures. The optimum rate for growth was found to be 0.5 g of N per liter. Once the optimum rate was established, several sources of nitrogen were tested at this rate. It was found that the dollar spot organism can utilize nitrate sources of nitrogen better than the ammonical sources. Casein hydrolysate was the best source tested. Since the nitrogen in casein hydrolysate is derived from a mixture of amino acids, aspartic and glutamic acids, were very good sources for the growth of S. homoeocarpa. The amides of these two acids, glutamine and asparigine, were also very good sources of nitrogen. Leucine and alanine were the best sources from among the neutral amino acid group. Methionine was lowest in this group.

When the basic amino acids were tested, we found that the fungus did not grow in lysine treatment. We didn't know whether it couldn't utilize lysine as a nitrogen source or whether lysine was inhibitory. Therefore, we added sodium nitrate to the lysine treatments under the assumption that, if it had been inability on the part of the fungus to utilize the lysine as a nitrogen source, adding sodium nitrate would

would allow the fungus to grow. It didn't grow, so we assumed that lysine was inhibitory. We later found that casein hydrolysate will overcome the inhibitory effect of lysine on the growth of Sclerotinia. We had wanted to postulate that lysine was instrumental in making the high nitrogen fertilized grass more resistant to disease. But this last finding confused our thinking on this point. We later found that several other amino acids would counteract lysine inhibition, so it is very doubtful that lysine is actually acting to make the grass resistant.

Pythium Blight

We also were interested in the effect of nitrogen on Pythium because we find that increasing nitrogen fertilization of ryegrass reduces disease severity. Disease severity is reduced as the nitrogen rate is increased. Our preliminary tests were carried out in a homogenous soil that may have contained varying degrees of nitrogen. Later, tests were carried out in growth chambers in liquid culture, so we could more accurately control our nitrogen source and inoculation procedures. Results from these tests were similar to those in soil. This nitrogen-disease relationship was first brought to our attention when many of the golf courses who used ammonium nitrate during the winter months had less disease damage than those using organic sources that release the nitrogen slower. In other words, courses on which organic or slow release sources of nitrogen were used during the winter, had more Pythium damage than courses where inorganic or faster release sources were utilized. Our results had given us an insight as to why this was happening. This work is still in the embryonic stage, but we are rather excited about the potential that we have for control of this serious disease by manipulating the nitrogen fertilization program. Again, we don't know how the nitrogen affects a change in disease severity.

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DISCUSSION PERIOD

Dr. Altman: I would just like to clarify one thing with regards to your winter brown patch and our summer brown patch. I think we are all in the same boat since Alaska is part of the country and Colorado is now considered in the South, too. But our summer daytime temperatures are between 80 and 90 degrees and I think you mentioned this as your winter temperatures. Our evening temperatures are 55 degrees, so we are talking about the same temperatures at different times of the year.

Dr. Freeman: You're right about that. We're talking about the same kind of conditions. During the summer our day temperatures often exceed 90 degrees in June, July, August and into September. Your summer conditions, in reality, are very close to our winter conditions.

Mr. Gabert: Is St. Augustine leaf spot called gray leaf spot? Also, is there a control for it?

Dr. Freeman: Yes. We find several fungicides will control it. In fact, most of the turf fungicides will adequately control gray leaf spot. It's a matter of timing the application. The disease occurs during the summer months in Florida when we have rain practically every day. The grass is growing very rapidly and it is important to keep the fungicide on the leaf surface, as a protective measure. Practically all the turf fungicides, such as thiram and mercuries, will give adequate control.

Dr. Harrison: When you were reducing the amount of dollar spot with nitrogen, in what form were you applying the nitrogen? Was that a liquid or a soluble?

Dr. Freeman: It was a soluble source. Ammonium nitrate, 1 lb/1,000 sq ft, in this particular test.

Dr. Endo: Your results with Pythium were very interesting. Do you think that nitrogen is affecting mycelial growth, infection, or subsequent colonization?

Dr. Freeman: I don't know. We first noticed this with ammonium nitrate. I had thought, at first, we were affecting the growth of the parasite. Perhaps we were getting ammonium released that was toxic to it. But we have done this outside the growth chamber. Right now I have a tendency to think it is making the grass more resistant.

Dr. Endo: Have you looked at those plants microscopically? Did they have as much mycelium growing on the surface?

Dr. Freeman: No. In fact this is what we based most of our criteria for disease development on, the size of the disease spots. The actual disease spots are smaller, and there is not as much mycelium growing on the surface.

Dr. Endo: Pythium aphanidermatum is really unique because it's one of the few species of Pythium that emerges from the soil to attack the foliage, even mature tissues. Do you know if the fungus requires a food base in the form of an infected lower leaf before it can attack the rest of the foliage, or can it grow abundantly as mycelium without it?

Dr. Freeman: I think the latter would be right. We get very abundant growth of this fungus over the entire plant tissue. Under ideal disease conditions, it's really an aerial blighting in which the fungus grows profusely over the surface of the plants.

Dr. Endo: That suggests to me that, somehow, turf is providing abundant exogenous nutrients to nourish hyphal growth.

Dr. Freeman: That's correct.

Mr. Simmons: Do you get this same disease during the summer and on warm season grasses?

Dr. Freeman: I should have pointed this out. Bermudagrass is the only one we found that is susceptible to Pythium, and we have tested all of our warm season grasses. It is least severe on ryegrass. Pythium is not as much concern during the summer months because the grass is more resistant, but we get a lot of damage from this disease on our

bermudagrasses when we overseed. We have been wondering about this recently because we are seeing more and more of it. Certainly, with the affinity of the fungus for high temperature, it would be really rough if bermudagrass were as susceptible as overseeded grasses.

Dr. Altman: Have you compared an ammonium sulfate with ammonium nitrate for control effectiveness?

Dr. Freeman: Yes, in the case of Pythium blight we did this and they seemed to work the same way, but we got damage to grass with ammonium sulfate under the test system employed.

Dr. Altman: What kind of damage did you get?

Dr. Freeman: We got physical damage from the toxicity of the ammonium sulfate.

Dr. Altman: What was the pH of the soil in the outside field plots where you were getting some control?

Dr. Freeman: In most cases the pH was 6 to 6.5.

Dr. Altman: Did you compare ammonium nitrate and ammonium sulfate?

Dr. Freeman: I have not for dollar spot, but Dr. Horn has made studies on this comparison. He found that it works quite well and, in fact, he thinks the sulfate has something to do with the control of the dollar spot, as well as the nitrogen. In other words, he can get an increase in disease control using ammonium sulfate.

Dr. Altman: We made a comparison and found where Helminthosporium occurs in the leaf spot phase, there was less disease with the ammonium sulfate fertilizer than ammonium nitrate.

Dr. Freeman: This is what we find with dollar spot, too. I don't know about Helminthosporium. We are just beginning a project on the effect of nitrogen on Helminthosporium disease in bermudagrass.

TURFGRASS DISEASES IN WESTERN CANADA

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At Lethbridge, Alberta, we are doing considerable research on turfgrasses, principally on species suitable for putting and bowling greens. The construction of new golf courses and the conversion of greens from sand to grass on established courses in Canada have greatly increased the culturing of grass species that are highly susceptible to winter injury. Consequently, there is a greater need for effective methods of grass culture designed to produce greens of good quality in the early spring. I have been testing varieties and techniques for winter protection of turfgrass for the past ten years.

Turfgrass failures are often traced to the wrong selection of grass strains for the location. Monocultures of grass in golf greens scarcely exist in the Canadian prairies. Most greens have been initiated with the Colonial type of bentgrass. These soon undergo succession to annual bluegrass and various wild strains of creeping bent. All greens in western Canada eventually contain mixtures of these types with the predominant species being annual bluegrass. The more northerly the area, with severe winter conditions, the more rapidly this succession occurs. Most golf course superintendents accept this succession as inevitable. I am still of the opinion that with proper management, a monoculture of a suitable grass can be maintained in golf greens in western Canada.

Winter Survival

Winter-killing of turfgrass seldom results from sub-zero temperatures alone; it is also caused by desiccation or attack by low-temperature fungi. These psychrophilic pathogens cause a disease commonly called snow mold. One of the most serious effects of winter-killing is the invasion of the killed-out areas by weeds and undesirable grasses.

Canadians swarm to golf courses and other recreational grounds after the long winter and sports turf should be ready for use. The most important practices in golf and bowling green culture are those that will bring grass through the winter in a healthy condition. Poor greens discourage golfing and many clubs charge only half the regular fee until the greens are suitable for play. If grass is severely damaged during the winter, it is often July in the Canadian prairies before the turf recovers sufficiently to be suitable for play. Often the grass is damaged so badly that seeding or sodding is required to reestablish the turf.

Reaction of Species

Grasses suitable for golf and bowling greens are generally more susceptible to damage from low temperatures and snow mold than grasses used for lawns or recreational grounds. Grasses that are resistant to cold injury and desiccation are also resistant to snow mold. None of the grasses suitable for bowling and golf greens survives the severe winters of the Canadian prairies uninjured, although some strains are more resistant than others to winter damage. Creeping bentgrass (Agrostis palustris Huds.), for example, is more resistant to cold injury than annual bluegrass (Poa annua L.).

Grasses suitable for golf greens on the Canadian prairies are chosen not only for winter hardiness, but also for player acceptance and ease of management. Henderson creeping bentgrass, for example, is winter hardy but does not meet the requirements of player acceptance or easy management. Results from experimental plots should not be considered as final, subject to performance under playing conditions. Several strains of creeping bentgrass are

being tested in Alberta for golf-green culture. The most promising strains are: Northland, Waukanda, Toronto, Penncross, Congressional and Cohansey.

Penncross and Northland have been tested in Alberta on modern golf courses. To date, Northland has met all requirements better than Penncross. Northland has shown good resistance to cold injury and snow mold, and persisted as a pure stand for five years in greens at the Willow Park Golf Club at Calgary, Alberta. The greens at Willow Park are under close observation for winter survival, resistance to invasion by annual bluegrass and player acceptance.

Northland does not solve all problems involved in culture of greens for the prairie region. If managed properly, however, it is recommended for our northern climate. On the other hand, it is not suitable for warmer regions with humid summers. Northland is vigorous and must be thinned periodically, but this is not a difficult task with modern thinning equipment. It is easier to thin grass than to grow it, particularly when spring temperatures are below optimum for growth.

Turf Protection

Various methods for protecting turfgrass during the winter are in use in Canada and the United States. Grass is protected from snow mold by the application of inorganic mercury compounds once or twice in the autumn. Greens are protected from desiccation and cold injury by covering with manure, peat moss, brush, polyethylene or fiber glass.

Watson et al. (1960) made a major contribution to the protection of turf in winter with their work on polyethylene covers for turfgrass during the winter. Their techniques are being widely used in the northern United States and in Canada to prevent winter-killing on golf greens. The cover prevents extreme drops in soil temperature and lessens cold injury and damage caused by snow mold and desiccation. In southern Alberta, polyethylene covers increased the effectiveness of inorganic mercurial fungicides for control of snow mold. Less than half the recommended

rate was adequate to control the disease when the turf was covered with polyethylene sheets immediately after the treatment. The use of covers for protection, however, has several disadvantages. These are (a) the plastic covers are difficult to fasten securely to the ground in windy regions; (b) the grass is not always protected; (c) the cover cannot be removed safely in the spring until threats of damage by freezing are over; (d) grass often grows rapidly under the cover but cannot be clipped; (e) considerable labor and expense are required to supply and maintain covers each year, and (f) turfgrass is not usable during the long period of cover.

Treatment of turf with fungicides and the use of various types of soil-insulating materials have reduced winter-killing. These techniques have many disadvantages and do not always produce the desired results. Soil warming below the surface appears to be a solution to this problem.

Results from experiments conducted at Lethbridge, Alberta, since 1960, indicate that turf heating with electrical cables can ensure winter survival of non-hardy turfgrass. Snow mold and other causes of winter-killing were controlled by raising the temperature of the soil a few degrees during cold periods. Minimum temperatures at a one-inch depth in turf plots were maintained by thermostatically controlled soil-heating cables (LeBeau, 1964). Results also indicate that consumption of electrical energy required to bring turfgrass through the winter in a healthy condition is in the economic range. Turfgrass held at minimum temperatures of 3° and 6°C. was severely damaged by excessive heat and electrical consumption was uneconomical, whereas turfgrass held at minimum temperatures of 0° and -3°C. survived the winter in good condition and electrical consumption was economical.

The temperature in the unheated plots dropped to -11°C. in December. In only two periods of the winter was electrical power required to maintain minimum temperatures above 13°C. These periods occurred when low air temperature coincided with limited snow cover.

The first practical test of turf heating in Canada was initiated in 1966 when electrical cables were installed under

a putting green at the Banff Springs Golf Course. The project is a joint venture of the Canada Agriculture Research Station at Lethbridge, the Canadian Pacific Railway Company, the Calgary Power Company and the Canadian General Electric Company.

Soil warming is still in the experimental stage in Canada. We have neither promoted nor condemmed it as a practical method for turf culture because the answers are not complete. The results from our experiments at Banff indicate that the cost of installation and operation of electrical heating systems for golf greens appears to be within the economical range for high-budget golf courses.

Increased Growth Response (I.G.R.)

Partial sterilization of soil has increased the production of alfalfa, winter cereals and sugar beets (Altman and King, 1965; Buchenaw, 1963; Webster et al., 1967). Recently, at Lethbridge, we have demonstrated I.G.R. in turfgrass. In addition to controlling snow mold with mercuric chloride, the partial sterilization with this chemical has stimulated the growth of the grass in early spring. The chemical treatments were made in the fall.

Snow Mold Identification and Control

In central Alberta, snow mold is caused by an unidentified low-temperature Basidiomycete. Dead patches in early spring on golf courses, bowling greens and lawns are often caused by this pathogen; however, similar damage to turfgrass may result from desiccation, trampling and frost injury. A simple method for distinguishing snow mold from these other types of injury seemed desirable. The synthesis of hydrogen cyanide (HCN) by the Basidiomycete has been demonstrated under artificial conditions. The production of this gas by the fungus suggested a quick method for identifying the disease. It was considered that chemical tests that established the presence of HCN in turf samples would differentiate between damage attributable to the snow mold pathogen and that caused by the other factors noted above.

All samples taken in April from areas containing infected plants gave positive tests for HCN, whereas the controls showed no trace of cyanide. Samples taken in early April gave stronger tests for HCN than those removed later in the month, but those taken in May failed to give positive tests. Hydrogen cyanide apparently was not fixed in the plant tissue or soil and was volatilized as soon as the temperatures rose in the spring. Isolation results confirmed the presence of the low-temperature Basidiomycete in all samples containing HCN.

Fungicidal Treatments of Turfgrass for Snow Mold

Inorganic mercury compounds have proven to be effective fungicides against snow mold organisms prevalent in Canada. A mixture of corrosive sublimate ($HgCl_2$) and calomel (Hg_2Cl_2) is generally recommended and is sold under several trade names. Companies merchandise these two chemicals in mixtures on the assumption that the more soluble corrosive sublimate is readily available to control snow mold in the fall, whereas calomel, being sparingly soluble, persists longer and is more effective in the spring.

To test the validity of this hypothesis, an experiment was conducted. Plots of Agrostis tenuis L, 8 ft sq infested with snow mold were treated in the fall. Calomel and corrosive sublimate were applied at the same rate (mercury equivalent) and at half the rate of the mixture (Calo-Clor). The treatments were replicated three times and the plots were rated for survival the following spring. The results provided no evidence to support the view that a combination of the two forms of mercury is more effective than either one used alone.

Tests with other chemicals are also conducted every year. Some organic compounds are showing promise, particularly when two or more applications are made in the fall.

Fairy Rings

Nitrogen fertilizers and fungicides may be beneficial in the control of fairy ring; however, the results of our study

showed that complete soaking of the infected area was the essential factor. Fairy rings are most severe on grass inhabited soils that are dry and low in fertility. This statement is often disputed by home owners and park superintendents who claim they have supplied their lawns with sufficient amounts of fertilizer and water and are still plagued with fairy rings. The disease is prevalent on golf course fairways but seldom occurs on the greens, and the latter are fertilized and watered more heavily than any other turf area.

In recommending soaking as a method of control, it should be stressed how difficult it is to penetrate a well developed fairy ring with water. The soaking should be done by persistent watering each day for at least a month until the ring is soft. The fungus does not develop under these extremely wet conditions and gradually disappears. The reaction may be due to increased bacterial and fungal activity in the soil leading to the destruction of M. oreades or to the hydrophobic property of the pathogen.

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Dr. Altman: You stated that fairy rings can be controlled by soaking the area and applying a high rate of nitrogen. How long does this control last with flooding?

Dr. LeBeau: It will last as long as you maintain a regular feeding and watering program.

Dr. Altman: I've heard of this and tried it but had only a temporary effect, sort of a masking effect. Do you suggest that you can get permanent control?

Dr. LeBeau: I would suggest this. My argument reverts back again to the fact that you don't get fairy rings on well watered and fertilized golf greens. On the other hand, this may be too much water to ask homeowners to put on their lawn. I do know that lack of water and nitrogen are important. Lawns on a slope, where it's difficult to get proper irrigation and runoff occurs are very susceptible to fairy ring. Occasionally you will find a fairy ring on a golf green located in a high, dry area.

Dr. Endo: When you get threads of hyphae growing through the soil, they are quite water repellent, aren't they?

Dr. LeBeau: Yes, very difficult to soak.

Dr. Endo: Do surfactants help?

Dr. LeBeau: Surfactants weren't used, but they probably would help. We just wanted to see if we could get control with persistent watering, and I mean really persistent watering. We didn't even use a probe, but we did use a hand aerifier and punched holes in the ring. Then we watered and soaked it everyday. We kept this up until it got real mushy.

Dr. Endo: Every day for how long?

Dr. LeBeau: Well, I shouldn't say everyday. It probably was every second day. It took about a month before we finished.

Dr. Endo: How about IGR? Do you have any idea what caused it?

Dr. LeBeau: I don't know. It may be a release of nutrients, but it's more than that. On the sick alfalfa problem they are finding out they are getting stimulation and nodulation on the alfalfa after soil sterilization.

Dr. Endo: Do you know if the roots of your plants that have shown this IGR have mycorrhiza?

Dr. LeBeau: I don't know. I haven't done any research on this phenomenon. I am saying that it occurs on turfgrass as well as on alfalfa, winter wheat, sugar beets and other winter cereals.

Dr. Fenwick: You stated that the one-shot application for control of severe snow mold is no longer recommended. What do you recommend?

Dr. LeBeau: It is a little premature to say exactly, but we are working with inorganic and organic mercurials. We are convinced, from our experiments this year, that two or three applications in the fall offer much better control. For example, we're not getting bad results with Panogen. Using one-shot applications of six ounces of Panogen per thousand, we didn't get good control, perhaps 50-60% control. But if we put three applications at three ounces, and Panogen is a relatively cheap fungicide compared with inorganic mercurials, we get much better control. With our inorganic mercurials, either mercuric or mercurous chloride, we find that we need at least two applications. For the exact timing, we want to get some trials out in various areas of western Canada. Say we were going to put treatments on September 15th, the end of September and again on October 15th. If we are going to do this at eight locations, we need some cooperative trials. I think uniform testing is something that is really needed. Even though a lot of people are treating, they

are just not getting good results. I think the time of application is very important. I realize that extra applications take a little more labor, but if you could get good control with a fungicide at about a tenth of the cost of inorganic mercurials, the savings might apply toward your extra labor costs.

Mr. Gabert: On that unidentified Basidiomycete, were you able to get spores from it or get in it to grow culture?

Dr. LeBeau: I refuse to even think about it. I have wasted a lot of time trying to get that pathogen to sporulate. In fact, we don't know how it over-summers. We have never seen spores and we don't know how the pathogen is disseminated. All we know is that it is ubiquitious throughout the Canadian prairies.

Dr. Worf: Is the Typhula organism associated with any of your snow mold problems?

Dr. LeBeau: Typhula is sporadic and does occur, but it certainly is of very minor importance in our area.

Dr. Worf: You mentioned the HCN content of Marasmius. Do you associate the HCN with a part of a pathogenic relationship there?

Dr. LeBeau: I don't. I would not speculate that HCN is the toxic principle in Marasmius. I know it produces HCN in the soil as well as in the mycelium and in the fruiting structures. However, I have not associated it at all with pathogensis. It may have something to do with that nitrogen stimulation, the green effect you get in the rings.

Dr. Altman: I would like to respond very briefly to your comment about this increased growth response (IGR) and a little bit of the work that I've done. There is a stimulation in release of nitrogenous compounds, including several amino acids and ammonia. In addition, there is a micro-ecological change that occurs in treated soil. There is also a predominance of saprophytic Corynebacterium that contributes to this stimulation. It can be isolated and grown in vitro. I have done this on many occasions.

Dr. Long: Dr. Ferguson, at the University of Manitoba, indicated that, if he used a wood fiber material to cover bent greens, he was able to reduce the activity of the low temperature Basidiomycete. I'm not sure whether this was in combination with a fungicide or not, but he said it was quite effective. Are you familiar with the work there?

Dr. LeBeau: I know Dr. Ferguson quite well, but I don't know of this experiment. I know some of the superintendents are using a seaweed type of product for covering turf and I would certainly think Dr. Ferguson has treated the turf with a fungicide, too. Any covering I have seen does not reduce snow mold infection; it usually stimulates it, unless there is some fungicidal action. A polyethylene cover certainly would increase the efficiency of mercurial fungicides. This covering may reduce the loss of the chemical or raise the soil temperature. I don't know which of these two factors are responsible for increasing the efficiency of the fungicide.

Mr. Simmons: Could you time treatments at the end of the season when the snow is melting, or has melted, and obtain control of snow mold?

Dr. LeBeau: No, but we used to think this could be done. I'd say you can with Fusarium nivale because it comes later. With low temperature Basidiomycete we get maximum HCN production, starting the first part of January, and by the middle of February and early March the damage has been done. I wouldn't say that the plants were all killed then, but they will die with the snow melting.

Mr. Simmons: We seem to notice from some of our work here that good control will occur all the way up to that final snow melt. Then some disease will occur after that time.

Dr. LeBeau: In a lot of cases, this is masked. You know the grass will come through and look pretty nice when the snow first melts. If the snow stays for a long time, damage is still occurring. With this Basidiomylete, however, its too late to control the disease in the spring.

Dr. Lukens: I have one comment about fairy rings. It's true they are rarely seen in putting green turf. However,

several years ago I tried to control fairy rings in Penncross greens on one golf course. It was a new course that was carved out of wooded land on a hilltop in Connecticut. In about four years, fairy rings popped up all over the place--fairways, tees and greens. The superintendent was able to stop the symptoms with a wetting agent and water on the greens for about two years. Later, in spite of those treatments, the grass wilted and the typical fairy rings reappeared. Shortly thereafter, I ran many treatments, some where the fungicide in water was injected under pressure beneath the sod with a tree-feeder needle; other treatments were drenched on the turf. Nothing we did seemed to stop the grass from wilting out. The problem is still there.

Dr. LeBeau: This is very unusual. We've had them all over the fairways on our courses, but very rarely on the greens.

Dr. Endo: Dr. Lukens, were you referring to Marasmius, or Lepiota? Does this treatment work for Lepiota, also?

Dr. Lukens: We were concerned with Marasmius. I don't know about Lepiota.

ST. AUGUSTINE DECLINE (SAD) -- A NEW LAWNGRASS DISEASE

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Lawn and turfgrass production is a major enterprise in Texas. These grasses represent a permanent investment in better living, the value of which cannot be calculated in dollars alone. Cost of turf and lawn maintenance in Texas annually is estimated at $211 million (Holt et al., 1964). It is also estimated that 41% of the home lawns in Texas are in bermuda, 56% in St. Augustine and 3% in other grasses. Along the Gulf Coast of Texas, 96% of the lawns are in St. Augustine.

The first account of a virus disease of St. Augustinegrass (Stenotaphrum secundatum) was reported by Todd (1964). He described the disease as being transmitted by mechanical means from St. Augustinegrass to sugarcane and sorghum, and back to St. Augustinegrass. Todd also determined the mosaic virus strain infecting St. Augustinegrass to be an undescribed strain similar to that which occurs in sugarcane variety F 31-407. The effect of the disease on St. Augustine was not known at that time.

Abbott and Tippett (1964) reported that St. Augustinegrass grown from stolons and inoculated with an airbrush became infected with strains of A, B, D and H of sugarcane mosaic virus (SCMV). The virus was transmitted from St. Augustinegrass back to sugarcane. A natural infection of the mosaic disease had been observed on St. Augustine in Louisiana.

Dale (1966) reported St. Augustinegrass seedlings mechanically inoculated with maize dwarf mosiac virus (MDMV) and SCMV resulted in 80% and 76% infection, respectively.

MDMV has been shown to be similar in many respects to SCMV and the viruses have been shown to be serologically related (Williams and Alexander, 1965). Although the two viruses are similar, they do differ in one respect. MDMV will readily infect Johnsongrass, both in nature and experimentally. SCMV has not been found on this plant in nature, and in only one instance has infection from mechanical inoculation been reported (Abbott and Tippett, 1964).

In Texas, the new mosaic disease of St. Augustinegrass was first observed in the Lower Rio Grande Valley in 1966 (Toler et al., 1969). By May of 1969 the disease had been observed in 13 counties in Texas (Fig. 1).

Grain sorghum (Sorghum vulgare) is a host for both MDMV (Abbott and Tippett, 1964; Kuhn and Kozelnicky, 1968; Sehgal, 1966; Shepherd, 1965) and SCMV (Dale, 1966; Summers et al., 1948; Todd, 1964), but has not been demonstrated to be a host for the agent causing decline in St. Augustinegrass.

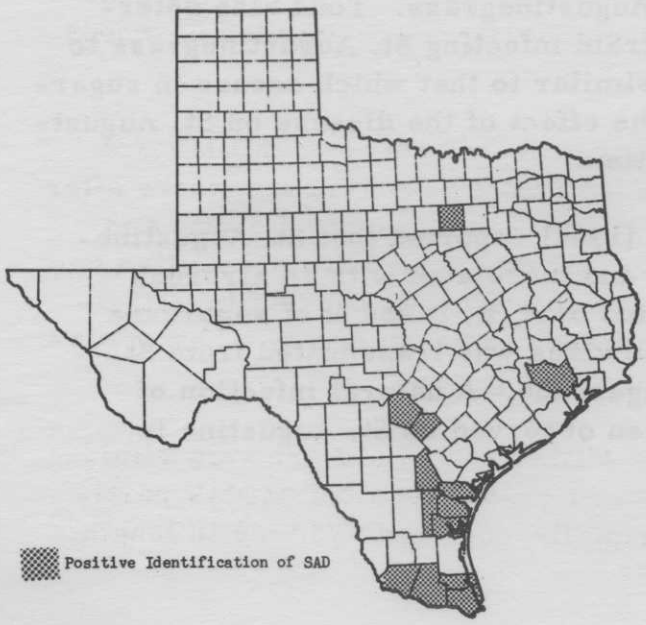

Fig. 1

County map showing distribution of St. Augustine Decline (SAD) in Texas during 1969.

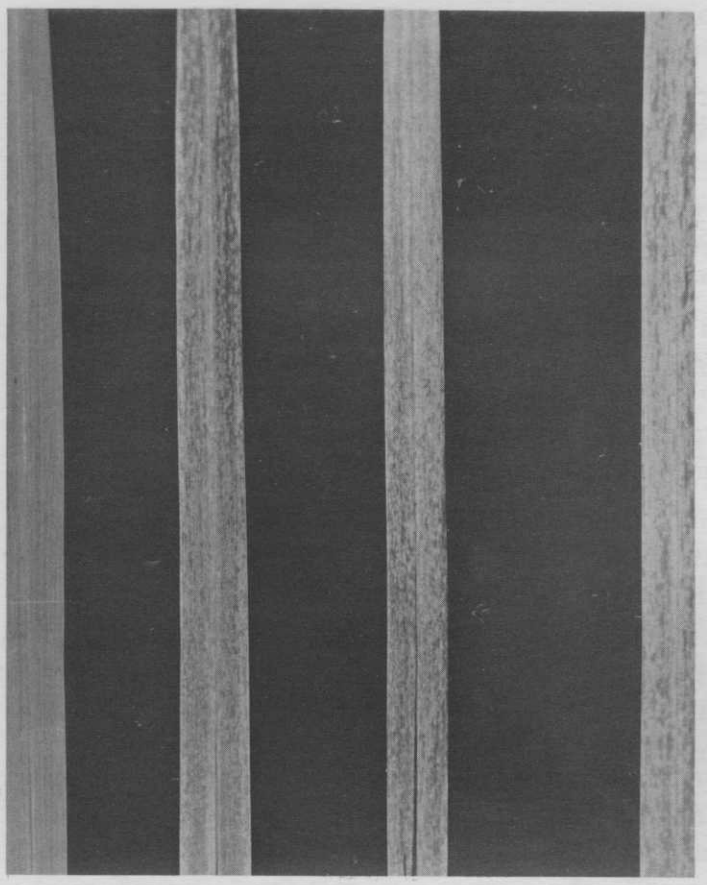

Fig. 2

Symptoms of St. Augustine Decline (SAD) on St. Augustine (Stenotaphrum recundatum). Healthy leaf on the left; three infected leaves on the right.

The symptom of the new disease in St. Augustine is a chlorotic mottling (Fig 2). The mottling becomes more severe until the leaves are yellow. Stolon growth is retarded and shortened internodes may be observed. In advanced stages, necrosis of leaves and stolons occurs causing dead patches in the turf. These areas are soon invaded by weeds and other grasses.

Symptoms of St. Augustine infected with MDMV are similar to those infected with SCMV. Within two weeks after inoculation with MDMV or SCMV, elongated chlorotic and green islands develop in contrast to the chlorotic nottled appearance for the mosaic condition under current investigation. The SAD disease appears to be caused by a new virus or a mutated strain of an existing virus.

The morphology of MDMV and SCMV are very similar. Williams and Alexander (1965) reported MDMV particles to be flexuous or semi-flexuous rods 750 mu in length.

Frazier et al. (1965) demonstrated SCMV particles to be long, sinuous particles with a mean length of 743 mu. Other researchers have found the mean length to be 755 mu (Pirone and Anzalone, 1966).

Maize dwarf mosaic virus has a non-persistent or stylet-borne relationship with its aphid vectors (Messieha, 1967). The corn leaf aphid, Rhopalosiphum maidis (Fitch), has been found to be one of the principal vectors of MDMV (Toler et al., 1967).

Physical property studies by Shephard (1965) have shown the virus to be thermally inactivated in 10 minutes at 55°C, but not at 50°C. Infectivity was retained at dilutions of 10^{-2}, but not at 10^{-3}. Extracted sap held at room temperature was infectious after one day but not after two days. MDMV has properties similar to those of SCMV (Ross, 1964). The host range of MDMV has been found to be similar to that of SCMV. Shephard (1965) reports that host range for both viruses is limited to the Gramineae.

Losses

Figure 1 shows the distribution of SAD in Texas. In Nueces County, it is estimated that 85% of the lawns are infected. Severity of the disease in infected lawns ranged from trace amounts to as high as 100%. In Corpus Christi alone, the loss to homeowners is estimated at $18 million.

Symptoms in St. Augustinegrass

Symptoms of this disease can be used as the diagnostic characteristic in identifying the problem. The first symptom is a mottled appearance of the infected leaves that could be described as spotted or stippled. The mottled condition appears as small chlorotic spots in a green leaf. As symptoms progress, these spots coalesce and larger mottled spots occur. The entire leaf may eventually be overcome with chlorosis. This disease should not be confused with iron chlorosis. With iron chlorosis, a yellow streaking runs parallel with the leaf veins.

During the second year of infection, the grass continues to become chlorotic and weakened to the extent that weeds and grasses begin to invade the lawn. Very slow growth is noted and numerous chlorotic leaves are observed. Any new growth is slightly chlorotic and mottled. During the third year after infection, the grass thins out and begins to die in large patches. The leaves generally begin to die first, then the stolons.

Host Range

Most of the common varieties of St. Augustine tested by manual inoculation have been found susceptible to SAD. SAD failed to reproduce in common bermuda grass, Johnsongrass, wheat, oats, barley, rice, grain sorghum, Kleingrass or corn. However, three millets were found to be susceptible: (1) German foxtail; (2) pearl, and (3) proso. Proso millet is the best indicator and is a diagnostically-useful tool. Symptoms appear within 6 days after inoculation and the disease progresses in a lethal manner causing death of the plants in 14 days. Proso is, therefore, an excellent indicator host. Diagnostically this cuts identification time from 30 days, the normal time necessary for St. Augustine to show SAD symptoms, to only 6 days from inoculation to symptom expression in proso millet.

Transmission

Mechanical transmission with carborundum and buffer rubbing is fairly successful. The artist airbrush transmission has not been too effective at 100 psi and additional work at varing pressures, flow rates and times are under study. Preliminary studies indicated that lawn mower transmission was evident at Edinburg, Texas, three months after inter-planting healthy grass. Soil transmission studies have all been negative. Insects investigated as vectors without evidence of transmission are chinch bugs and leafhoppers, while still under study as possible vectors are mites and walking scale.

Pathogen

Transmission, host range, symptomology, severity, purification and isolations tentatively indicate the pathogen

is a mechanically transmissible virus similar to the rod-shaped sugarcane group. This information will be ultimately published upon completion of the studies currently underway on morphology, serology and biochemistry of the infectious particles that have been isolated.

Control

Studies are underway to evaluate various strains of St. Augustine for tolerance to the SAD disease. Surveys are currently being made to collect clones that have survived in areas heavily infected with SAD. In addition, genetic sources are also being screened from the St. Augustine world collection maintained by Dr. G. C. Horn at the University of Florida, Gainesville. Promising clones have been found to be resistant to infection from mechanical inoculation. This material is being tested for field resistance and will serve as the basis for the new breeding program currently being initiated by Dr. George McBee at College Station, Texas.

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DISCUSSION PERIOD

Dr. Weihing: What are the characteristics of the particle?

Dr. Toler: Some observations have been made and we found a rod-shaped particle. We have also gone through some purification procedures and found a straight to slightly flexuous rod. It seems to be fairly long, around 700 millimicrons plus. This material is being further purified, and our serology is under way at present. Also, we're trying to build up a titer.

Dr. Weihing: Do you feel that the primary transmission of the virus has been mechanical throughout the area, or is there a biological factor?

Dr. Toler: Most of the disease that we encountered at first was in older lawns, which indicated that probably some type of vector was involved as well as mechanical transmission. Of course, there is always the possibility of vegetative spread from the nurseries since we have not surveyed the nurseries nor examined any of them. We know that the virus can be spread quite readily through the stolons and that it is translocated rapidly after infection. Planting material is a source for spreading it to new areas. We feel that mowers have been a contributing factor, particularly the rotary mower and the use of custom mowing in areas that are heavily infected. We have worked very closely with some of the lawn-care services. We feel that the rotary mowers that mow several lawns per day have contributed to this rapid spread. However, we still feel that there is a biological agent, probably an insect of some type, contributing to some of the spread that we can't account for by mowers or by planting infected material.

Dr. Wittenbrook: Do you know of any chemical control, however slight, that has been tried on this?

Dr. Toler: Nothing, other than the nutritional amendments.

Dr. Wittenbrook: In a lawn that has been killed by this virus, can you come back and clean it off, replant and get a normal lawn that is not infected?

Dr. Toler: We have some areas in which we have removed the old thatch, fumigated with methylbromide and sprigged in healthy material that was grown from seedlings that we had multiplied in isolation. At this time it is still healthy. But this is an isolated area near San Antonio and it is completely surrounded by cactus and mesquite. I can't guarantee that we may not pick up some transmission later on. This is one reason we are checking it for biological agents. We are looking for mites and various insects, mostly soil inhabitors and vegetative feeders, to see if we pick up any biological agent that could transmit in the area.

Mr. Simmons: Do you see any reason why this virus won't continue to move north?

Dr. Toler: It's moved quite rapidly in the last couple of years and I see no reason why it cannot continue to move into new areas where St. Augustine is grown. The use of disease-free planting stock and disinfesting mowers may help cut down immediate spread in some areas and your long distant spread by man. I don't anticipate that it will suddenly stop spreading. However, I mentioned we have not, to date, identified a vector.

Dr. Harrison: Have you had the chance to check on the possibility of nematodes as vectors?

Dr. Toler: We have. Dr. Thames and Mr. McCoy at Texas A & M have checked some three or four species of nematodes as possible vectors and isolated them in pure cultures. We fed them on the material and moved them into healthy plants. As yet, we have not positively transmitted the virus under these conditions. This does not rule out nematodes as possible vectors.

Dr. Harrison: Do you know what species of nematodes were used?

Dr. Toler: Yes.

Dr. Holcomb: You haven't said much about regulation. Are your regulatory people aware of this, or have they been made aware of this in relation to interstate shipment of turf?

Dr. Toler: They are aware of it, but since this is a different area from research, I leave it up to the extension and regulatory people.

Dr. Worf: This is quite timely because I was going to ask the same question. You've indicated some of the problems that would be involved with trying to minimize the spread through individual action, but is the area of distribution confined to Texas at present and is there a possibility, theoretically, of controlling SAD through quarantine restrictions?

Dr. Toler: From a theoretical standpoint, yes, if the quarantine could be made 100% effective. Even though it is not absolute, I would say that it probably would help in slowing the spread, if you could keep the plant materials completely restricted and prevent homeowners from shipping samples at random. To date, no other state has reported the disease.

Dr. Scott: You mentioned proso millet and foxtail as hosts. Have you found any natural infections in these species?

Dr. Toler: No, we haven't found any natural infections. The millets are grown in the western part of the state and the Lubbock area in the South Plains. The seed production blocks are in this area. We have not found SAD in our survey of that particular area. However, they are sometimes infected with maize dwarf mosaic virus.

Dr. Carlstrom: What about seed transmission?

Dr. Toler: We have been unable to obtain sufficient seed from infected material to get an evaluation on seed transmission of the virus.

Dr. Elliott: Would you like to predict whether this is going to be a maize dwarf mosaic or a sugar cane mosaic related virus?

Dr. Toler: This may be a little difficult. From host range and some of our physical and chemical property studies, it appears to be somewhat different. However, the particle size is within the range of SCMV and MDMV. It doesn't appear to be a flexuous rod at this time so it makes it diffi-

cult to speculate until we can make our complete comparisons and collect our serology data. We'll have to rely more on serology.

Dr. Lloyd: Does maize dwarf mosaic virus or sugar cane mosaic virus infect St. Augustinegrass?

Dr. Toler: Yes, they go to the clones that are also susceptible to SAD. During some phases of the syndrome, they produce a slightly different symptom. The maize dwarf and sugar cane mosaic symptoms appear to be somewhat more broken interveinal lines. In other words, you get more of a broken pattern of lines in green areas that I like to call "green islands." Also, they tend to be more confined to interveinal areas and are somewhat more broken and chlorotic in the green areas than grass infected with SAD. SAD tends to show more yellow and more of a block-type of mosaic pattern than we usually find with maize dwarf or sugar cane mosaic in St. Augustine. However, the symptoms alone are not always sufficient to tell them apart. But there is a difference, if it is observed very closely. Symptom comparison as a clinical tool is not as conclusive as use of millet indicators. We have also tried to infect sugar cane unsuccessfully with SAD, at least with the one variety we have inoculated up to this time. We do plan to inoculate other varieties of sugar cane that are known to be susceptible to the different strains of sugar cane mosaic virus.

Dr. Long: Dr. Partyka, you mentioned that you would like to have someone elaborate on the use of infrared detecting devices for studying disease infestation development. Would you want to comment on that?

Dr. Partyka: There is some discussion because I know many of the folks are concerned with evaluating plots. I think most people are aware of disease detection methods. I'm just wondering whether this could be applied in some method to come up with a uniform reading for losses or estimates of a disease problem in turf. I think we all realize that there are many complicating factors when you work with turf plots. Rather than making leaf spot counts, or so-called eyeball evaluations, it has been mentioned that disease might be assessed on a more detailed basis with infrared photographs put through a scanning device. This would give some idea as to exact color differences rather than relying on the eyeball method of evaluation. Experiments are being made with other techniques. I think a lot of people are familiar with remote sensing devices. Disease losses are always of concern, but sometimes the question comes up as to whether they're really that important in certain crops. Sometimes, if we knew exactly what was involved, we might be able to get more money for research. Looking ahead to the future, turf and ornamentals are going to be the biggest crops in the country.

Dr. Altman: I have a student who has been doing some work with infrared. Aside from the turf work, I'm also project leader for sugar beet diseases. In sugar beets the infrared has been picking up growth-response differences following soil fumigation in the field. These infrared pictures can be reproduced. You can detect differences in nitrogen, or apparent nitrogen in the leaves. You can also detect differences from airplanes. Grain fields infested with rust are visible because they emit different color bands of light and you can pick them up. I'm wondering if, in addition to disease, you could use infrared as a possibility for evaluating the fertility level on golf courses and parks.

Mr. Brown: Remote sensing has been successful when used in a satellite. The satellite is in a very specific orbit. They take pictures and get the same light intensity everytime it orbits. By the time a plane covers a large area, it may be subjecting itself to different light intensities. I was wondering what instrument could be used that would be portable enough and would not be subjected to these variables?

Dr. Wadsworth: Several years ago we got involved in this work on cereal diseases, primarily leaf rust on wheat. This was an aerial reconnaissance photographing mission that took a very close program of ground support to find out exactly what the reflectance differences were showing. In our particular case, we found some of the variances in reflectance were due to differences in varieties. Without very close ground support, the reflectance differences were not very meaningful in crops that were maturing at different times. Recent information on the satellite program indicated that differences could now be detected on as little as ten acres. Apparently, considerable progress has been made.

Dr. Harrison: Perhaps a little more practical and important to me is the question of rating and reporting of disease incidences and the degree of control obtained. The systems I use differ from those used by others in determining the degree of control obtained with experimental chemicals on Helminthosporium leaf spot, dollar spot and brown patch. I wonder whether the people here think it would be desirable to work toward a standardization for determining the degree of control obtained with experimental chemicals for the more common turf diseases. I would like to get some opinion from the people here and an idea of the different rating or evaluating techniques used.

One of the speakers yesterday mentioned that, in the evaluating process, he judged the degree of disease control. Some workers with Helminthosporium leaf spot, for example, pick 100 blades at random and simply count any grass blade that has spots, regardless of the number of spots on that blade. With stripe smut, I know some workers who are counting the number of tillers with smutted leaves per given area of a plot. With dollar spot, three of my assistants evaluate the

plots by visual inspection and, to avoid bias, they note their observations on records with the treatments omitted. We use a purely objective rating of the plots for brown patch. With other diseases, we do it in a very subjective sort of way. I just wondered how others evaluate disease control and whether or not the people though it would be of any value to discuss the topic now.

Dr. Lukens: I think everybody chooses the means that is most convenient for him. It would be difficult to get us all thinking in the same groove. We do run into problems because one worker reports control with a chemical in one rating system and another reports the same chemical without effect. Primarily, they are talking about two different systems of rating disease control.

For instance, with stripe smut, my results differed from those of Dr. P. M. Halisky in New Jersey. I was working on a lawn that was heavily infested with stripe smut and looking at it from the viewpoint of the homeowner as to what he expected in control and appearance. I counted the number of patches (about the size of silver dollar) blighted with stripe smut. The grass was actually yellow and dying. Dr. Halisky was counting the number of tillers that he found smutted in green turf. Naturally, we drew different conclusions from our results. I didn't have to look at blighted tillers in my system because most were blighted. I was evaluating how much area of turf was discolored by disease.

Turning to Helminthosporium, I compared several methods of recording data with a view to precision and time required to do the job. I counted the leaf spots per blade and analyzed for chlorophyll. Then I counted the number of square inches in a square foot diagram that contained leaf spot and crown rot. Finally, I estimated visually, using the Horsfall-Barret system, the area of turf brown from melting-out. The data all came out about the same when converted to percent control of disease by a particular chemical. The visual estimate was by far the simpliest and quickest way of measuring disease. I base my field data now on the visual estimates of percent area brown. Any greater precision that one may get from some other method isn't worth the extra time it takes to get the data.

I count the number of leaf spots per unit length of blade in the greenhouse and, in certain situations, the size of the spots is recorded. The number of spots is a measure of infectivity of the fungus whereas the size of the spots measures disease development. However, the time required for collecting data is very substantial.

I don't think we can all use one system unless we are rating performance of fungicides on turf. This is where things can be standardized. Some people don't like to use a logarithmic visual system, which is an accurate way of measuring, because what is measured is one's ability to distinguish between differences. You can smell one rotten apple in a whole bushel as well as you can smell 50 rotten apples. Why not utilize this precision of distinguishing the fine trace and have it reflected in the data? With arithmetical grades, say five equal grades between 0-100, one loses precision near the ends. Since values above 80% are of greater interest in disease control, arithmetical grading is a poor method of visual estimation.

Dr. LeBeau: I think this is an important subject worthy of a few comments. It is an aid to all of us that are having to stand up and be counted, and justify our existence. I know this is particularly true in Canada. Currently, we are having to justify most of our research projects. They have set up a special program to try and develop methods of determining how much loss we get from plant diseases in Canada. This, of course, is a very difficult problem. When you are dealing with rust of wheat, it's quite simple. You've got a devastating disease, a chemical method of control, and resistant varieties. Using these, they've made some very accurate estimates and have shown we can lose 100-million bushels of wheat in western Canada from rust. When we get into other areas, such as turf and forage crops, estimates are going to be much more difficult. This is an area that we are going to see some action in the future, especially in turf work in the United States. In Pennsylvania, they claim turf is their number one agricultural industry. I think that we need to estimate the loss from turf diseases in terms of the gross national product. Not only the value of fungicides, but how many man hours does this add to our dollar value in labor? What does this do to stimulate the economy? I

think you can relate the value of turf culture, turf maintenance and installation and it's importance in the national economy. There is room for a survey noting how important it is to control these diseases. The more pressure we can advance for this type of survey - the better we might make our cause look, especially in the area of turfgrass pathology.

Dr. Lukens: A couple of years ago I had the problem of trying to evaluate the dollar value of turf in Connecticut. In addition, I tried to estimate disease loss from Helminthosporium blight, the most important disease of bluegrass. When to employ control measures, replace turf, or do nothing is questionable? Every man has his own castle and he has his own budget for that castle. Wealthy people may act at the first sign of a loss in aesthetic value. Others go along with a disfigured lawn without giving it any concern. How do you measure dollar loss in this situation? Probably, most homeowners would act if deterioration exceeds 75% in their lawns.

Dr. Harrison: I would like to ask a specific question about brown patch, one disease which I have always been very concerned about the way I was rating. To those people who have worked, or who are working with any control materials on brown patch, how do you rate your plots for control for brown patch--according to the various materials used? I used a very simple eyeball technique for getting some sort of evaluation and tried to put it on a 1 to 10 scale. I'm not sure it is the best method to use. I would like to hear if there are any other methods being used so I can, perhaps, modify my own in order to get a better system of evaluating the amount of Rhizoctonia brown patch. In my system, I give my assistants blank copies of the plot plan and ask them to rate the plots weekly as long as the disease is present. Then at the end of 5 or 6 weeks, we try to put the data together on a 1 to 10 scale. Are there any other techniques being used with Rhizoctonia brown patch in particular?

Dr. Altman: I don't think you can get an evaluation of brown patch in two weeks. We put fungicide on 50 or 100 sq ft plots and evaluate them every month for the presence of brown patch. Then, by going through approximately a five-month evaluation with fungicides, we can tell if the disease is pre-

sent. On a five week basis, all you can do is reduce the size of the smoke ring or the size of the brown patch areas. The disease spreads if you restrict your applications. This would indicate that the time period is a little too short to make a fair evaluation.

Dr. Wadsworth: We have carried out a study on the golf course for a number of years, and our two primary diseases are brown patch and dollar spot. In this case, we use a control-type spray program for one year. Materials that look good at the end of this period go into a preventative program on the golf course the following year. Consequently, we get two years of data on these materials. In our evaluation program we measure the number of square feet of affected turf at irregular intervals and put this on a 1,000 sq ft basis, since our plots on the green are not all the same size. This has worked out very well for us.

Dr. Dale: It wouldn't seem to matter what type of system is used if it can be converted to a common factor. For example, in Gould's work I believe he uses the 1 to 5 system in his evaluations. In cooperative corn work in the south, we have all agreed to use a common disease system rating of 1, 3, 5, 7 and 9. If someone does not wish to evaluate this closely, they can use 1, 5 and 9. This can be averaged in with the 1, 3, 5, 7 and 9. As long as people agreed on a system that can intermesh -- 1 to 10, 1 to 5, or something else -- it would not seem to matter what system is used.

Dr. Long: In the fungicide-nematocide tests published each year, different rating procedures are used. It would aid communication if a universal rating procedure could be adopted.

Dr. Altman: I would like to go back to Dr. LeBeau's comment. Recently I made an estimate of the value of turf on the eastern slope of the Rockies in Colorado. I came up with the figure of $350-million, including both the value of turf and maintenance. A banker and several golf course superintendents independently came up with a figure of $325-million. In the sugar beet industry, production is valued at about $35-million, so there is a tenfold difference in value of there two industries. Yet, experiment station support for turf is one-tenth of that for sugar beets. With the turf and ornamental industry expanding,

I think is rather important that we try and come up with a little better selling job in this area.

Dr. Carlstrom: One more small point on this. If we are going to be making evaluations on the value of turf disease control as a selling point, I think we should throw in ornamentals there, too. This would probably comprise 50% of the total disease losses experienced in all crops each year.

Dr. Worf: I believe there is another aspect of reporting results that has not been mentioned. We have been talking about comparison of treatments and material for control and, also, in terms of reporting them. In pursuing the literature, one of the common problems in reports giving a comparison of treatments is that there generally isn't a summary indicating whether any of the treatments are adequate. In other words, we may compare two dosages. One may be bad and the other one very bad. To a person not aquainted with the situation, such a report doesn't indicate if there is anything really encouraging that should be looked at as a possible control.

Dr. Harrison: There were some other points I thought I would like to mention with regard to leaf spot. I know there are several systems being used and I wondered whether anyone felt there were any differences between systems for rating the leaf spot phase. I know some people count the number of leaves with spots, while others actually count the number of spots per leaf. With the same disease, on the other hand, others simply try to estimate the amount of dead turf per plot. I wonder whether or not anyone here has an opinion one way or the other on how that should be done with regards to Helminthosporium leaf spot?

Dr. Worf: Our greatest concern hasn't been with the leaf spot phase, but with the damage that occurs as the disease develops in the crown area. In our own particular situation, we have rather ignored the leaf spot response and placed more emphasis upon the amount of total death that takes place. This is the stage of the disease that the homelawn owner or the golf course superintendent sees and becomes concerned about. I don't know, but I think the question has to do with whether there is a correlation between the amount

of leaf spot that would occur at one time and the amount of crown rot that would develop, let's say, at a later stage in the development of the disease.

Dr. Lukens: We consider the leaf spot stage as a minor nuisance. Turf is lost from crown infections. We do not recommend the removal of clippings as a measure of disease control. However, in Maryland, people are advised to remove clippings in the spring to reduce the severity of this disease. I didn't go along with this advice until I visited this state and saw their bluegrass in the spring. The leaves were plastered with leaf spot. In addition to their unsightly appearance, the infected leaves contributed substantially to the total inoculum. Because of such an over abundance of inoculum for crown rot, it appeared wise to remove leaves under these extreme conditions. Thus, the seriousness of a stage of disease can vary with location.

Dr. Worf: I think your comment begs a question, too, with respect to Helminthosporium. We've talked a lot about that disease but I wonder if, when we are dealing with these Helminthosporium control plots, we should also be taking the time to determine what species is present. I don't want to break the line of thinking with respect to rating systems, but I would be interested in an appraisal of the group here in terms of the relative importance of H. vagans versus H. sativum, or other species in terms of leaf spot, crown rot or even root rot development. Has there been some definitive work indicating that we might have leaf spot coming from one Helminthosporium and crown rot from another Helminthosporium?

Dr. Partyka: Since we're on leaf spot, I wonder if we should take a closer look at the fertilizer rates and nutritional aspects of this problem. It seems that many disease problems are building up since we have gone to higher fertilizer rates. Maybe different types of nitrogen and different times of year, in relationship to application, should be considered a little bit more.

Dr. Lukens: I will try to answer Dr. Worf's question on discerning the several species of Helminthosporium that attack bluegrass. We have isolated H. vagans from leaf and

crown infections during times of epidemics. Isolates taken in summer are difficult to sporulate in the laboratory, but the few spores produced behave as H. vagans. Crown rot from H. vagans and H. sativum have many characteristics in common and both respond to the same control measures. To distinguish between the two may be of little practical value, but may be of academic interest. However, isolates of H. vagans can vary. In my talk I have referred to differences in sporulation with temperature of two isolates from leaf spots of the same patch of diseased turf, moreover, one can change morphological characteristics and physiological performance drastically by manipulating cultural procedures.

On appraising the performance of fungicides, I find that visual estimates of turf diseases are reliable under many circumstances. Because numerous readings can be obtained, one can assess the performance of a treatment by the rate of change in disease during the season. The effect of treatment on the rate of development of diseases yields more information on performance than does data describing the effect of treatment on disease at one time. Also, when considering rate of change, performance is not entirely dependent upon the absolute levels of infection. Thus, one can obtain reliable data on the performance of a treatment in seasons of low levels of disease.

Dr. Elliott: Back to this question about the different species, I expect people looking for resistant bluegrass varieties would be very interested in knowing what fungus species are involved. According to reports, essentially all of the leaf spot in Maryland is caused by H. dictyoides, not by H. sativum or H. vagans. I think you'll find in reports from Minnesota that the species is mainly H. sativum. Since Maryland is neighboring on West Virginia, we thought maybe we had some H. dictyoides on Kentucky bluegrass. We looked quite extensively at bluegrass, not necessarily lawns, but any bluegrass in West Virginia. Ninety-five percent or higher was H. vagans and we never did find H. dictyoides. The only other species we ever find is H. sativum in bluegrass. It is very important that people know what species they are dealing with before they select a material for disease resistance.

Dr. Lloyd: I can appreciate Dr. Elliott's remark about the area around Minnesota because I believe that happens to be North Dakota. H. sativum is the major pathogen in the lawn grasses there. From my own standpoint, I'm interested in the population of H. sativum in this area because it is a predominant pathogen causing kernel black point, leaf blotch and root rot of wheat, barley and durum.

Dr. Freeman: Regarding ratings for disease control, I use a visual rating system. I wanted to make a comment on this Helminthosporium species question. On bermudagrass, we run into several species commonly attacking the grass at the same time. One isolation may be Helminthosporium cynodontis and the next may be Helminthosporium stenospilum. Quite frequently it's a complex of organisms, at least as far as the Helminthosporium species are concerned on bermudagrass.

Dr. Long: Do you use the designation, Helminthosporium?

Dr. Freeman: That's right.

Mr. Bangs: Studies are frequently reported on the control of naturally-occurring fungus activity, regardless of which disease we are talking about. In many instances, no disease occurs during the treatment studies. Why don't researchers inoculate the disease plots so there is some disease in every plot studied?

Dr. Freeman: We inoculate with Pythium in our test plots, and we do it routinely. Pythium is grown on sterilized oats, then mixed up in a bucket and spread in equal portions over each plot. We assume we are starting with equal inoculum in each plot. It has worked out quite well for us. That's the only one we have been able to successfully inoculate with in the field. I don't know why.

Dr. LeBeau: I think there is a great danger in this procedure if you're not careful. If you just throw out the inoculum and then put on a control chemical, you may not be getting natural infection. On the other hand, if you can put your inoculum out and build up a disease nursery, and maybe

use it next year, it would be more effective. I think it is much better if you can get natural infection and have a well replicated experiment rather than using artifical infection.

Dr. Worf: With respect to Helminthosporium, we've had perplexing problems upon occasion. We have felt that, in the absence of disease, the environmental conditions were not appropriate for disease development. Here we would be dealing with an attempt to modify a local environment in order to bring about the onset of disease. In the research we have conducted, we have not felt it was the absence of the inoculum that was limiting disease development.

Dr. Long: Dr. Freeman, do you recall whether they inoculated the centipedegrass in the brown patch plots at Fort Lauderdale? The infestations were uniform there last year.

Dr. Freeman: They did artifically inoculate those plots. How they did it, I don't know. I think this comment about modifying the environment is important. One way they can do it in Fort Lauderdale is to put sand on their St. Augustinegrass, and they will invaribly get brown patch. I tried to inoculate once in the field by covering the plots with burlap for 24 hours, but it was too much trouble.

Dr. Long: Some researchers indicate that you get much more uniform and heavier infestations of gray leaf spot if you have a sprig St. Augustine planting with bare soil areas. Sprinkling or rainfall that splatters the soil on to the grass leaves appears to enhance the spread of this disease.

Dr. Freeman: This is true with gray leaf spot. It appears the disease is much more severe on young, rapidly growing tissue. Therefore, we can get a very good test by using newly sprigged areas. Then we can get some idea of the number of leaf spots and their prevelence. After a few weeks we can evaluate our disease control by the grass coverage of the various plots. Those that we got the best control in will have the most grass; those with the least control will have the least grass.

Dr. Endo: I think this question of why we don't have more disease epidemics is very important since the turfgrass community represents a kind of maximum expression of the three essential conditions required for disease. If one considers all the turfgrass acreage, it's remarkable that we don't have more disease. Why is that? Numerous references refer to the apparent survival of faculatative fungal parasites in infected turf debris. But I'm not convinced that this debris is always important and is always functioning as a source of inoculum. Since the facultative parasites must complete with the saprophytic micro-organisms in the litter, an understanding of the factors that determine the outcome of this competition may be the key to predicting the development of turfgrass diseases caused by facultative fungal parasites.

It's very strange that we can not predict the development of disease caused by facultative fungal parasites, only diseases caused by obligate parasites. Why is this? I think it is because the obligate parasite has essentially evolved a life cycle that has removed it from competition. Obligate parasites either produce their spores above the surface of the host, or within the host where it is isolated from competing microorganisms. When the spore is airborne, it is essentially isolated from competitors. It is well known that spores of the obligate parasite attach and develop best as mycelia in vigorous, actively-growing tissues. This adaptation also serves to remove them from competition since few organisms are present on the surfaces of actively growing leaves.

Dr. Leben has shown that the surfaces of leaves are remarkably free of saprophytes until the leaf has attained its' half-life. After this, there is a very rapid build-up of saprophytes and very weak parasites which would compete with facultative parasites for nutrients during their prepenetration and penetration stages. The lower senescent leaves of turf are undoubtedly colonized by a characteristic group of microorganisms which may inhibit or favor disease development. I consider this question of competition and its outcome as fundamental to understanding the development of turfgrass diseases.

Another important question that Dr. Worf referred to is determining the initial sites of infection. Do Helminthosporium infections start on the leaf, the stem base, or on the leaf sheath?

Is there a correlation between the amount of leaf infection and the amount of foot rot? I think that when the disease starts to develop in the spring in California, the infection frequently takes place on the leaf sheath. The leaf sheath is a very favorable ecological niche because the sheath clasps the stem and the result is a perfect incubation chamber essentially protected from competitors. When successive leaf sheaths are removed, the mycelium of Helminthosporium can be seen growing out of the leaf sheaths and colonizing successive sheaths. Infection of the stem base or crown frequently follows. It has been made very clear that the foot rot stage is the most important. It is imperative, therefore, to separate these two sites of infection and evaluate this relationship.

Dr. Scott: Somewhat in relation to what Dr. Endo was mentioning, during my work with the cereals using Helminthosporium sativum, I found that this organism was an extremely poor root pathogen. Very little infection could be obtained by root inoculations; however the leaf sheath, leaf and crown tissues were highly susceptible to infection by this fungus. In all cases I found that crown infections orginated from leaf sheath infections. Where I did get root infection with Helminthosporium sativum was in the adventitious roots of the crown. The fungus always stopped right at the edge of the crown tissue. It never entered into the crown, but the leaf sheath infections easily spread into the crown tissues.

Dr. Weihing: In Nebraska, we had exactly the same experience in following through with the crown and root rot of Helminthosporium sativum on cereals. The development into the roots never occurred until the plant was under considerable stress. We see the same thing in bluegrass lawns in our area where they undergo tremendous heat and drought stress during the summer. Throughout this decline, caused by environmental stress, there is rapid involvement of the root system and crowns with Helminthosporium.

Dr. Long: Do you think over-saturation of the soil in cool, wet periods could contribute to stress?

Dr. Weihing: That certainly could cause stress.

Dr. Endo: I have been trying to decide what would represent the best stress on turfgrass. Dr. Couch has repeatedly shown that, with three facultative fungal parasites, he produced the most severe disease on turfgrass when plants were under moisture stress. Why should that be? H. L. Russell has reported that desiccation causes severe damage to the root cortex and injury to the plasma membrane. Leakage of nutrients would follow that germination of dormant fungal structures and byphal growth. Now if moisture stress is placed on a plant, and both old and young leaves are present, the older leaves would be colonized by saprophytic-microorganisms and probably would represent unfavorable substrates. On the other hand, young, vigorously growing tissues would tend to lack the population of microorganisms that would compete with our facultative fungal parasites. Some symptoms of drought are obvious. The one ingredient necessary for fungal activity is usually supplied--water. Contrary to what is recommended, many golf courses in California are watered daily. I told growers that it was an ideal disease situation but they frequently claimed a low incidence of disease. It may be that light daily watering favors the build-up of the litter, inhabiting saprophytic micro-organisms which compete with our facultative fungal parasites.

Dr. Scott: As I said earlier, we found Helminthosporium sativum a very poor root pathogen. Root infection occurred only under variable soil water conditions in greenhouse studies. Plants were watered to soil saturation point, then allowed to dry to near the wilting point before watering again. Under these conditions, we obtained a small amount of root infection by Helminthosporium sativum. Where soil moisture was maintained at a constant level near field capacity, we did not obtain root infection.

Mr. Simmons: This comment concerns leaf spot. It's been spoken of as a spring disease in most cases. In many of our observations here, we feel that the cool, wet weather condition in the fall favor leaf spot activity. You will find leaf spot quite active at that time and I wonder if this doesn't account for the foot-rot stage that you see in the spring. As a follow-up, we have found that fall applications of certain

fungicides, principally PCNB, will carry over through the winter and provide leaf spot control the following spring.

Dr. Lloyd: In partial agreement with Dr. Scott are the data in F. C. Butter's monograph on "Root and Foot Rot Diseases of Wheat." He states that H. sativum has two soil moisture optima: above 60% moisture holding capacity and below 30% are the most favorable optium for Helminthosporium infection. At least in wheat crown and roots.

GUTTATION FLUID AND ROOT TIP DEGENERATION IN TURFGRASSES

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I won't say much about guttation fluid and its effects on disease development because I have done very little additional work on it. However, I believe there are some very important ecological niches in turf that favor the growth of fungal hyphae over the aerial surfaces of grass plants. Guttation fluid may be one means by which fungi obtain the necessary nutrients and energy necessary for mycelial growth and infection. There are at least six facultative fungal parasites affecting turfgrass that are capable of growing and spreading from plant to plant by means of ectotrophic hyphae and causing infection. I don't know of any other crop where above ground parts are attached by hyphae of more than one or two facultative fungal parasites. The exogenous source of nutrients and/or food base necessary for ectotrophic hyphal growth must come from somewhere--but where?

Moisture and Drought Stress

I think the literature provides us with some clues. Katznelson et al. (1955) have shown that if moisture stress is placed on plant roots, they will exude more amino acids. Russell (1970) has demonstrated that the root cortex of barley collapses after short periods of desiccation. Tukey (1970) has shown that more nutrients are leached from the aerial organs of plants into water droplets if they are injured, and that senescent leaves leak more than immature or mature leaves. Das and Leopold (1964) have indicated that there is an increase in permeability as leaves approach senescence. Others have pointed out that lack of nutrients, water, shading, high temperatures, or any factor that limits growth, initiates senescence.

Drought injury appears to offer the best conditions for disease development in turfgrass because the injured leaves, following the application of water to relieve the water stress, tend to leak the nutrients necessary for the germination of dormant fungal structures in the thatch and soil, and the subsequent growth of hyphae. If the period of drought stress is prolonged, the activity of competing saprophytes may be reduced; especially if soil temperatures were unfavorable for their development (eg 50oF) but still favorable for pathogen development.

Although numerous sources of host nutrients, such as guttation fluid, are available to nourish fungal hyphae, it is obvious that competing saprophytes may utilize them as well. In fact, it is highly probable in most cases that the competing microorganisms use the host nutrients more effectively than the parasites do, thereby inhibiting their development. This is particularly true of guttation fluid occurring on a senescent turfgrass leaf. Leben (1965) has shown that as a bean leaf passes its half-life, its surface is rapidly colonized by various species of bacteria, yeast and fungi. A similar situation probably also occurs in turf and could effectively suppress growth, infection and pathogen colonization. I suspect that if we could examine the disease development curve for any facultative fungal parasite and compare it with its activity curve, we would probably find that disease development frequently depends upon how well the fungal parasites are doing in their competition with competing saprophytic microorganisms.

Root Tip Degeneration

Another topic I would like to consider is root tip degeneration. In California the roots of cool season turfgrasses frequently exhibit a degenerated condition of their root tips. Only the immature cells and tissues manifest this degeneration; the mature tissues apparently are not affected. The condition is more prevalent during the summer and early fall when soil temperatures are high, and less prevalent during the winter and early spring when soil temperatures are cool. No consistent fungal parasite has been isolated from these degenerated root tips and inoculations have not succeeded in reproducing these symptoms, except with the possible exception of Sclerotinia homoeocarpa.

Microscopic examination showed that a degenerated root apex is not only swollen due to hypertrophy of the cells, but is occasionally curved. The size of the cells is greatly enlarged in the regions of elongation and differentiation. Affected cells are devoid of nuclei and cytoplasm. (Endo and Malca, 1965) If the meristematic cells at the root apex become enlarged they lose the capacity to divide and the root ceases to elongate, the root cap completely disappears and the root becomes truncate. As the root cap cells slough off, they are no longer replaced by additional root cap cells. Since the meristem cells no longer divide, a few immature cells may enlarge to form root hairs near the root apex. Following cellular hypertrophy, the cytoplasm and nuclei disappear. This is why I refer to the condition as root tip degeneration.

What I don't know is how much damage root tip degeneration is causing to plants in the field. A few root tips are degenerated prematurely during the winter, but the loss is compensated for by the formation of lateral roots. The root systems of cool season grasses are, however, short and greatly reduced in number during the summer in California. The presence of root tip degeneration further weakens such plants and the high summer soil temperatures suppress the formation and elongation of both new and old roots.

In pursuing the cause of root tip degeneration, I have followed Kerr (1956), who showed that Sclerotinia homoeocarpa killed the root tips of pea seedlings. The seedlings were placed in cellophane bags containing soil, and the soil surrounding the bags were inoculated with the fungus. He concluded that the fungus produced a toxin which killed the root tips, since the mycelia of the fungus did not penetrate the cellophane bag. I verified Kerr's results and found that the fungus infested the leaves of bentgrass seedlings very readily, but not the roots. However, the fungus produced a toxin which killed the root tips.

Since microscopic examination revealed that the Sclerotinia affected root tips manifested symptoms very similar to root tip degeneration, Dr. Malca and I (1965) decided to look further into this problem. We attempted to produce the toxin by growing Sclerotinia homoeocarpa in a synthetic medium containing lactose as a carbon source because it caused the least damage to healthy root tips. After seven days, the culture fil-

trate was found to be toxic to the root tips of bentgrass, but microscopic studies showed that the symptoms caused by the toxic factor in the culture filtrate was different from that caused by Sclerotinia. When we identified the toxic product, it turned out to be D-galactose. The fungus was utilizing the glucose moiety of lactose, and allowing D-galactose to accumulate in the culture filtrate.

Guttation Fluid

We decided to continue this problem because D-galactose killed root tips of bentgrass at a concentration of 30 ppm in 16 hours and because Stenlid (1957) had not only reported D-galactose and D-mannose as toxic to root tips, but also galactosamine and glucosamine. All of these compounds occur as components of cell walls. Galactose is a component of galactans in hemicellulose, mannose is a component of mannans and glucosamine is a component of chitin. This suggested that if the products accumulate for even a brief time in the surface litter following the breakdown of cell walls of plants, insects, and fungi, and are taken up by roots, they might cause root tip degeneration.

An anatomical study of galactose-affected root tips revealed: (1) a hypertrophy of all the cells of the root apex; (2) a degeneration and disappearance of the cytoplasm and nuclei, particularly in the cells in the region of elongation; (3) a separation of cells in the region of the procambium, and (4) the presence of two nuclei in some cells, suggesting an inhibition of cell plate formation (Endo and Malca, 1965). These light microscope effects suggested that D-galactose was inhibiting the synthesis of cell walls. This was also verified in our electron microscope studies of galactose-affected root tips (Endo et al., 1968). These studies revealed that D-galactose inhibited cell plate formation by inhibiting the formation of vesicles by the dictyosomes and by inhibiting the formation of phragmoplast microtubules. We have also noted that the cell walls are thickened and greatly convoluted, suggesting that the cell wall has somehow been weakened by affecting the activity of the cell wall degrading enzymes that are normally present in cell walls.

To date, biochemical studies by Schmitt and Endo (1969) have shown that D-galactose does not inhibit respiration until 16 hours after treatment. This suggests that galactose is not interfering with energy metabolism. We also found, using radio active glucose, that galactose inhibited the incorporation of glucose into pectin, hemicellulose and cellulose, and caused a 20% to 30% increase in cell wall protein.

Summary

In summary, D-galactose appears to cause root tip degeneration by inhibiting the synthesis of cell walls. Whether it is responsible for causing root tip degeneration of cool season grasses in the field has not been determined. The so-called mat and thatch which consists of fresh and decomposing turfgrass debris does, however, offer uniquely favorable conditions for the break-down of cell wall components such as D-galactose and D-mannose, glucosamine, "etc," which may repress cell wall synthesis.

In conclusion, I am interested in learning whether others have observed what I have been calling root tip degeneration.

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Dr. Altman: I have observed root tip degeneration but haven't done much with it. If you recall, Dr. Wilhelm reported on root tip degeneration on strawberries a few years ago. I would like to ask you a question. What's the difference between degeneration and senescence as it occurs in roots? Dr. Wilhelm suggested that this was a natural process with strawberry roots. There was a replacement that permitted the strawberry plant to produce a crop after substantial degeneration had occurred.

Dr. Endo: It is probably not a normal dying of roots because it happens to seedling and young roots, as well as to old roots. Its distribution pattern does not conform to the age of the roots and it's much more prevalent on cool season grasses during the warm months.

Dr. Lukens: The same question occurred to me when you mentioned that this is most prevalent during your cool season, which is also your wet season, is it not?

Dr. Endo: No, it is more prevalent during our warm season. I don't believe root tip degeneration is due to normal aging of the roots. However, I do think that we need to know more about the normal development of roots; how long they live under different conditions, and what effect such factors as clipping, compaction and high soil temperatures have on root longevity.

Dr. Lukens: Several years ago, Dr. E. J. Bredakis at the University of Massachusetts studied the relationship between cutting height and root length of fine leaf fescues and bluegrass. Whether the increase in root development was long rootlets or continuous side branching was not explained. Dr. Bredakis found a direct relationship between cutting height and root length. Below two inches, root development was drastically reduced in most grasses.

Dr. Endo: Is this greenhouse work?

Dr. Lukens: Yes.

Dr. Endo: I think we have to be somewhat cautious about clipping experiments because one can get quite dramatic effects from clipping if the plants are allowed to grow for a period, and then are suddenly clipped. This is a terrific stress on the plant. When I did this, many of the root tips died very rapidly.

Dr. Lukens: Apparently the tops of all treatments were clipped at the same time. I think frequency of cut was examined, too.

Dr. Endo: Did they first allow the plants to become adjusted gradually to the clipping treatments before they imposed a particular clipping schedule?

Dr. Lukens: I think clipping was started in the seedling stage.

Dr. Altman: You referred to toxicity from galactose with root tips. Are you familiar with toxicity as a result of an amino acid. There was some work done in 1941 in which various amino acids were placed on tobacco meristems and frenching was induced. This frenching was attributed to cell wall development elongation as part of the complex involved.

Dr. Endo: Yes, I am familiar with Steinberg's work. He reported that quite a number of amino acids would cause frenching and root tip necrosis. We have found that the root tip is very responsive to adverse environmental effects and chemicals. Root growth may be inhibited rapidly under laboratory conditions.

Dr. Altman: Have you done any work to evaluate the actual level of root development required to maintain what we consider a normal above ground growth?

Dr. Endo: No, but I think that would constitute a good research problem. I have been searching around in my mind as how one could do that. Do you have any suggestions? I have been interested in trying to find a system where I could remove a predetermined amount of root hairs with detergents, but the experiments failed. (See Endo et al. 1969. Agronomy Journal 61:850-854).

Dr. Altman: How serious is this root degeneration, in regards to overall crop production or yeild of a particular plant?

Dr. Endo: I don't know. I think it is mainly a problem in California during the hot, dry summer months because such plants succumb readily to heat and moisture stress.

Dr. Worf: I missed, at the outset, the conditions that brought about your looking at the root system to detect this degeneration.

Dr. Endo: I was interested in evaluating the relation of species of Pythium to root troubles. In attempting to study this, I encountered the problem of root tip degeneration. Also, plants that died from moisture and heat stress in the summer usually had markedly reduced root systems and the root tips were frequently degenerated.

Dr. Worf: Did you perceive any external above ground symptoms then, which initiated your underground inquiry?

Dr. Endo: There were some, but these differences were mostly in reduced vigor, size of plants and number of tillers. There were no obvious pathological lesions on the upper portions of the plant.

Mr. Simmons: I understood that this work was primarily with the cool season grasses. Have you looked at the warm season grasses, and do they do the same thing at this elevated temperature?

Dr. Endo: I haven't looked at the warm season grasses because we have few disease problems on them.

Mr. Stottlemyer: When I first looked at those micrographs it reminded me of boron deficiency. I realize that boron deficiency has never been shown on grasses, however they look alike and have the same symptoms. Once again, when you have boron deficiency, your zones of elongation and meristematic activity disappear. You essentially have differentiation to the root tips. I was wondering whether you have ruled out that possibility.

Dr. Endo: No, I haven't ruled out boron deficiency. It is a possibility, since Dr. Dugger has indicated a relationship between boron and sugar transport.

EXTRACELLULAR RIBONUCLEASE PRODUCTION BY FUNGI

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Ribonuclease or RNase, has been shown to be produced by pathogenic fungi, however, its significance in host-parasite relationships has not been established. In theory, it could play a significant role in the modification and disruption of the host metabolism.

A simplified method of qualitatively assaying RNase produced by culturable fungi was developed by utilizing reagents to precipitate RNA in media. The test medium was made up using 2 grams of yeast RNA, 20 grams of Bacto Peptone, 15 grams of agar and 1 liter of distilled water. The components were dissolved in water and adjusted to pH 5.4, sterilized at 15 pounds for 20 minutes and poured into petri plates. After cooling, the plates were inoculated with the fungi to be tested and incubated 4-6 days. The plates were then flooded with Uranyl reagent (1 gram uranyl acetate in 100 ml. of 10% perchloric acid) to precipitate any unhydrolyzed RNA in the agar. Cleared areas around the colonies of fungi demonstrated the presence of RNase activity (Fig. 1). Acetone and HCl can also be used as reagents for precipitation, however, acetone precipitates other material in addition to RNA. The media must, therefore, be tested before use. HCl gives a somewhat weaker reaction than Uranyl reagent and lasts for only a few hours.

This method was compared with two other methods using liquid cultures. First, turbidometric measurements were made with a Spectronic 20 at 625 mu, on culture filtrates containing RNA, after precipitation with Uranyl reagent.

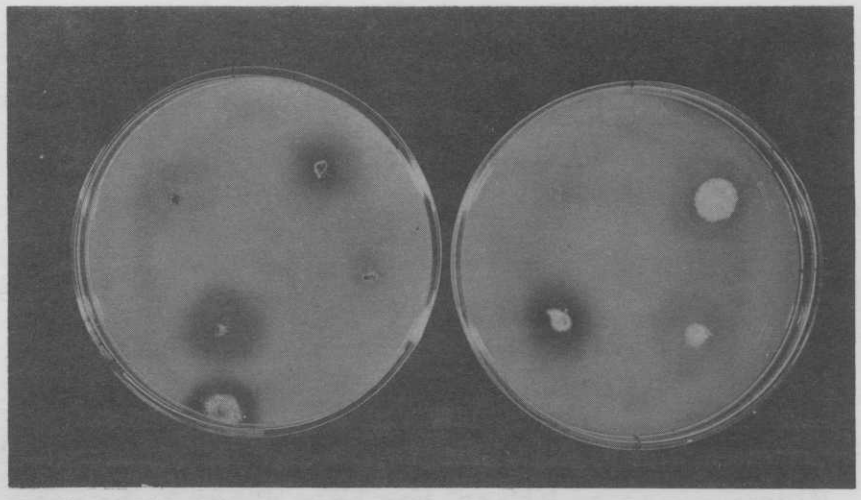

Fig. 1. Fungi on RNA agar after growth and development with Uranyl Reagent. Cleared areas around RNase active fungi are dark due to black background. Top row left to right: Pseudoplea trifolii, Kabatiella caulivora, Drechslera poae, Stemphyllium loti. Lower row: Sclerotinia sp., Stagonospora meliloti, Heterosporium phlei, Drechslera phlei, a positive unidentified contaminant at the bottom of left plate.

The reduction in turbidity was a measure of RNase activity. Second, an increase in optical density due to soluble breakdown products after precipitation and centrifugation was measured with a Beckman DU at 260 mu. Both of these methods were less sensitive and slower, requiring about nine days incubation; however, they were quantitative. The depression of RNase production in liquid may be due to a lack of aeration.

The plate tests offer a quick, simple, but only qualitative assay of culturable micro-organisms for their ability to produce extracellular RNase. Some of the fungi tested and found to produce RNase were: Ascochyta caulicola, A. imperfecta, Alternaria sp., Heterosporium phlei, Kabatiella caulivora and Pseudoplea trifolii. Those not producing RNase were: Bipolaris sorokiniana, Drechslera poae, D. phlei, D. bromi, Fusarium avenaceum, F. oxysporum, F. solani, Sclerotinia trifoliorum, Stagonospora meliloti and Stemphyllium loti.

Dr. Lukens: Can RNase be involved in anyway in pathogenesis?

Dr. Berkenkamp: I would suspect so.

Dr. Lukens: Could you give us any idea as to how you feel it acts?

Dr. Berkenkamp: Since RNA is the mechanism of action from the gene to metabolism, I would say there is a possibility of changing the metabolism in favor of the parasite. I would like to prove this, but I don't know how.

Dr. Lukens: Is there any work that may suggest this?

Dr. Berkenkamp: No, not that I know of. There is quite a bit of work on virus RNase in virus infected plants and some on obligate parasites but, other than production of RNase, nothing on culturable parasites.

Dr. Altman: Could you use your assay technique as a means of evaluating purity in a particular fungus? Also, would you be able to evaluate virulence or loss of virulence by this technique?

Dr. Berkenkamp: I couldn't say. I would expect it to be related to virulence, but not necessarily.

Dr. Altman: Are the precipitates that come out of these white, opaque areas characteristic for a particular fungus?

Dr. Berkenkamp: No, the precipitate is the undegraded RNA. The cleared areas are where RNase has been active in degrading the medium. The result is a fungus colony, a clear area, and the rest of the plate is opaque (precipitated RNA).

Dr. Altman: Does a specific fungus give you a different type of a clear area or are they all similar?

Dr. Berkenkamp: No, usually they're distinct. I compared this with liquid tests and found the plate cultures to be more sensitive than those cultured in tubes. In comparing various cultures, some fungi are positive and others are negative. There are some questionable responses in fungi, but primarily it's yes or no, especially if you use the low pH.

Mr. Gabert: What fungi have you tried with this test?

Dr. Berkenkamp: A large number of legume pathogens, Ascochyta imperfecta, Heterosporium phlei causing timothy eye spot, and some fusaria.

Mr. Gabert: Did you notice any significant differences in the amount produced over a given time between the different species?

Dr. Berkenkamp: The test is not quantitative. If we assume there are isozymes, they would have different diffusion rates. The size of the cleared areas would not be quantitative. I would like to test a large number of fungi and relate the characteristics of this feature to gain some insight. To-date I haven't tested enough fungi.

Dr. Freeman: You brought up a point concerning the Helminthosporium classification. Does any one have any comments on this? I know the people in Canada have been working on changing the name of the genus Helminthosporium but does any one here use those new classifications? Do they still speak of Helminthosporium or of Bipolaris and Drechslera?

Dr. Berkenkamp: Bipolaris is the one that germinates only from the end cells of the conidia, and Drechslera germinates from any cell. I'm not a taxonomist, but I wondered if there was a reason why these names were not acceptable? I would like to know.

Dr. Freeman: Maybe all of us are stuck in our ways. Do you think it is a valid reclassification?

Dr. Berkenkamp: I was asking you.

Dr. Freeman: I'll ask somebody else for their opinion.

Dr. Dale: You were talking about Bipolaris. I'm not a Helminthosporium expert but have done some work with H. spiciferum, somewhat similar to that Dr. Wadsworth did on the spring dead spot disease. In this work, a student studied germination of Bipolaris spores using a range of temperatures. Bipolar germination occurred at some temperatures, but at others, germination was just at one end. Bipolar germination may be a good taxonomic character, however, workers should specify exactly what temperatures are used when studying spore germination. I don't know the answer to Dr. Freeman's question, but believe we need more criteria in classifying Helminthosporium. We may need some "lumpers" instead of "splitters."

Dr. Jackson: Bipolaris and Drechslera are included in Shoemaker's recent classification. He is a Canadian. Are you familiar with him?

Dr. Berkenkamp: Yes, but Drechslera was originally described in Japan by Ito.

Dr. Jackson: There seems to be some difficulty in Shoemaker, Luttrell, Ellis and others agreeing on this group. I think the British Mycology Society has accepted Shoemaker's classification since it is now used in the Review of Applied Mycology. But because the name Helminthosporium is so well known, it is retained for general use.

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