A PROGRESS REPORT

The Nature and Control of Decline and Dying-Out of Toronto C-15 Bentgrass

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IN AUGUST, 1980, a major research effort was launched to identify the cause and develop a control for the decline and dying-out that has been devastating Toronto C-15 bentgrass in the midwestern United States for the past decade. The program is under the joint sponsorship of the United States Golf Association, the Golf Course Superintendents Association of America, and the Chicago District Golf Foundation.

In organizing the program, it was decided to use a research team. This system has the advantage of concentrating a broad base of research expertise on a problem within a short period. As such, then, it offers greater possibility of arriving at a solution at an earlier date than the more classic approach of having a single investigator explore each factor of the problem a unit at a time. The responsibility for carrying out the various parts of the investigation was assumed by Dr. Houston B. Couch, of Virginia Tech; Dr. Philip Larsen, of the Ohio State University; Dr. David Wehner, of the University of Illinois; and Dr. Charles Krause, of the U.S. Department of Agriculture's Research Center at Delaware, Ohio. Dr. Couch serves as the coordinator.

The extent of the research to date has included both field and laboratory experiments. The field research has centered primarily around the development of an integrated control program based on the use of pesticides in conjunction with certain management practices that may in themselves be effective in reducing the severity of the disease. The laboratory-based experiments have concentrated on the identification of the microorganism that is causing the disease and determining what weather and management conditions are most conducive for its development. In these later studies, particular emphasis has been placed on testing the theory that the disease is caused by a bacterium. However, the possible role of other organisms such as nematodes and fungi have also been given consideration in both the field and laboratory tests.

During the initial phase of the research, a field survey was conducted in the Chicago area. Questionnaires were distributed to the respective golf courses, and personal, on-site interviews and observations were made by a graduate research assistant from Virginia Tech trained in turfgrass management and in plant pathology. The purpose of the survey was to provide a thorough history of both the management practices of the affected courses and of golf courses in the immediate vicinity not affected by the disease. Also, during these visits, plant, soil and irrigation water samples were collected for laboratory studies.

THE SOIL SAMPLES were taken from the putting surface and the collars of greens that were showing visible symptoms of C-15 decline and were dying out, and from greens free of the disease. Irrigation water samples were taken from the water supplies of courses affected by the disease, and from nearby courses that have Toronto C-15 bentgrass, but do not have a history of this disease. Also, soil and water samples were taken from the two sod nurseries that have been serving as major suppliers of Toronto C-15 sod to the Chicago area.

Analyses were performed on all of these samples for pH, soluble salts, available nitrogen, phosphorus, calcium, copper, zinc, magnesium, manganese, boron, iron, cadmium and mercury. Also, the soil samples were checked for total organic matter content. With the exception of iron, there were no positive correlations between available levels of these elements in the soil and the presence or absence of C-15 decline and dying-out.

All of the sites sampled that were free of decline and dying-out showed adequate levels of available iron in the soil, while those sites showing symptoms of the disease were low in iron. The analyses of the water samples did not show any correlations between concentration of any of these elements and the presence or absence of the disease.

In comparing locations with the disease and locations without C-15 decline, it was found that there was no relationship among these different sites in either the amount of fertilizer applied or in the manufacturer of the fertilizer. There does appear to be a relationship, however, between both the amount and the frequency of application of iron on these different locations. The Toronto C-15 in the sites showing decline and dying-out received iron at less frequent intervals or in less total amounts than areas where the disease was absent.

There is a relationship between the height of cut and the appearance of C-15 decline and dying-out. Low-cut grass (2/16 inch) is more likely to show the disease than plants at higher cuts. The disease does develop in the collars, but it is generally less severe in these areas than on the putting surface.

There is also a relationship between rainfall, air temperature, moisture-saturated soil, and radiant energy levels and the development of decline and dying-out in Toronto C-15. The disease is most severe following periods of cool air temperatures and continuing rainfall, particularly where soils drain slowly. Also, outbreaks of the disease are usually more severe when these periods of rainfall are followed by two or three bright days.

The soil samples collected on the survey were also assayed for parasitic nematodes. These determinations were performed by Dr. L. T. Lucas, of the North Carolina State University. Eight species of parasitic forms were found. Certain of the populations were high in locations where the disease was present, but they were also high in certain areas where decline and dying-out were not evident. Also, population counts were low in samples taken from areas where the disease was high in severity. On the basis of these tests, then, it has been concluded that the presence of parasitic nematodes is not a necessary requirement in order for decline and dying-out to occur in Toronto C-15.

LABORATORY TESTS for the purpose of identifying the fungi associated with C-15 plants showing symptoms of decline and dying-out are being carried out by Dr. Philip Larsen. To date, the species of Helminthosporium that causes Helminthosporium leaf spot has been isolated from a few of the diseased specimens. There has been no consistency of these isolations with the outbreaks of the disease, however, and the species that causes red leaf spot has not been detected in any of the samples collected from C-15 greens that show symptoms of decline and dying-out. Also, the cool-temperature species of Pythium has not been found in any of the diseased plants. Other Pythium species have been isolated, however, and while it is felt that they are probably not the primary cause of this disease, it is possible that they do contribute to the stress pattern that precedes its appearance. Therefore, experiments designed to determine the capacity of these various isolates to infect and colonize Toronto C-15 plants and an assessment of the damage they are capable of inciting are being carried out by Dr. Larsen.

Histological examinations of the diseased plants have been carried out by Dr. Houston Couch. These studies have revealed that the disease is primarily one of the root system, and possibly in the later stages of development, the lower portions of the crown are involved. The tissue most affected appears to be the xylem (water-conducting elements). However, adjacent tissue becomes diseased also. The foliar symptoms associated with decline and dying-out are probably brought on by the disruption of the root system — specifically, the breakdown of the water-conducting tissue.

Dr. Charles Krause has been conducting studies of diseased C-15 tissue

Figure 1.

Individual Toronto C-15 bentgrass plants affected by the decline and dying-out syndrome first show a tip dieback. At this time, they also become a darker green and become twisted and shriveled. In the final stages of disease development, the leaves turn brown, and the crowns and roots of the plants become brown and decomposed. In these last stages of development, the stand of grass commonly shows large areas of dead plants with intermingled tufts or streaks of green plants (see Figures 1 and 2).

Figure 2.

with the electron microscope. The purpose of this work has been to search for the presence of microorganisms and determine how consistently they are associated with diseased plants. Through this type of study, oftentimes a lead can be obtained on what particular type of causal agents to target for isolation and pathogenicity studies.

In this work, Dr. Krause has found one bacterial type with characteristic cell wall markings in the xylem of diseased plants. The healthy C-15 plants are free of these bacteria. What appears to be this same species of bacterium has also been found in the xylem of diseased C-15 plants by Dr. Karen Baker and Mr. David Roberts, of the Center for Electron Optics, at Michigan State University. While the findings at these two laboratories are not conclusive proof that this particular bacterium is actually the cause of decline and dying-out of Toronto C-15, they do provide good circumstantial evidence in support of the theory that the disease is bacterial in origin.

The field research in the Chicago area has added weight to the theory that a species of bacterium causes C-15 decline. This work is under the supervision of Dr. David Wehner. In these trials, fungicides were tested for control of the disease that are known to be effective in the control of Pythium-incited diseases and red leaf spot. In addition, two bactericides were tested — streptomycin and tetracycline. Also, the possible role of iron in reducing the severity of the disease was studied in these trials.

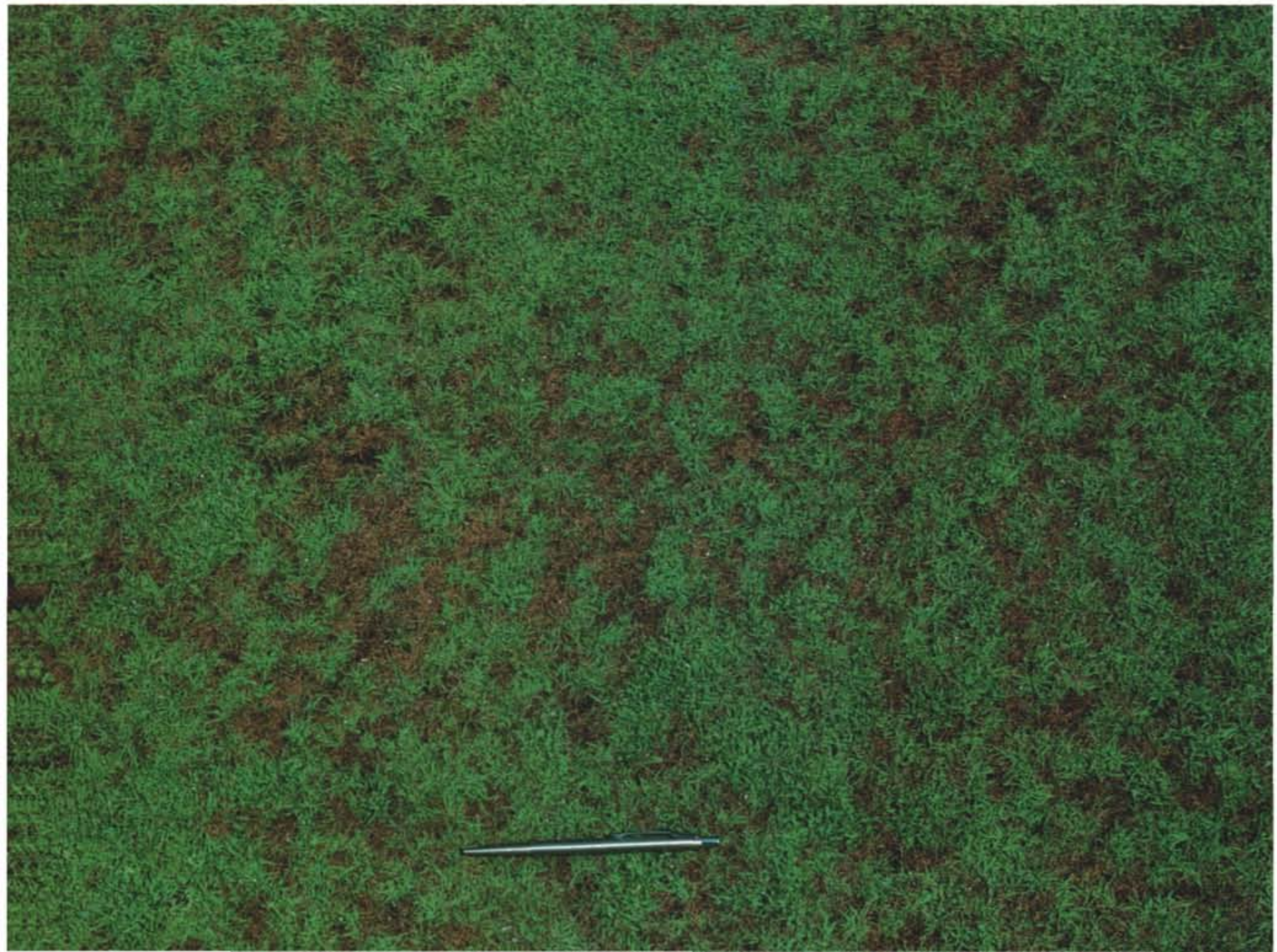
The fungicides did not provide any measure of disease control. These results, coupled with the failure to isolate either the cool-temperature species of Pythium or the red leaf spot pathogen from diseased C-15 tissue has led us to conclude that this disease is not related to either of these problems.

Of the two bactericides tested, the applications of streptomycin did not reduce the severity of the disease. Tetracycline, on the other hand, gave very good control. (Note Figures 3 and 4.) These results, coupled with the electron microscopy studies of Drs. Krause and Baker, lends considerable weight in support of the theory that decline and dying-out of Toronto C-15 is caused by a bacterium. The final proof of this theory rests with pathogenicity tests. These are being conducted by both Dr. Couch and Mr. Roberts.

IN SUMMARY, the research to date has led the research team to conclude that the decline and dying-out of Toronto C-15 bentgrass that was in evidence in the Chicago area in 1980-81 was not red leaf spot, nor was it caused by a cool-temperature species of Pythium. Also, plant parasitic nematodes are not the causal agents of the disease. While it is possible that soil pH and certain management practices may add to the severity of the disease,

In field trials in the Chicago area, effective control of decline and dying-out of Toronto C-15 has been achieved with applications of the bactericide tetracycline. The plot shown in Figure 3 was a non-treated control. The plot treated with tetracycline, shown in Figure 4, was immediately adjacent to the plot in Figure 3.

Figure 3.



they are not in themselves the cause of the malady. Also, there is no relationship between manufacturer, analysis of fertilizer, or types and amounts of pesticides used and the occurrence of decline and dying-out of Toronto C-15. The observed occurrence of large numbers of bacterial cells in the xylem of diseased plants and their absence from healthy plants, and the control of the disease in the field with the bactericide tetracycline, are strong evidence in support of the theory that the disease is caused by a bacterium. The conditions that appear to be involved in outbreaks of the disease are high soil moisture brought about by poor drainage and/or prolonged periods of rainfall, low air temperatures, bright days immediately after the periods of rain, and low cutting heights. Another factor that contributes to the outbreaks of the disease is iron deficiency.

While the research to date has uncovered much about the nature and control of decline and dying-out of Toronto C-15 bentgrass, there still remains much to be done. Proof of pathogenicity of the bacterium found in association with the disease needs to be established. If this organism is indeed the pathogen, then the next step should be a determination of how it spreads from diseased to healthy plants in the field. Also, it will need to be determined whether or not other varieties of creeping bentgrass are susceptible to it, and if they are affected by it, what their respective levels of vulnerability are. Top priority needs to be given also to the development of laboratory procedures for quick and accurate diagnosis of the disease in its early stages of development. Finally, the field research needs to be continued to develop a more complete picture of both rates and timing of pesticide applications for maximum effectiveness in the control of the disease. The technology to achieve these goals is available.

Figure 4.

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