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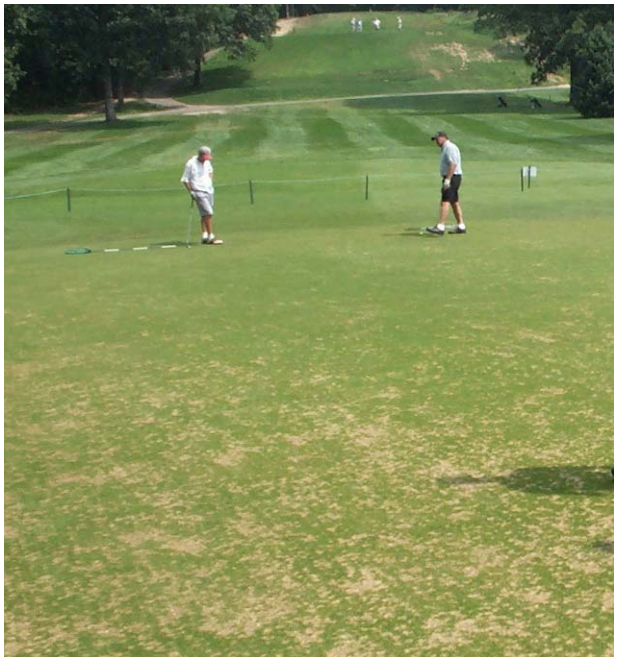

More resources are expended on fungicides to control dollar spot disease, caused by S*clerotinia homoeocarpa* than any other in the USA and most of the fungicide applications are to golf courses. It was the goal of this research at Cornell University to investigate the biology and diversity of *S. homoeocarpa* to discover those variables that are important to the management of dollar spot. *Photo credit: Dr. Jennifer Grant*

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PURPOSE

The purpose of *USGA Turfgrass and Environmental Research Online* is to effectively communicate the results of research projects funded under USGA's Turfgrass and Environmental Research Program to all who can benefit from such knowledge. Since 1983, the USGA has funded more than 225 projects at a cost of $25 million. The private, non-profit research program provides funding opportunities to university faculty interested in working on environmental and turf management problems affecting golf courses. The outstanding playing conditions of today's golf courses are a direct result of ***using science to benefit golf***.

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Diversity and Biology of the Dollar Spot Organism, *Sclerotinia homoeocarpa*, and Its Implications

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SUMMARY

This study on the diversity and biology of the dollar spot organism, *Sclerotinia homoeocarpa*, was undertaken to increase the knowledge base on this organism with a view to improving control and management of the disease. The work indicated that there are substantial lacks in our understanding of this pathogen. Among the findings and hypotheses:

- We were able to isolate the pathogen from still-green, but diseased foliage on the periphery of dollar spot patches, but not from the center of the dead turf. Additionally, diseased turf patches in subsequent years did not occur in the same locations as the previous year. This suggested the concept that disease occurs as a consequence of inoculum arising from overwintering diseased foliage may need revision.
- We artificially inoculated turf seedlings with the pathogen and found that the pathogen preferentially colonized the root-crown region. This is in contrast to the field location of the pathogen, where the pathogen usually is located in the leaves.
- These observations are consistent with the suggestion that the initial disease occurs infrequently and with few symptoms, perhaps in the root-crown portion of the plant. Then, when these individual diseased seedlings are mowed, the disease is spread. The diseased seedling fragments would provide a highly nutritious inoculum source, permitting disease on leaves. If this suggestion is true, then (a) the epiphytotic phase of dollar spot is in reality the second stage of the disease, with the initial infections occurring earlier and (b) if we could control the hypothetical first stage of the disease, then the disease could be more easily controlled with lower levels of pesticides. This hypothesis may also explain why the disease occurs with such explosive speed in the absence of adequate control measures.
- There is significant genetic diversity in the pathogen and some limited evidence suggesting that certain genotypes may be more prevalent in certain regions of North America than others. If so, then this diversity may provide management challenges because different strains may respond differently to inputs.
- The research also provided evidence for the suggestion that populations within particular regions or specific sites may have arisen from small founding populations, perhaps single strains. If so, this suggests the populations of the pathogen probably are being introduced at the time new turf is established. This allows us to question the origin of the pathogen. Presumably, it is being introduced with some materials used to establish the green or other grass surface. If so, is it possible to exclude this inoculum source and thereby control the disease?

Dollar spot disease, caused by *Sclerotinia homoeocarpa*, is intensively managed by fungicide applications and to a lesser extent by cultural practices. Probably more resources are expended on fungicides to control this disease than any other in the USA and most of the fungicide applications are to golf courses.

Pest control frequently is managed efficiently and economically by an extensive knowledge of the biology of the pathogen, including an understanding of its genetic diversity. However, this is not the case with *S. homoeocarpa* in many

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Dollar spot on a bentgrass green indicating the appearance of the dollar spot patches that develop rapidly in the absence of appropriate control measures.

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respects. Some examples of knowledge gaps and the implications of these for dollar spot management follow.

The disease has an unusual cycle of development in that the disease remains quiescent for most of the year and then suddenly develops as an explosive epidemic. In New York, the rapid development of the disease begins in mid- to late July (6) while in Kentucky the disease begins in June (14). There is no documented reason why the observed disease develops explosively, nor is there solid data on where the pathogen is, or its state, when the disease is not evident.

There is an assumption that the pathogen overwinters on diseased turf but solid data on this is lacking. However, in our view, this information is critically important. We will present information suggesting that the initial stages of the disease occurs in widely scattered areas with slight disease symptoms, but that the explosive stage of the disease occurs because tissue from a few infected plants is mechanically spread by mowing or similar processes and that this gives rise to the typical dollar spot epidemic. If this is true, then control of this initial stage might provide economical means to eliminate or minimize the expensive process of repeated application of fungicides. In other words, it may be possible to control the disease while it still is nearly invisible and avoid its epidemic phase.

Further, knowledge of the genetic diversity of the pathogen is quite important. For example, if the organism is quite diverse, then the pathogen in different sites or areas may respond differently to fungicides or management systems. Further, numerous programs are breeding for resistance in turfgrass to the pathogen. There is at least a theoretical risk that breeding programs that develop varieties resistant to a narrow range of isolates may produce varieties that will respond differently (i.e., be less or more resistant to strains of the pathogen in an area from which they were not tested). It was the goal of our research program to investigate the biology and diversity of *S. homoeocarpa* to discover those variables that are important to the management of dollar spot.

One of the most important issues to us was the location of the pathogen when the disease is not epidemic. We also were interested in the first infection foci. The Compendium of Turfgrass Diseases (11) states that "The dollar spot fungus survives unfavorable periods as mycelia in infected plants and as stroma on foliage surfaces." However, most turf pathologists regard this statement as unproven. Further, there is little or no information on the events at the beginning of the disease cycle that precede the explosive development of the pathogen. We set up a series of experiments designed to answer these
questions.

Methods and Materials

Inoculum source added to sites

We expected that we would be able to follow the disease cycle if we added the pathogen at high levels in systems that we could monitor. We separately placed a) dollar spot-infected turf leaves and b) heavy growth of the pathogen on autoclaved wheat seeds in nylon mesh bags (4 x 2 cm, 75 micron openings) that were closed with a heat sealer. In the spring of 1998, we placed the bags with inoculum in three different soils/conditions: a sand research green and a soil research green, both at the Cornell University Turfgrass Research Facility, and a fairway at the Robert Trent Jones Golf Course in Ithaca, NY. Fungicides were not applied to these areas.

A total of 26 bags were placed in the turf that contained the wheat inoculum and 52 that contained infected turf inoculum. We recorded the disease that arose from the inoculum both before and during the time when the epidemic occurred elsewhere in the plot. We also recovered the bags and attempted to isolate the pathogen from them over the course of the summer and into the fall.

Natural inoculum studies

It seemed to us that if the infected tissues from the previous year were the source of epidemics, dollar spot patches in succeeding years should begin near the sites where patches were the previous year. Conversely, if this pattern was not

A photograph of a typical dollar spot patch. We were able to isolate the pathogen from the still-green, but diseased, tissue on the periphery of such patches, but not from the center. We also marked such patches with golf tees and then observed whether the disease occurred at the same site the next year. There was no indication that patches occurred at the same specific spot in the next season.

the case, then other sources of inoculum gave rise to the disease, or at least the likelihood of any specific old patch site giving rise to new disease is rare. To investigate this, we placed golf tees at the centers of patches and in apparently healthy turf. In the summer of 1999 we marked a total of 12 diseased patches and 8 healthy sites in four separate areas of the turf. We also marked another three healthy and three diseased sites in the summer of 2000. The next year, each of the sites was rated as to whether or not there was a dollar spot patch at the golf tee site.

We also attempted to isolate the pathogen using standard microbiological media from the centers and the edges of the plots. Separate isolations were attempted in the root, crown and leaf areas. Different parts of the plant were surface-sterilized or not and were ground or not. There were well over 100 attempts to isolate the pathogen.

Artificial inoculation of bentgrass seedlings. We followed the pattern of infection of seedlings after inoculation with artificial inoculum. Using established methods (6), seedlings of 'Pencross' bentgrass were established and then inoculated by placing inoculum of the pathogen in a corner of the flat. The disease progressed from these sites. The seedlings were examined microscopically following clearing and then staining with trypan blue, which specifically stains fungal structures (13). Hyphae of the pathogen were easily seen as robust dark hyphae.

Results

Inoculum added to sites

No disease ever developed near buried bags that contained artificially infected bentgrass. Disease did develop near bags that contained wheat inoculum just after placement but no disease developed thereafter, including at the time of the natural epidemic. There was no obvious growth of the pathogen from or on the bags that contained the bentgrass inoculum, but the surface of the bags containing the wheat inoculum were covered with a heavy black rind, or stroma, of the pathogen.

We could not isolate *S. homoeocarpa* from this rind, but fungal cultures did grow from the residual wheat material. These cultures were small and not recognizable as the pathogen at first, but many of them underwent a phase shift from the slow growing type to the rapid growth characteristic of the pathogen. Thus, the organism clearly existed as two separate growth types, the first being unrecognizable as *S. homoeocarpa*. Such phase shifts occur in other fungi; for example, the human pathogenic yeast *Candida albicans* undergoes reversible changes to several different morphological types (10) and these may be associated with changes in virulence (12).

Natural inoculum studies

There was no association whatsoever between the location of dollar spot patches or healthy turf in one year relative to the sites of patches the succeeding year. This suggests strongly that the infections in the succeeding year did not arise directly from the inoculum from diseased sites the preceding year.

We were unable to isolate the pathogen

#[a]	Isolate ID	Received From	Type[b]	Host	Location
1	sh101ko	K. Ondik	Turf Sample	Creeping bentgrass "Penncross"	New York
2	sh102ko	K. Ondik	Turf Sample	Creeping bentgrass "Cobra"	New York
3	sh103ko	K. Ondik	Turf Sample	Creeping bentgrass "Cobra"	New York
4	DS-21c	E. Nelson	Culture	Creeping bentgrass	New York
5	sh105ko	C. Peacock	Turf Sample	Creeping bentgrass "Crenshaw"	North Carolina
6	sh106ko	D. Hearn	Turf Sample	Creeping bent./Annu. Bluegrass	Massachusetts
7	16A	J. Vargas	Culture	Creeping bent./Annu. Bluegrass	Michigan
8	16B	J. Vargas	Culture	Creeping bentgrass	Michigan
9	16C	J. Vargas	Culture	Creeping bentgrass	Michigan
10	16E-19d	J. Vargas	Culture	Creeping bentgrass	Michigan
11	SH1-A	L. Giesler	Culture	Bluegrass	Nebraska
12	SH1-B	L. Giesler	Culture	Bluegrass	Nebraska
13	sh107ko	B. Clarke	Turf Sample	Creeping bentgrass "Crenshaw"	New Jersey
14	sh108ko	B. Clarke	Turf Sample	Creeping bentgrass "Crenshaw"	New Jersey
15	sh109ko	M. Chant	Turf Sample	Kentucky bluegrass/rye/fescue	Massachusetts
16	sh110ko	M. Chant	Turf Sample	Kentucky bluegrass/rye/fescue	Massachusetts
17	sh111ko	K. Ondik	Turf Sample	Creeping bentgrass	New York
18	sh112ko	K. Ondik	Turf Sample	Creeping bentgrass	New York
19	sh113ko	K. Ondik	Turf Sample	Creeping bentgrass	New York
20	S-9	W. Uddin	Culture	Unknown	Pennsylvania
21	S-82	W. Uddin	Culture	Unknown	Pennsylvania
22	S-83	W. Uddin	Culture	Unknown	Pennsylvania
27	Sh123BW	B. Walsh	Culture	Creeping bentgrass	Ontario
28	ShVWA3	B. Walsh	Culture	Creeping bent./Annu. Bluegrass	Ontario
29	ShVWC4	B. Walsh	Culture	Creeping bent./Annu. Bluegrass	Ontario
30	ShVWD3	B. Walsh	Culture	Creeping bent./Annu. Bluegrass	Ontario
31	ShVWF8	B. Walsh	Culture	Creeping bent./Annu. Bluegrass	Ontario
32	ShVWK1	B. Walsh	Culture	Creeping bent./Annu. Bluegrass	Ontario
33	UK-1	P. Vincelli	Culture	Unknown	Kentucky
34	UK-2	P. Vincelli	Culture	Unknown	Kentucky
35	CB-1	P. Vincelli	Culture	Unknown	Kentucky
36	CB-2	P. Vincelli	Culture	Unknown	Kentucky
37	sh114ko	J. Jacobs	Turf Sample	Unknown	New York
38	sh115ko	J. Jacobs	Turf Sample	Unknown	New York
39	sh116ko	J. Jacobs	Turf Sample	Unknown	New York
40	sh117ko	S. Humphreys	Turf Sample	Unknown	New York
41	sh118ko	S. Humphreys	Turf Sample	Unknown	New York
42	sh119ko	S. Humphreys	Turf Sample	Unknown	New York

[a] Isolate designations used throughout the text and in the graphs.

[b] Strains from turf samples isolated by K. Ondik.

Table 1. *Sclerotinia homoeocarpa* isolates used in this study

from any part of the killed turf in the centers of patches regardless of the method of sample preparation or whether or not we surface sterilized the tissues. Similarly, the pathogen could also not be isolated from the root-soil zone in killed plants at the center of patches. We were not, however, able to develop a selective medium for the pathogen because saprophytic fungi, especially *Trichoderma*, frequently developed. These known biocontrol fungi might have prevented identification of the slow-growing phase of the pathogen that was obtained from the centers of the wheat inoculum bags. It is likely, however, that we would have been able to isolate and identify the rapid-growing phase if it was present.

However, the pathogen was easily and reliably isolated from diseased, but still green, leaf tissue at the edge of the patches. These isolates were all of the rapid-growing phase. Thus, our methods of isolation were clearly able to detect and isolate the rapid-growing phase of the organism.

Artificial inoculation of bentgrass seedlings

Microscopic observation of seedlings from artificial inoculation always demonstrated the presence of the pathogen in the root and crown region of the plant, but never in the leaves. This is in marked contrast to the situation in the epidemic phase of the disease, where lesions typically occur on the leaves (11). Therefore, we also examined young lesions on leaves during the epidemic phase of the disease and were readily able to identify the typical hyphae of the pathogen.

Discussion

These results call into question some of our assumptions about dollar spot. First, the natural inoculation studies suggest that the patches that arise in one year did not result directly from infections that occurred the preceding year. If the patches in the second year arose directly from the previous year's inoculum they should have been located at similar sites. A possible reason for the

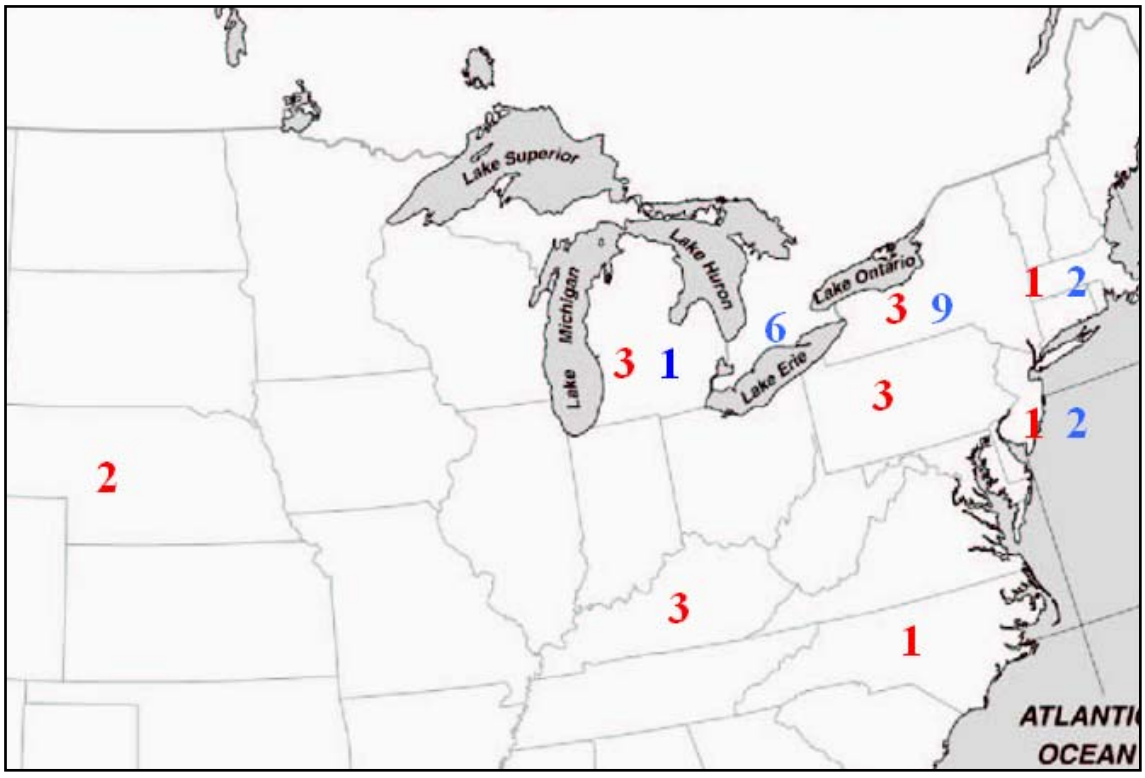


Figure 1. The strain collection divided into two separate groups that appeared to be geographically grouped. Strains in clade 1 (red numbers) were found in the western and southern states of our sample area (NE, MI, KY, NC) and those in clade 2 (blue numbers) were found in the northeastern USA and Ontario.

lack of inoculum from first-year patches giving rise to those in the second year is suggested by lack of viable isolatable pathogen from dead, diseased turf in the centers of dollar spot patches.

Second, there is a suggestion from the wheat inoculum studies that the pathogen exists in different phases. One of these may be more pathogenic than the other, but much more needs to be done before any conclusions can be made in this regard. If the pathogen in killed tissue reverts to the slow growing phase, this might explain our inability to isolate the pathogen from the dead parts of patches relative to our abilities to isolate from the still-green, but diseased, tissues.

The observation that the artificial inoculation results in infection of a different site of the plant than that observed in the field is also surprising. All of these observations suggest that there is much more to be learned about the biology of this pathogen.

From a practical point of view, a very important consideration is the concept that the disease we observe during the epidemic phase of the disease, and that is so difficult and expensive to control, is in reality the second phase of the disease. The first phase would be the initial establishment of the disease, perhaps in only a few places in the turf area and conceivably in the root and crown region. Then, and especially in close-mown turf such as that on golf greens, this infected material could be spread as infected turf tissues throughout the site. Such initial inoculum ought to be highly infective since it would be present in damaged grass particles that are highly nutritious to the pathogen.

This postulated disease cycle for dollar spot is similar to that known to occur with *Sclerotinia sclerotiorum* on beans. The pathogen survives as dark sclerotia that are similar to the stroma produced by *S. homoeocarpa*. These sclerotia give rise to sporulating bodies that eject spores into the bean leaf canopy. Generally, if the spores land on leaves they cannot cause infection because they have insufficient energy, but they can infect blossoms. Once these highly infected and nutritious blossoms come in contact with leaves, *S. sclerotiorum* has sufficient energy and 'inoculum potential' to infect the leaves especially if wounds are present (9).

If the idea that the epidemic phase of dollar spot results from secondary spread of an initial phase with few symptoms, the epidemic could be controlled before it starts. One “shot gun” method might be to start fungicide applications a month earlier than the epidemic is expected at a particular location and conclude spray applications if the dollar spot epidemic does not develop. This could be timed with weather data and other epidemiological forecasting systems.

An even more satisfactory long-term solution is to use modern molecular marking techniques to identify and follow the pathogen and thereby learn its basic biology. Such marking techniques could employ both toxicant resistance and visual markers such as green fluorescent protein and should permit specific identification and isolation of the pathogen in controlled lab and field situations and thereby overcome the limitations of the study we report here. The basic science that could be learned could be invaluable for improving more environmentally friendly and less costly disease control.

Diversity of the Pathogen

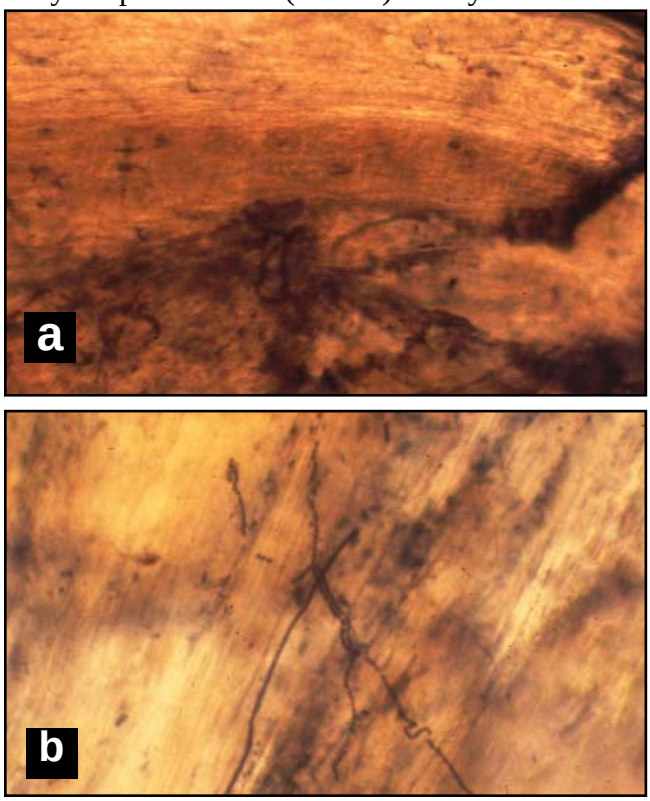
There are differing opinions as to the degree of genetic differences (diversity) between strains of the pathogen. Powell and Vargas suggested *S. homoeocarpa* is a single species with differing vegetative compatibility groups (7) while Kohn (5) suggested that the pathogen might include different species. Conversely, others have suggested that there is limited diversity (1, 3, 4), but these studies were conducted primarily on strains from a single geographical location (Ontario, Canada).

If the pathogen is highly genetically diverse, then management systems probably will be more difficult than if diversity is small. High levels of diversity may provide greater differences in innate or developed resistance to fungicides, different responses to management practices and different responses to changes in the genetics of turf, including differing responses to putatively

resistant cultivars. A possible consequence of the last point is that turf varieties developed to be resistant in one area or to a narrow range of isolates of *S. homoeocarpa* may be susceptible to strains not tested.

Methods and Materials

We first made a concerted effort to gather isolates through USGA networks and Cornell resources so that we would have a substantial amount of diversity based on geography and on host plants from which the isolates were obtained (Table 1). The principal method of examining the genetic diversity was based on differences in DNA similarities using Random Amplified Polymorphic DNA (RAPD) analyses. In this process, DNA is isolated from the organism (each separate strain of *S. homoeocarpa*) and subject to Polymerase Chain Reaction (PCR) amplification with known sequences of DNA as random primers.



(a) Infection of a the crown region of a bentgrass seedling by the pathogen, the hyphae are stained and are darker than the surrounding tissue, and (b) infection of leaf tissue by the pathogen that arose from natural infection from tissue stained similarly to that of the artificial inoculum. It must be pointed out that, in the natural infection, the hyphae are presumed to be that of *S. homoeocarpa* based on symptom expression and similarity to the appearance of hyphae in artificially inoculated plants, but since the infection occurred in the field, the identity of the hyphae cannot be absolutely determined.

In PCR, the primers bind to sections of the test DNA (template) to which they are identical, or nearly so. From the primer region, the enzyme DNA polymerase duplicates the DNA to give a double-stranded chain. This is heated for a few seconds to separate the new strand of DNA from the existing template and then cooled. Then, the primers not used in the previous cycle will bind to the newly produced DNA to start a new copy cycle.

After a few cycles of this reaction, specific DNA segments are produced. If the primer sequences are fairly commonly found in the target DNA, then a series of DNA fragments will be produced that are of different sizes that can be separated using gel electrophoresis. The more two strains are closely related the more the fragment pattern will be alike. The degree of similarity between patterns (and thus between strains in a collection) can be evaluated using statistical procedures.

In this study, we used two different primers (TTGAGCCACCC and GGTCAACCCT). The full methodology is reported in (2). Bands were visualized on Kodak Digital Science ID Image Analysis Software 3.0 (Rochester, NY). Data for each strain and primer combination were derived from two or more separate PCR runs. The degree of similarity (phylogenetic relationship) was estimated using RAPDistance, version 1.04 (The Institute for Advanced Study, The University of Western Australia, Crawley, WA) and used to build a plot (dendrogram) that describes graphically the relationship among strains and group of strains.

Results

Based on phylogenetic analysis, our strain collection exhibited a high degree of diversity. In fact, the strain collection divided into two separate

groups that appeared to be geographically grouped (Figure 1). Strains in clade 1 were found in the western and southern states of our sample area (NE, MI, KY, NC) and those in clade 2 were found in the northeastern USA and Ontario, Canada. Some strains from clade 1 were also found in NY, MA and NJ.

Clearly, the collection we assembled is too small to draw firm conclusions-more strains from each area should be assessed. However, these data suggest: (a) that there is significant diversity in the pathogen and that (b) some types of strains may be more adapted to particular areas than are others.

This data is somewhat at odds with conclusions from other research groups where the diversity has been suggested to be small (3, 4, 8). However, these results were primarily with strains from a limited geographical area, either Ontario, Canada, or Rhode Island. Our results, especially with strains from Ontario, suggest that diversity in that region is indeed limited, but when the entire midwestern, north-central, and northeast USA and Ontario are included, the diversity is larger than has been earlier suggested.

Further, Hsiang and Mahuku (3) have suggested that those populations within particular regions or sites may have been derived from small founding populations. Our results supported this conclusion. For example, we collected several strains from two different greens at the Cornell Turf Research area. Three strains from a new sand green were genetically quite similar and were in clade 2, while two strains from an older soil green were also similar to each other, but in clade 1. Therefore, isolates from each green were similar to each other but quite different from the strains from the other green.

If these conclusions are correct, then probably: (a) the pathogen is being introduced into greens at the time of establishment by unknown means and (b) that the pathogen is not native or endemic prior to green establishment. If so, and if the source of infection could be ascertained, then it might be possible, through sanitation, to avoid infection from occurring. This no doubt would be difficult given the spiked foot traffic that occurs on golf courses, but if it could be managed, it would provide better playing surfaces at lower costs than presently is the case. For example, the presence of low levels of the pathogen on turf seeds has not been examined.

Conclusions and Summary

The results of this study suggest that much more knowledge regarding the biology and diversity of the dollar spot pathogen could result in less disease, which would result in better playing surfaces at less cost to golf course managers. Items needing specific attention include the following.

1. What is nature of the structures by which *S. homoeocarpa* survives when it is not in its epidemic phase? For at least some of this time, environmental conditions would seem to be favorable for disease development.

2. Why does the epidemic phase of the disease appear suddenly and then diminish?

3. Is there an early, unrecognized phase of the disease and is dollar spot disease caused by spread from an initial source with few symptoms?

4. If so, can we intercept the disease before it becomes epidemic? This would reduce disease, increase turf quality and reduce environmental and financial costs associated with current disease management practices.

5. How much genetic diversity is there within *S. homoeocarpa*?

6. Are there geographic adaptations/differences in the pathogen population in different regions of North America?

7. If there is substantial diversity, is there reason to suspect that different pathogen types will differ in their resistance to new fungicides or that they may overcome resistance in new turf varieties?

8. Is the pathogen introduced into new golf course sites when it is not present in the region naturally? If so, can the source of infection be identified and controlled? If so, this also would reduce disease, increase turf quality and reduce environmental and financial costs.

Acknowledgements

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