**Geostatistical Analysis of Dollar Spot Epidemics Occurring on a Mixed Sward of Creeping Bentgrass and Annual Bluegrass**

B. J. Horvath,* A. N. Kravchenko, G. P. Robertson, and J. M. Vargas, Jr.

**ABSTRACT**

Dollar spot (Sclerotinia homoeocarpa F.T. Bennett) causes one of the most important diseases faced by golf course superintendents throughout the USA. Fungicides are effective, but fungicide resistance and regulation have restricted their use. Site-specific application allows more judicious use of fungicides, but the spatial heterogeneity of dollar spot incidence has been unknown. An intensive study was designed to determine the spatial distribution of dollar spot incidence using geostatistical analysis. Dollar spot foci were manually counted every 2 to 3 d in 200 quadrats in 2000, and in 888 quadrats in 2001 and 2002. Variograms showed spatial aggregation of dollar spot foci on a small scale, and remained stable throughout the growing season despite increasing disease incidence. The range parameter of the variograms was stable within a season, with values ranging between 2.5 and 9.5, 1.3 and 1.9, and 1.2 and 2.2 m in 2000, 2001, and 2002, respectively. Kriged maps of dollar spot incidence also show a stable pattern of spatial aggregation remains throughout the growing season. The locations of dollar spot clusters changed between seasons, but the varigrams from each season were similar, further suggesting stability of the spatial structure. It appears that limited secondary dispersal of S. homoeocarpa occurs during epidemics, as evidenced by the stable varigrams observed throughout the three growing seasons. The underlying factors involved in the observed stability are unknown.

B.J. Horvath, Dep. of Plant Pathology, Physiology, and Weed Science, Virginia Polytechnic Inst. and State Univ., Blacksburg, VA 24061; J.M. Vargas, Jr., Dep. of Plant Pathology, Michigan State Univ., East Lansing, MI 48824; A.N. Kravchenko and G.P. Robertson, Dep. of Crop and Soil Sciences, Michigan State Univ., East Lansing, MI 48824. This work was supported by a Project GREEEN grant (GR00-037) from the Mich. Agric. Exp. Stn., East Lansing. Received 5 Sept. 2006. *Corresponding author (bhorvath@vt.edu).

Abbreviations: PSV, proportion of structural variance; VCG, vegetative compatibility groups.

Dollar spot is caused by the pathogen Sclerotinia homoeocarpa F.T. Bennett (Bennett, 1937) and is a common pathogen throughout the world. In North America, the disease is an important pathogen destructive to both cool- and warm-season grasses (Smith et al., 1989; Smiley et al., 2005; Vargas, 2005). The disease is a major problem for most golf courses in Michigan, with epidemics beginning in June and continuing through late September. Dollar spot generally occurs during periods of warm days, cool nights, and heavy dewfall with leaf wetness periods conducive to infection (Warren et al., 1974; Smith et al., 1989; Vargas, 2005). Symptoms of the disease begin as small, hourglass-shaped lesions on leaves that can expand to blight the entire plant. The disease can also blight large areas of turf as a result of coalescing disease foci, causing extensive damage if left untreated. Diseased turf impairs the playing surface by creating depressions that affect golf ball roll on putting greens and leaves areas of bare soil where weed species can germinate and grow (Smith et al., 1989; Vargas, 2005).

The biology of S. homoeocarpa has not been studied extensively due to the relative ease with which this disease can be controlled with fungicides. Two reports from Great Britain suggest
that the pathogen is capable of producing sexual spores, asexual spores, or both (Jackson, 1973; Baldwin and Newell, 1992). Hsiang and Mahuku (1998) reported that a S. homoeocarpa population from Ontario exhibited random amplification of polymorphic DNA (RAPD) and intergeneric spacer–restriction fragment length polymorphism (IGS–RFLP) fingerprints that were in linkage disequilibrium indicating genetic recombination, but no fruiting bodies or spores were observed in their study. Other genetic diversity studies show S. homoeocarpa as a predominantly clonal pathogen (Raina et al., 1997; Powell and Vargas, 2001; Viji et al., 2004). Little research exists on inoculum sources, but a few reports contend that stromatized, infected tissue is the primary inoculum source for S. homoeocarpa (Fenstermacher, 1980; Smith et al., 1989; Vargas, 2005). Spread of this disease is widely believed (Fenstermacher, 1980; Smith et al., 1989; Smiley et al., 2005; Vargas, 2005) to be the result of mechanical movement of the pathogen on infected leaf blades during mowing operations and human transport of infected clippings on shoes, golf balls, etc., but Williams et al. (1996) showed no significant differences in the amount of dollar spot in plots where clippings were collected vs. plots where clippings were left in place.

Management of dollar spot is usually accomplished by N fertilization and fungicide applications. Increased N fertility leads to a reduction in dollar spot disease severity (Markland et al., 1969; Smith et al., 1989; Williams et al., 1996; Smiley et al., 2005; Vargas, 2005), but work by Couch and Bloom (1960) reported that the susceptibility of Poa pratensis L. was actually increased in higher fertility treatments in the greenhouse. They suggested, however, that this effect would probably be masked in the field because symptoms would not appear before the rapidly growing, infected leaf blades were mowed. Many fungicides are available to control dollar spot, but the presence of fungicide-resistant S. homoeocarpa populations to demethylation-inhibiting (Golembiewski et al., 1995), benimidazole (Detweiler et al., 1983), and dicarboximide (Detweiler et al., 1983) fungicides have forced many golf course superintendents to consider using the contact fungicide chlorothalonil (2,4,5,6-tetrachloro-1,3-benzenedicarbonitrile) to manage dollar spot. The USEPA has banned the use of chlorothalonil on home lawns and placed restrictions on its commercial use (USEPA, 1999). Because of these restrictions, more judicious use of chlorothalonil by golf course superintendents is needed. Making site-specific applications of N fertilizers and fungicides to manage dollar spot is desirable, but the spatial variability of dollar spot disease incidence is unknown.

Traditional statistical techniques used in the analysis of epidemiological data require adherence to assumptions such as the independence of samples and normality of data. It makes intuitive sense, however, that plants located closer in space to a diseased plant have a higher probability of becoming infected than plants located farther away. Important epidemiological questions arise from spatially explicit descriptions of disease incidence and severity.

Originally developed for the study of geological phenomena, geostatistics has found wide application in a number of fields including phytopathology (Lecoustre et al., 1989; Johnson et al., 1991; Munkvold et al., 1993; Stein et al., 1994; Dandurand et al., 1997; Larkin et al., 1995; Xiao et al., 1997; Rekah et al., 1999; Gavassoni et al., 2001; Luo et al., 2001; Savary et al., 2001; Paulitz et al., 2003). These tools have also been used to study the physicochemical properties of soils (Rossi et al., 1992; Robertson and Gross, 1995; Goovaerts, 1998), plant distributions and ecology (Oliver and Webster, 1986; Rossi et al., 1992), and microbial (Wollum and Cassel, 1984; Dandurand et al., 1995; Robertson et al., 1997; Franklin and Mills, 2003) and nematode (Todd and Tisserat, 1990; Webster and Boag, 1992; Robertson and Freckman, 1995) distributions. Geostatistics is not bound by the requirements of stationarity, which is the property of a data set in which the mean and variance do not change with location, but rather seeks to explain the variation between samples as a function of separation distance (Goovaerts, 1997; Isaaks and Srivastava, 1989). Spatial dependence, or semivariance, is detected by measuring the variation among samples separated by the same distance (lag) using the equation

$$\gamma(h) = \frac{1}{2N(h)} \sum_{(i,j) \in \Omega_h} (v_i - v_j)^2$$

where $\gamma$ is the semivariance, $h$ is the average separation distance between sample pairs, $N$ is the number of sample pairs, and $v_i$ and $v_j$ are the $i$th and $j$th data values at separation distance $h$ (Goovaerts, 1997; Isaaks and Srivastava, 1989). Results are plotted as a variogram that is displayed as semivariance vs. distance. Generally, as the distance between sample pairs increases, semivariance will also increase. Three key parameters describe the variogram: the nugget, sill, and range. The nugget is the discontinuity associated with experimental error and variability at scales smaller than the smallest sample interval, and is theoretically nil when $h = 0$, but in practice is defined as the $y$ intercept of the variogram (Isaaks and Srivastava, 1989). The range, which is the distance where semivariance does not increase with increasing separation distance, defines the boundary where samples are no longer spatially dependent. The sill is the semivariance value at the range, and is a measure of the overall sample variance. If the sill value is less than the total variance, then there may be spatial structure at scales greater than those sampled. The proportion of structural variance (PSV) can be expressed as a proportion of the total variance that is spatially structured. When this value approaches 1, a large proportion of
factors involved in an epidemic (Lecoultre et al., 1989; Gavassoni et al., 2001). Ideally, examining multiple locations with large sample sizes would enable the detection of spatial structure and the frequency with which any spatial structure occurs. Limited resources require a compromise, however, between the number of locations sampled and the sample frequency within a location. The impact of such compromises on the resulting geostatistical analyses is not trivial; different conclusions about spatial structure can arise depending on the scale studied.

Knowledge about the heterogeneity of dollar spot disease incidence is essential for the development of effective disease management strategies. The objectives of this study were to determine the spatial structure of dollar spot disease incidence, and evaluate changes in the structure on a putting green with time using geostatistical analysis.

MATERIALS AND METHODS

Sampling
Our study site was established in 2000 at the Robert Hancock Turfgrass Research Center in East Lansing, MI (42°43´48˝ N, 84°28´35˝ W) on a 10-yr-old, mixed sward of creeping bentgrass (Agrostis palustris Huds.) and annual bluegrass (Poa annua L.) on an Owosso–Marlette sandy loam (fine-loamy, mixed, semi-active, mesic Typic Hapludalf–Oxyaquic Glossudalf). An area of 0.017 ha (9.1 × 18.3 m) was maintained as a putting green and mowed in different radial directions 5 d wk−1 at a mowing height of 0.4 cm. Clippings were collected throughout the study. Nitrogen was applied at a rate of 24.4 kg N ha−1 mo−1 during the growing season from April to September of each year. No fungicide applications were made to the site throughout the duration of our study. We divided the site into 200 0.09-m² quadrats on a regular grid at 0.9-m intervals in 2000 (Fig. 1). In 2001 22 additional 0.09-m² quadrats were established arbitrarily at random locations within the study site, and all 222 quadrats were subdivided into four 0.023-m² subquadrats (Fig. 2). These additional locations were added and all quadrats subdivided to increase the number of sample locations at small separation distances. A 0.09-m² wooden frame divided into quadrants was placed on the centroid of each quadrat to delineate the four subquadrats at the time of sampling. Two corner positions at each location were marked on the turf with paint to place the frame in the same orientation at each sample time.

Data Collection
Dollar spot foci were counted three times per week in 2000 and twice per week in 2001 and 2002 at each location in the study area. Foci were counted when they became large enough to observe (~1.2 cm diam.). Counting within the study area was stopped when disease became so severe that there were too many disease foci to count accurately due to coalescing foci.

Geostatistical Analysis
Spatial continuity was measured using the variogram, calculated for each date using the geostatistical software package GS+ version 5.3b (Gamma Design Software, Plainwell, MI).
General geostatistical methods that were used in this study are described by Isaaks and Srivastava (1989). Lag distance and lag interval (separation distance class) were defined iteratively based on the distance and interval that yielded a smooth, well-behaved variogram. In 2000, a 12.2-m lag distance with a 1.5-m lag interval was used, and in 2001 and 2002, a 6.1-m lag distance with a 0.61-m lag interval was used due to the greater sample density. The resulting variograms were automatically fit using the GS+ software, which evaluated the goodness of fit based on least-squares weighting to the linear, exponential, spherical, and Gaussian models. The fits were then adjusted visually to avoid overextrapolation of the variogram model at small separation distances. A spherical model for variograms from two dates in each year, representing early and late epidemic development, were used to estimate dollar spot incidence using kriging, an interpolation technique. Kriged estimates of dollar spot incidence were generated for a grid of 0.019-m² with a search radius that was 70% of the range for a particular date. Because dollar spots were counted in an area and not at a discrete point, estimates at four grid nodes were averaged to produce a block estimate, and maps of dollar spot incidence were generated using the block estimates.

RESULTS

Sampling and Data Collection
Disease was observed and the number of dollar spot foci counted at each location beginning in early June of each year and continuing until 25 Aug. 2000, 9 Sept. 2001, and 13 Sept. 2002. Overall disease progress curves of the number of dollar spot foci per square meter and the area under the disease progress curve values show that the epidemic in 2002 was the most severe, followed by the epidemics in 2000 and 2001 (Fig. 3). Each progress curve shows an early-season disease period that is not as severe as the late-season period beginning in early August and continuing into September.

Geostatistical Analysis
Variograms calculated for each date in 2000 to 2002 show dollar spot counts were correlated across small separation distances, indicating spatial structure (Table 1, Fig. 4, 5, and 6). A spherical model best described the spatial structure present throughout most of the study (Table 1). Three dates, 6 June 2000, 9 June 2000, and 12 July 2002, showed no spatial structure (nugget effect). The yearly average PSV was 43% in 2000 and 2001, and 41% in 2002. The PSV generally ranged between 30 and 60% of the total variation for each date a variogram was calculated (Fig. 7). Several dates at the end of 2000 had PSVs that increased beyond the 30 to 60% range. Range values for each variogram in 2000 increased from 2.5 to 9.5 m through the early part of the epidemic, but several dates at the end of 2000 had ranges that increased from 13 to 31 m (Table 1). Ranges for 2001 and 2002 remained stable for the entire year, with values between 1.3 and 1.9 m in 2001 and 1.2 and 2.2 m in 2002 (Table 1). For illustration purposes, six representative variograms were selected from each year to account for disease progress during different phases of the epidemic (Fig. 4–6). Kriged maps of dollar spot incidence from the beginning and end of the epidemic in each year showed that dollar spot begins as clusters of individual infection foci that remain visible even as disease incidence increases late in the epidemic (Fig. 8). Anisotropy, or a change in spatial variability that changes with direction, was examined, and no anisotropic trends were apparent (data not shown).

DISCUSSION
This study quantified the spatial pattern dynamics of dollar spot incidence on a golf putting green during a 3-yr period using geostatistical analysis. To our knowledge, this is the first report using geostatistics to study the spatial pattern of dollar spot incidence. While our study was performed at a single location for 3 yr, raising questions of replication and repeatability, Campbell and Madden (1990) suggested that this is always a concern with spatial pattern studies and that true replication is usually unfeasible, if not impossible, given the constraints of natural environments and limited resources. We designed an intensive sampling scheme that enabled us to capture spatial information at a variety of...
separation distances from 0.15 to >19 m because there was no previous research to indicate an appropriate sample scale. Samples could have been allocated to more locations with fewer samples per location, but the lack of knowledge about the spatial structure of dollar spot incidence meant that there was a reasonable risk of not detecting spatial structure due to inappropriate sampling. Therefore, we chose to focus our efforts on detecting spatial structure, if it was present, and designed our study accordingly. The results of this study may or may not apply to other locations, as the spatial structure of disease incidence is probably influenced by many, currently unknown factors. The development of site-specific management practices in the future will rely on the knowledge and background of the underlying spatial structure of a disease. Our study does provide an estimation of the spatial structure of *S. homoeocarpa* and suggests testable hypotheses suitable for future research that may yield results that can be used in the development of site-specific management of dollar spot.

Disease progress curves in all 3 yr were similar and showed that dollar spot epidemics in Michigan begin in early to mid-June with a minor outbreak and then 

<table>
<thead>
<tr>
<th>Year</th>
<th>Date</th>
<th>Model type</th>
<th>Nugget (C₀)</th>
<th>Sill (C₀ + C)</th>
<th>Range (m)</th>
<th>PSV [C₀(C₀ + C)]</th>
<th>R²</th>
</tr>
</thead>
<tbody>
<tr>
<td>2000</td>
<td>6 June</td>
<td>Linear</td>
<td>0.82</td>
<td>0.82</td>
<td>0.507</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>9 June</td>
<td>Linear</td>
<td>0.13</td>
<td>0.13</td>
<td>0.209</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>12 June</td>
<td>Spherical</td>
<td>0.08</td>
<td>0.11</td>
<td>0.438</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>15 June</td>
<td>Spherical</td>
<td>0.57</td>
<td>0.83</td>
<td>0.312</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>19 June</td>
<td>Spherical</td>
<td>0.49</td>
<td>0.80</td>
<td>0.583</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>22 June</td>
<td>Spherical</td>
<td>2.00</td>
<td>3.77</td>
<td>0.789</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>26 June</td>
<td>Spherical</td>
<td>2.70</td>
<td>4.27</td>
<td>0.832</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>28 June</td>
<td>Spherical</td>
<td>4.10</td>
<td>7.00</td>
<td>0.934</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 1. Summary of model type, parameters (nugget, sill, and range), proportion of structural variance (PSV), and coefficient of determination (R²) of variograms based on geostatistical analysis of dollar spot epidemics on a mixed sward of creeping bentgrass and annual bluegrass in 2000, 2001, and 2002.
become much more severe during the months of August and September (Fig. 3). Our data agree with others who have shown the late-season phase of the epidemic is most damaging to a turf area (Fenstermacher, 1980; Smith et al., 1989; Powell and Vargas, 2001; Vargas, 2005). Dollar spot incidence declined during July, presumably because of hotter and more humid conditions that were less conducive for disease development.

Spatial structure was detected at small lag distances throughout epidemics in all 3 yr studied. The range parameters for each date do not measure the physical location of disease incidence, but they do show the average cluster size of dollar spot foci, or the distance across which counts of dollar spot foci are correlated. Therefore, if the range parameters change with time, then changes in the overall spatial structure would also be occurring. The range values estimated by the spherical variogram model for each date were less stable in 2000, possibly due to the rapid increase in disease incidence later in the season (Fig. 3) and the lack of small-scale sampling. After the sampling design was changed to a high-density design, the range values for variograms from 2001 and 2002 were stable for the entire year (Table 1, Fig. 7), with values varying by 0.6 m in 2001 and 1.0 m in 2002. Spatial structure was not detected (nugget effect) on three dates (Table 1) that had very low disease incidences, indicating that the sampling design was not capable of detecting spatial structure below a certain disease incidence threshold. Stable PSV values (Fig. 7) throughout the study and disease incidence maps (Fig. 8) generated by interpolation (kriging) of disease incidence data support the variogram data and illustrate the stability of the spatial structure with time. The maps show that dollar spot epidemics develop as small clusters of infection foci, and while the physical location

Figure 4. Variograms of dollar spot epidemic caused by S. homoeocarpa on a mixed sward of creeping bentgrass and annual bluegrass on six dates in 2000 selected to be representative of changes in disease progress (Fig. 3) throughout the growing season.
of the clusters is different each year, spatial structure that is established at the onset of the epidemic remains present as disease incidence increases throughout the season. The overall stability of these values with time suggests that the spatial structure of disease incidence is governed by underlying factors that are similarly stable.

Several working hypotheses can be generated that support our finding of a stable spatial structure for dollar spot disease incidence. Our study was not able to determine which of these factors influence the observed spatial structure, but we can suggest possible influences that are consistent with our data. One possibility is that the ability of the pathogen to spread is limited. Others include variation in the virulence of the pathogen, or variation in the susceptibility of the host plants.

Spread of a pathogen can be accomplished either by the dissemination of inoculum through spore production or other means, or through the direct movement of infected materials from one place to another. The method of dissemination used by *S. homoeocarpa* for dispersal is unclear. The pathogen has been shown to produce ascospores and conidia in laboratory experiments and in the field in the United Kingdom (Bennett, 1937; Jackson, 1973; Baldwin and Newell, 1992), but spore production in the field, if it occurs, has not been observed outside of the U.K. Four of eight populations in a field experiment (Hsiang and Mahuku, 1998) in Ontario showed significant linkage disequilibrium, indicating that these populations were not random mating. Asexual reproduction through mycelial fragments seems to be the major inoculum source for this pathogen, and fits the observed linkage disequilibrium data; however, linkage disequilibrium values in the other four populations indicated that it was possible that sexual recombination was taking place. If *S. homoeocarpa*

**Figure 5.** Variograms of dollar spot epidemic caused by *S. homoeocarpa* on a mixed sward of creeping bentgrass and annual bluegrass on six dates in 2001 selected to be representative of changes in disease progress (Fig. 3) throughout the growing season.
inoculum were dispersed (via spores or other means) in the present study, the resultant polycyclic disease development would alter the spatial structure. More specifically, the range values of calculated variograms would be expected to change during the polycyclic phase, even if dispersal were limited. Previous studies in the *Venturia inaequalis* pathosystem have shown that dispersal of *V. inaequalis* ascospores is sharply reduced beyond 5 to 6 m from a source (Kaplan, 1986), and that the effective dispersal radius is about 20 m (Hsiang et al., 2000). Our data, from 888 locations, shows range values were stable in 2001 and 2002, with values generally between 1 and 2 m and varying by just 0.6 m in 2001 and 1.0 m in 2002 after sampling was increased. The stability and small variance associated with the range parameters support the possibility that limited spore dispersal and movement take place.

Another possible method of spread would be through direct movement of the pathogen. Several reports have ascribed dispersal of *S. homoeocarpa* to the movement of mycelial fragments on diseased tissue via human and mechanical transport (Fenstermacher, 1980; Smith et al., 1989; Vargas, 2005). We are confident that dispersal of infected tissue is possible; it has been used as an inoculation technique for establishing dollar spot in experimental plots at our research center. We would expect an increase in the variogram range with time as clusters expanded radially if mowing practices were a factor in the dispersal of *S. homoeocarpa*. While the study area was mowed in different radial directions five times per week and clippings were collected, however, range values for variograms in 2001 and 2002 fluctuated by <1 m. Other geostatistical studies of plant pathosystems have generally observed changes in spatial structure as epidemics progress.
Direct movement of the pathogen is another source of spread that could be involved in the observed spatial pattern. Spatial analysis of *Fusarium oxysporum* f. sp. *radicis-lycopersici* epidemics in tomato and work on soybean [Glycine max (L.) Merr.] sudden death syndrome showed that movement of the pathogens along roots was effective as a short-distance dispersal mechanism (Rekah et al., 1999; Luo et al., 2001). Epidemics of rice (*Oryza sativa* L.) sheath blight, caused by *Rhizoctonia solani*, showed a large nugget effect for spatial structure occurring at separation distances greater than four hills (~1 m), and suggested that processes occurring at the single-hill level (~0.2 m), where individual tillers could come into contact with neighboring diseased or healthy tillers, is crucial for development of spatial structure in epidemics (Savary et al., 2001). Direct contact of healthy plants by infected plants in our study area would take place across a very small scale because of the very small size of the turfgrass plants on a putting surface. It is not known if these methods of spread occur in the dollar spot pathosystem, but changes occurring at these scales could easily accumulate during the growing season and would result in a small variation in the range values similar to the variation we observed in 2001 and 2002 (0.6 and 1.0 m, respectively). These possibilities could be tested in the future by evaluating *S. homoeocarpa*’s ability to move from plant to plant, through thatch, and through soil.

The susceptibility of the host and virulence of the pathogen may also play a role in spatial structuring of dollar spot epidemics. The predominant grasses found on golf course putting surfaces are creeping bentgrass and annual bluegrass. Both of these grasses are nonuniform in their susceptibilities to *S. homoeocarpa* (Cole et al., 1969; Vincelli and Doney, 1997; Viji et al., 2004). Creeping bentgrass is a synthetic cultivar, meaning that each seed is genetically distinct, and results in a range of variation in susceptibility and resistance to dollar spot even within a particular cultivar. Annual bluegrass is a noncultivated grass that invades putting surfaces as a weed, and also is known to be genetically variable (Vargas, 2005). The area we studied was at least 10 yr old and was a mixed sward of creeping bentgrass and annual bluegrass. With time, the competitiveness of each seedling could govern the particular genotypes that established a site. These successful genotypes would then be more or less susceptible to dollar spot, and this effect would be observed as a mosaic of disease incidence with a spatial structure corresponding to the spatial structure of the grasses. Variability within the pathogen could also play a role in the spatial structure we observed. Powell and Vargas (2001) showed seasonal variation in the vegetative compatibility groups (VCGs) of *S. homoeocarpa* present in
a turf area. Viji et al. (2004) also showed differences in pathogenicity of different VCGs. Thus, it is possible that variation in the pathogen could lead to changes in the spatial structure of epidemics, depending on the makeup of the S. homoeocarpa population present. Since previously infested tissue is believed to be the primary inoculum source for dollar spot (Fenstermacher, 1980; Smith et al., 1989; Vargas, 2005), differences in the incidence of disease could also result in changes to the spatial structure.

Overall, we identified that dollar spot epidemics display a relatively stable spatial structure organized on a small scale that is relatively unaffected as disease incidence increases. While the factors underlying the observed spatial structure are unknown, it is possible that the factors responsible for the structure would be similarly stable. We suggest testable hypotheses that future studies can determine: what influences, such as mowing, spore dispersal, local spread of mycelium through contacts with diseased tissue, variation in susceptibility of hosts, and differences in the pathogenicity of S. homoeocarpa isolates, affect spatial structuring that is present. It is likely that many factors contribute to the spatial structure of S. homoeocarpa, and future research should include an evaluation of other geographic locations to determine the generality of the spatial structure that we observed, and testing of the proposed hypotheses to determine those factors responsible for any spatial structure that is present.

**References**


Fenstermacher, J.M. 1980. Certain features of dollar spot dis-
ease and its causal organism *Sclerotinia homoeocarpa*. p. 49–53. 
In P.O. Larsen and B.G. Joyner (ed.) Advances in turfgrass pathology. Harcourt, Brace, Jovanovich, Duluth, MN.


