

**EPIDEMIOLOGY AND DISEASE FORECASTING SYSTEM FOR DOLLAR SPOT
CAUSED BY *SCLEROTINIA HOMOEOCARPA* F. T. BENNETT**

A Thesis

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by

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ABSTRACT

EPIDEMIOLOGY AND DISEASE FORECASTING SYSTEM FOR DOLLAR SPOT CAUSED BY *SCLEROTINIA HOMOEOCARPA* F. T. BENNETT

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University of Guelph, 2000

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The influence of leaf wetness duration (L) and temperature (T) on dollar spot (*Sclerotinia homoeocarpa*) severity in controlled environments was determined by inoculating pots of creeping bentgrass (*Agrostis palustris* 'Penncross') and placing them in cabinets set at 10, 17.5 or 25 °C for 0, 12, 24, 36, or 48 h of L. Linear regression analysis was used to develop the model that estimated focus diameter, y (cm), for L and T combinations, where:

$$y = -0.3 + [-1.475 + (2.087 \times 10^{-1})T + (2.497 \times 10^{-2})L + (-4.284 \times 10^{-3})T^2 + (-6.485 \times 10^{-4})TL]^2.$$

This model explained 83.9 % of the variation within the data ($P < 1.0 \times 10^{-38}$, $n = 141$). Epidemiology studies were conducted during 1996 - 1998 and disease progress curves were best fit to exponential or logistic models, both of which describe polycyclic diseases. Dollar spot epidemics started when 9 - 10 days with mean air T >16 °C accumulated after May first. Step-wise multiple regression did not correlate weather variables with dollar spot epidemics. In a two-year study, ten impedance leaf wetness sensors were evaluated for accuracy and precision of monitoring dew onset and dissipation on creeping bentgrass. The Campbell Scientific Model 237 sensor, when placed within a Kentucky bluegrass canopy maintained at fairway height, was the best sensor for monitoring leaf wetness duration (LWD) on

creeping bentgrass greens. For the entire dew duration of approximately 15 h, this sensor estimated on average within 1.7 h of the actual condition. The estimates from this sensor may be used in currently available turfgrass disease forecasting models because such variation in LWD measurement does not impact the capacity of the models to predict disease.

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1. LITERATURE REVIEW¹

1.1 INTRODUCTION

Turfgrass has been utilized by humans for more than 10 centuries to enhance their environment, but the modern turfgrass industry developed primarily during the past three decades, largely in response to increased population growth and urbanization. Turfgrass culture represents an important economic component of horticulture in North America with annual expenditures of \$25 to \$45 billion in the United States between 1982 and 1993 (Beard & Green 1994, Gibeault & Cockerham 1985). In return, turfgrass provides numerous benefits, including functional (e.g., reduction of soil erosion, pollution and noise), recreational (e.g., provision of recreational surfaces for improved health and safety) and aesthetic benefits (e.g., improved aesthetic value and community pride) (Beard & Green 1994, Gordon et al. 1996). Because of these economic and environmental benefits, turfgrass and turfgrass health have become increasingly important.

Dollar spot (*Sclerotinia homoeocarpa* F. T. Bennett) is one of the most important diseases that affect turfgrass; it can cause considerable damage, particularly to highly maintained golf course putting greens, closely mown fairways, and bowling greens (Goodman & Burpee 1991). This disease can also damage less intensively managed turfgrass grown on home lawns, recreational and athletic facilities, and educational or industrial properties. Dollar spot reduces the aesthetic and playing quality of infected

¹Modified from the publication Walsh, B., S. S. Ikeda, and G. J. Boland. 1999. Biology and management of dollar spot (*Sclerotinia homoeocarpa*); an important disease of turfgrass. HortScience 34: 13-21.

turfgrass, and can contribute to weed encroachment and plant death (Smith et al. 1989). Dollar spot is a widespread disease that affects many turfgrass species throughout North America, Central America, Australia, New Zealand, Japan, the British Isles and continental Europe (Couch 1995, Fenstermacher 1980, Vargas 1994). Except for western Canada and the Pacific northwest region of the United States, dollar spot is the most common disease of turfgrass in North America (Couch 1995). The persistent nature of this disease in turfgrass swards often requires intensive control measures. As a result, more money is spent to manage dollar spot than to control any other turfgrass disease on golf courses (Goodman & Burpee 1991).

1.2 DOLLAR SPOT

1.2.1 Disease Symptoms

On individual blades of grass, the first symptom of disease is yellow green blotches that progress to a water-soaked appearance (Smith 1955). As disease advances, infected tissues bleach to a straw-colored tan with reddish-brown borders, and lesions often enlarge across the entire leaf (Smiley et al. 1992, Smith 1955). Reddish-brown lesion borders do not typically occur on annual bluegrass (*Poa annua* L.) (Couch 1995, Vargas 1994). Usually, entire leaves are blighted but, in some cases, only portions of a leaf become necrotic (Couch 1995, Smiley et al. 1992).

Symptoms of dollar spot on turfgrass swards vary according to turfgrass species and management practices. On closely-mown turfgrass, such as that on golf course putting greens, sunken, circular, straw-colored patches develop that range in size from a few blades

of grass to spots the size of a silver dollar (5.0 to 7.5 cm diameter) (Smith 1955), hence the name “dollar spot” (Couch 1995, Vargas 1994). Necrotic patches stand out because they contrast sharply with adjacent healthy turfgrass. If disease progresses, these necrotic patches coalesce to form larger areas of necrotic, straw-colored turfgrass (Couch 1995, Smith 1955). As mowing height is increased on golf course fairways, parks, and home lawns, patches of blighted grass range from 6 to 12 cm in diameter, but may coalesce to form large, irregular shaped areas of injured turfgrass (Couch 1995).

In addition to symptoms of disease, mycelial growth of the pathogen also can be observed. When dew is present on leaves, or during extended periods of high relative humidity, white cobweb-like aerial mycelia of the pathogen can be seen growing on the surface of turfgrass and extending from leaf to leaf (Monteith & Dahl 1932, Smith 1955). However, mycelia can also be confused with spider webs, downy seed tufts of cottonwood trees (*Populus* spp.) (Smiley 1983), and mycelia of *Pythium*, *Rhizoctonia* and *Nigrospora* (Smiley et al., 1992). Therefore, correct diagnosis of disease is dependent on additional information, such as climatic conditions, history of the site, management practices and isolation of the pathogen.

1.2.2 The Pathogen

The pathogen that causes dollar spot is currently classified as *S. homoeocarpa*; however, the taxonomic status of this causal agent remains controversial. Most authorities believe this fungus will eventually be reclassified but difficulty in obtaining reproductive structures for study has prevented resolution of its taxonomic classification. In 1932, Monteith and Dahl first described the symptoms of dollar spot and considered the fungus to

be a *Rhizoctonia* sp. based on its similarity to brown patch (*R. solani* Kühn) of turfgrass. Later, Bennett (1937) examined isolates of the pathogen from Britain, the United States, and Australia, and categorized these isolates into three distinct strains of the fungus, described as: a) a "perfect strain" of British origin that produced ascospores and conidia; b) an "ascigerous strain" of British origin that produced ascospores and microconidia; and c) "non-sporing strains" of British, American, and Australian origin that did not produce ascospores, but did occasionally produce rudimentary apothecial initials. All three strains were considered a single fungus, despite differences among individual strains in ascospore size, and the presence of conidia or ascospores, or both. Comparisons of pathogenicity were not reported. Bennett (1937) noted that apothecia occasionally arose from small aggregates of sclerotial cells, termed "micro-sclerotia". He therefore considered the fungus to be a *Sclerotinia* sp. Fuckel., based on the broad definition of this genus at that time, which included fungi that produced conidia. Bennett assigned the species name *homoeocarpa* to these isolates because the "perfect strain" of the fungus produced cupulate or apothecial-shaped structures for production of both conidia and ascospores.

The original description of *S. homoeocarpa* by Bennett (1937) can be summarized as the "perfect" strain having cupulate (0.5 to 0.8 mm) to disk- or funnel-shaped (1.0 to 1.5 mm) apothecia, pale cinnamon to dark brown in colour, with prosenchymatic exciples, arising from microsclerotia or expansive sclerotial flakes or patches. The apothecial stalk was cylindrical, slender, flexuous, 5 to 10 mm long, arising singly or in clusters, simple or branched in the upper part. Asci were cylindroclavate, inoperculate, and commonly measured 150 to 165 x 10.4 μm . Ascospores were in groups of eight, uniserate, hyaline, oblong-elliptical, bi-guttulate, unicellular, often developed a delicate median septum during

germination, and commonly measured 16.0 x 5.5 μm .

In 1945, Whetzel described the family Sclerotiniaceae, restricting the genus *Sclerotinia* to include fungi in which the apothecium arose from a tuberoid sclerotium that was formed free on aerial mycelia. Based on these criteria, *S. homoeocarpa* was excluded from this family (Kohn 1979a, Whetzel 1945) because true sclerotia have not been found on turfgrass affected by dollar spot (Baldwin & Newell 1992). Bennett (1937) reported conidia were produced by the perfect strain, but other researchers have not found conidia (Baldwin & Newell 1992, Jackson 1973, Smith 1955). Whetzel (1946) and Jackson (1973) considered this fungus to be either a *Rutstroemia* sp., or possibly synonymous with a previously-described fungus, *Ciboria armeriae* Von Hohnel. Powell and Vargas (1999) proposed the telomorphic isolate from Britain used to describe the species *S. homoeocarpa* was more closely related to *Rutstroemia henningsiana* (Ploettn.) Dennis, and *Rutstroemia cuniculi*, than to isolates responsible for causing dollar spot. ITS1 and ITS2 sequences of isolates from North America expressed sequence divergences from isolates from Britain of 16 and 15 bases, respectively, and difference in mycelial and stromatal morphology between these groups also exist. However, the taxonomy of *Rutstroemia*, designated by White (1941), was deemed uncertain by Dumont and Korf (1971). Hence, the taxonomic status of this fungus was uncertain and remained as *S. homoeocarpa*.

Kohn (1979a), in a monographic revision of the genus *Sclerotinia*, concluded that *S. homoeocarpa* was not a true *Sclerotinia* sp. and, based on personal communication with Korf (Kohn 1979a), proposed that it was more appropriately classified in *Lanzia* Sacc. and *Moellerodiscus* Henn. Furthermore, Kohn (1979b) suggested that symptoms attributed to dollar spot were caused by more than one species. If this suggestion is correct, then the

correct term for dollar spot would be "dollar spot syndrome" (Jackson 1973, Smiley 1983).

Continued assessment of the taxonomic classification of this pathogen has been prevented by the recalcitrant nature of isolates to produce teleomorph or anamorph structures. Fertile apothecia are rarely observed in nature and cultures on artificial media often yield sterile apothecia (Fenstermacher 1970, Jackson 1973, Baldwin & Newell 1992). Production of fertile apothecia by isolates of the pathogen and satisfaction of Koch's postulates are required to clarify the proper name and taxonomic position of this pathogen. Fertile apothecia were reported from stromata in field samples of *Festuca* turfgrass (Baldwin & Newell 1992), and this report may stimulate renewed interest in taxonomic assessments of this pathogen. Proper taxonomic placement of the pathogen responsible for dollar spot remains unresolved (Rossman et al. 1987); hence, for the remainder of this review, the causal agent(s) of dollar spot is referred to as *S. homoeocarpa*.

1.2.3 Host Range

Sclerotinia homoeocarpa can cause disease in at least 40 plant hosts, but most hosts are classified within the grass family *Poaceae*. Additional hosts were reported from the *Cyperaceae* (sedge family), *Caryophyllaceae* (pink family), *Convolvulaceae* (morning-glory family) and the *Leguminosae* (pea family) (Table 1.1). Dollar spot in the United Kingdom is primarily confined to fescues (Couch 1995); however, in North America, all commonly cultivated turfgrass species are susceptible to *S. homoeocarpa*. Although all species are considered susceptible, variations in disease severity, and the ability to recover from disease, occur among cultivars of some species (Doney et al. 1994, Hodges et al. 1975, Hsiang 1995, Moss et al. 1995a, 1995b, Myer & Smejkal 1995, Vincelli et al. 1997). Cultivars that are less

Table 1.1 Known hosts of *Sclerotinia homoeocurpa*

Scientific name	Common name	Reference
Caryophyllaceae (= Alsiniaceae): Pink family^Z		
<i>Sagina procumbens</i> L. ^Y	Pearlwort Birdseye	Fenstermacher 1970
Convolvulaceae: Morning-glory family^W		
<i>Dichondra repens</i> J.R.Forst. & G. Forst. = <i>D. micrantha</i>	Mercury bay weed	Boesewinkel 1977
Cyperaceae: Sedge family^W		
<i>Cyperus esculentus</i> L. ^Y <i>Cyperus rotundus</i> L.	Nutsedge	Bain 1964
<i>Carex</i> sp. L.	Sedge	Whetzel 1946
Leguminosae (= Fabaceae): Pea family^W		
<i>Arachis glabrata</i> Benth. ^V	Perennial peanut	Hoover & Kucharek 1995
Poaceae (= Gramineae): Grass family^W		
<i>Agrostis</i> sp. L.	Bent grass	Bennett 1937, as cited by Smith 1955

Table 1.1 continued...

Scientific name	Common name	Reference
<i>Agrostis alba</i> L. ^Y	Redtop	Beard 1973, Britton 1969, Couch 1995, Sprague 1950, U.S. Dept. of Agriculture 1960
<i>Agrostis canina</i> L.	Velvet bent grass	Beard 1973, Britton 1969, Couch 1995, Fenstermacher 1970, Smith 1959, Sprague 1950, U.S. Dept. of Agriculture 1960
<i>Agrostis palustris</i> Huds. ^X <i>Agrostis stolonifera</i> L.	Creeping bent grass	Beard 1973, Britton 1969, Couch 1995, Fenstermacher 1970, Smith 1955, Smith 1959, Sprague 1950, U.S. Dept. of Agriculture 1960
<i>Agrostis tenuis</i> (L.) Sibth. ^X	Colonial bentgrass New Zealand browntop Rhode Island bent grass	Britton 1969, Boesewinkel 1977, Couch 1995, Smith 1955, Smith 1959, Sprague 1950, U.S. Dept. of Agriculture 1960
<i>Cynodon dactylon</i> (L.) Pers. ^Y	Coastal Bermudagrass Wiregrass Bermudagrass	Bain 1962, Beard 1973, Britton 1969, Couch 1995, Fenstermacher 1970, Freeman 1967
<i>Cynodon incompletus</i> var. <i>hirsutus</i> deWet et Harlan ^V	Bermudagrass	Beard 1973

Table 1.1 continued...

Scientific name	Common name	Reference
<i>Cynodon magennisii</i> Hurcombe ^v	Magennis Bermudagrass	Beard 1973
<i>Cynodon transvaalensis</i> Burt-Davy	Bermudagrass African Bermudagrass Transvaal dogtooth grass	Beard 1973
<i>Digitaria didactyla</i> Willd.	Blue couch grass	Simmonds 1958
<i>Digitaria ischaemum</i> (Schreb.) Muhl. ^y	Smooth crabgrass	Fenstermacher 1970
<i>Eremochloa ophiuroides</i> (Munro.) Hack.	Centipede grass	Beard 1973, Britton 1969, Couch 1995, Freeman 1967
<i>Festuca</i> sp. L.	Fescue	Bennett 1937, as cited by Smith 1955
<i>Festuca elatior</i> L. = <i>Festuca arundinacea</i> Schreb.	English bluegrass ^v Tall fescue Reed fescue Alta fescue	Couch 1995
<i>Festuca ovina</i> L. = <i>F. saximontana</i> Rydb.	Sheep fescue	Britton 1969, Couch 1995, Smith 1959, Sprague 1950

Table 1.1 continued...

Scientific name	Common name	Reference
<i>Festuca ovina</i> var. <i>duriuscula</i> (L.) Koch	Hard fescue ^v	Hodges et al. 1975
<i>Festuca rubra</i> L.	Red fescue	Beard 1973, Boeosewinkel 1977, Britton 1969, Couch 1995, Fenstermacher 1970, Hodges et al. 1975, Smith 1955
<i>Festuca rubra</i> var. <i>commutata</i> Gaud.-Beaup. = <i>Festuca fallax</i> Thuill.	Chewing fescue	Hodges et al. 1975, Smith 1955
<i>Holcus lanatus</i> L.	Common velvet grass Yorkshire fog	Couch 1995, Smith 1955
<i>Lolium multiflorum</i> Lam. = <i>Lolium italicum</i>	Italian ryegrass Australian ryegrass	Britton 1969, Freeman 1967
<i>Lolium perenne</i> L.	Perennial ryegrass Annual ryegrass	Wilkinson et al. 1975
<i>Paspalum notatum</i> Flügge.	Bahia grass	Bain 1962, Beard 1973, Britton 1969, Couch 1995, Fenstermacher 1970, Freeman 1967

Table 1.1 continued...

Scientific name	Common name	Reference
<i>Poa</i> sp. L.	Bluegrass	Bennett 1937, as cited by Smith 1955
<i>Poa annua</i> L.	Annual bluegrass Speargrass ^Y Low bluegrass Six weeks grass Dwarf meadow grass	Beard 1973, Boesewinkel 1977, Britton 1969, Couch 1995, Sprague 1950, U.S. Dept. of Agriculture 1960
<i>Poa arachnifera</i> Torr.	Texas bluegrass	U.S. Dept. of Agriculture 1960
<i>Poa arida</i> Vasey ^Y	Plains bluegrass	U.S. Dept. of Agriculture 1960
<i>Poa pratensis</i> L.	Kentucky bluegrass June grass Spear grass	Beard 1973, Britton 1969, Couch 1995, Fenstermacher 1970 Smith 1955, Sprague 1950 Wilkinson et al. 1975, U.S. Dept. of Agriculture 1960
<i>Poa trivialis</i> L.	Rough bluegrass Rough-stalk bluegrass Rough-stalk meadow grass	Smith 1959
<i>Puccinellia maritima</i> (Hudson) Parl. ^Y	Seaside alkali-grass	Halcrow 1965

Table 1.1 continued...

Scientific name	Common name	Reference
<i>Stenotaphrum secundatum</i> (Walt.) O. Kuntze	St. Augustinegrass	Beard 1973, Britton 1969, Freeman 1967
<i>Zoysia japonica</i> Steud.	Zoysiagrass ^v Korean or Japanese lawngrass	Beard 1973, Britton 1969, Couch 1995
<i>Zoysia matrella</i> (L.) Merrill.	Manila grass Zoysia grass Japanese carpet grass	Beard 1973
<i>Zoysia tenuifolia</i> Willd. ex. Trin.	Muscarene grass ^x Korean velvet grass	Beard 1973, Couch 1995

^z All plants were classified according to Bailey and Bailey (1976) unless indicated otherwise.

^y Taxa classified according to Gleason and Cronquist (1991).

^x Taxa classified according to Hitchcock (1971).

^w Families classified according to Smith (1977).

^v Taxa classified according to Brako et al. (1995).

susceptible to disease should be considered as part of an integrated disease management program. Until resistant cultivars become available, blends of turfgrass cultivars that are tolerant or resistant to prevalent diseases within a region are recommended for improved disease management (Schroeder & Sprague 1994).

1.2.4 Epidemiology

Sclerotinia homoeocarpa is believed to overwinter as darkly-pigmented stromata remaining on margins of dollar spot lesions from previous epidemics (Britton 1969, Couch 1995, Smiley et al. 1992). Fenstermacher (1980) suggested that *S. homoeocarpa* more likely survives as dormant mycelium in infected grass crowns and tissues. The pathogen primarily infects leaves via mycelial growth into cut leaf tips and stomata, but direct penetration into the leaves also occurs (Endo 1966, Monteith & Dahl 1932). Traditionally, conidia and ascospores were believed to be of minor importance to the spread of disease because these propagules were rarely observed in nature. However, fertile apothecia in turfgrass swards were reported, suggesting that ascospores are a potential source of initial inoculum in the spring (Baldwin & Newell 1992). Local distribution of dollar spot occurs when mycelium grows from a diseased leaf to a healthy leaf in close proximity. Over larger areas, the pathogen is distributed by physical displacement of the fungus. Humans can transport infested and diseased material, such as grass clippings, on the bottoms of golf shoes, on golf cart tires, and on maintenance equipment such as mowers, sprayers, and irrigation hoses (Smith 1955).

Environmental factors affect the rate of disease progress. Controlled temperature experiments revealed that the minimum, optimal and maximum temperatures for growth of

S. homoeocarpa were 4.5, 26.8 and >32 °C (32 °C was the highest temperature tested, at which the fungus still grew), respectively, on potato dextrose agar (PDA) (Endo 1963). *Sclerotinia homoeocarpa* is likely to infect and cause disease at temperatures ranging from 15 to 27 °C (Couch 1995, Endo 1963) but reaches its peak growth rate and maximum pathogenicity when temperatures are between 21 and 27 °C and atmospheric humidity is >85% (Couch 1995, Endo 1963). Although it does not infect roots directly, *S. homoeocarpa* has been associated with a root damaging mycotoxin produced at temperatures >15.5 °C. Culture filtrates of *S. homoeocarpa* contained a nonionic and heat stable chemical that caused creeping bentgrass (*Agrostis palustris* Huds.) roots to cease growth, thicken, and turn brown in color (Malca & Endo 1965). Baldwin and Newell (1992) found that ascospores were released from fertile apothecia after incubation at 21°C with a 12 h light cycle for 48 h, and ascospores germinated in water within 24 h at 20 °C. There is considerable variability in response to environmental conditions among isolates of *S. homoeocarpa* recovered from various locations (Bennett 1937, Endo 1963, Freeman 1967). In general, variants of dollar spot are active from late spring to autumn when days are warm and humid with nights that result in heavy dew (Smiley et al. 1992).

1.2.5 Disease Management

1.2.5.1 Cultural Controls

Cultural practices can be very effective for the management of dollar spot in turfgrass. Most fungi require free water on the leaf surface to infect; therefore, the amount and duration of leaf wetness is an important factor in dollar spot occurrence (Williams et al. 1996). Williams et al. (1996) found that dew displacement on turfgrasses reduced leaf

wetness duration by several hours. To minimize the dew period, the following practices have been suggested: pole, syringe, and mow turfgrass in early morning to remove water, and prune or remove trees and shrubs to increase air circulation and solar radiation so dew dries more quickly.

Moisture stress can also predispose turfgrass to dollar spot. Turfgrass suffering from moisture stress in greenhouse studies was more susceptible to disease (Couch & Bloom 1960) and, as a result, there may be a higher prevalence of dollar spot during dry seasons. A heavy thatch layer contributes to low soil moisture because it inhibits water penetration into soil. Irrigation to maintain soil moisture content above 75 % of field capacity will reduce disease severity (Couch 1995). Insufficient nitrogen (N) fertilizer will predispose foliage to dollar spot infection and disease severity (Endo 1966, Freeman 1969, Watkins & Wit 1995). *Sclerotinia homoeocarpa* requires an available food base for growth and appressorium formation; thus, plants that are N-stressed are more likely to develop senescent foliage that is more susceptible to infection than plants receiving adequate N (Endo 1966). Applications of N can be used to suppress dollar spot (Cook et al. 1964, Markland et al. 1969) because N-supplemented grass grows more quickly, therefore, more frequent mowing removes necrotic tissue during times when microclimate is less favorable for growth of the fungus (Couch 1995). Collection and disposal of clippings removes secondary inoculum and may result in less disease. Markland et al. (1969) proposed that vigorous plant growth reduced disease severity caused by *S. homoeocarpa*. Subsequently, Landschoot and McNitt (1997) demonstrated that disease suppression was positively correlated with dark green turfgrass, indicating that as N availability increased, disease severity decreased. Applications of readily available N, in the form of ammonium nitrate and sewage sludge, effectively

suppressed disease (Cook et al. 1964, Markland et al. 1969). Activated sewage sludge provided the greatest suppression, and also caused an increase in concentrations of iron, copper, and zinc in plant foliage. Markland et al. (1969) suggested that levels of these heavy metals in plant tissue were large enough to inhibit a fungal pathogen. The key to managing disease through N fertilization is moderation (Fenstermacher 1980), because insufficient fertilization will lead to dollar spot-susceptible turfgrass and excessive fertilization can promote other diseases.

1.2.5.2 Fungicides and Fungicide Resistance

During the last 40 years, fungicides have been the most successful tool for managing dollar spot. Often, numerous applications of fungicides are required to maintain disease-free turfgrass throughout a growing season and, as a result, resistance of *S. homoeocarpa* to fungicides has posed an ongoing challenge to the turfgrass industry. Resistance to heavy metal-based fungicides (Cole et al. 1968, Massie et al. 1968); benzimidazoles (Cole et al. 1974, Detweiler et al. 1983, Goldberg & Cole 1973, Warren et al. 1974); anilazine (Nicholson et al. 1971); dicarboximides (Detweiler et al. 1983); and demethylation inhibitors (DMIs) (Golembiewski et al. 1995) were reported from various regions throughout the United States. To compound this problem, strains of *S. homoeocarpa* that are resistant to one fungicide are often cross-resistant to other fungicides that share the same mechanism of action (Cole et al. 1974, Golembiewski et al. 1995, Warren et al. 1974).

From the 1940s to the mid-1960s, cadmium-based fungicides were used for dollar spot management with excellent results (Fenstermacher 1980). By the late 1960s, however, several cases of cadmium tolerance were reported, and tolerance of *S. homoeocarpa* to

mercuric fungicides also became widespread (Cole et al. 1968, Fenstermacher 1980, Massie et al. 1968, Smith et al. 1989). Cadmium-tolerant strains became predominant and remained after cadmium fungicide applications ceased, indicating that they possessed increased fitness (Warren et al. 1977). Once resistance developed, these fungicides continued to be ineffective against the pathogen, even if temporarily removed from the management program (Warren et al. 1977).

When first introduced in the late 1960s, the benzimidazole-type systemic fungicides provided excellent disease control at low dosages. However, resistant biotypes of the pathogen emerged relatively quickly (Fenstermacher 1980, Smith et al. 1989, Vargas 1994). Warren et al. (1977) demonstrated that the benzimidazole-resistant strains collected from the eastern and midwestern United States were less fit in fungal populations of the pathogen, and that populations would revert to the wild-type after discontinuation of benzimidazole application. In contrast, Vargas (1994) stated that tolerant strains were more fit and dominated wild-type populations long after application ceased. Increases in disease severity resulted when creeping bentgrass colonized with a strain of benzimidazole-resistant *S. homoeocarpa* was treated with this group of fungicides (Couch & Smith 1991).

Resistance of *S. homoeocarpa* to DMI fungicides was reported 11 years after the introduction of this family of fungicides for management of dollar spot in the United States (Doney & Vincelli 1993, Golembiewski et al. 1995, Vargas et al. 1992). These fungicides were registered for use on turfgrass in Canada in 1994; however, one isolate of *S. homoeocarpa* found in Ontario had reduced DMI sensitivity prior to registration (Hsiang et al. 1997). Resistance probably became a problem in Canada because of transportation of isolates across the border, or from illegal use of the fungicides (Hsiang et al. 1997). Hsiang

et al. (1998) found that there was a slight fitness cost for resistant isolates, which supports the recommendation that DMI fungicides not be used when the incidence of dollar spot is severe, or in consecutive years (Golembiewski et al. 1995, Vargas et al. 1992). This strategy reduces selection pressure on wild-type populations of the pathogen, allowing them to remain dominant in the population.

The occurrence of pesticide-resistance among populations of *S. homoeocarpa* is compounded by the development of multiple-resistance to fungicides in various families. Benzimidazole-resistant strains with resistance to cadmium (Warren et al. 1974, Warren et al. 1977) or dicarboximides (Detweiler et al. 1983) have been reported. Some DMI-resistant strains were found that were multi-resistant to benzimidazole and dicarboximide fungicides (Golembiewski et al. 1995, Vargas et al. 1992). Although resistance was more likely to occur in isolates that already possessed resistance to another fungicide group, pathogen strains that were double- or multi-resistant to dicarboximides did not persist in turfgrass once use of that group of fungicides was discontinued (Vargas et al. 1992, Vargas 1994). However, strains with benzimidazole and DMI resistance were persistent (Vargas 1994).

Fungicides are applied to turfgrass at regular intervals, but with an accurate disease forecasting model, fungicides could be applied only when weather conditions are favorable for fungal activity, thereby reducing selection pressure for resistant strains of the pathogen. Recommendations for chemical application have often emphasized the use of tank mixes of fungicides with different modes of action (Urech 1988). This strategy is believed to provide effective disease control while reducing the risk of developing fungicide-resistant pathogen biotypes (Couch 1995). Sanders et al. (1985) reported that half-rate (14.2 g product per 93 m²), two component mixtures of benzimidazoles, dicarboximides, and sterol biosynthesis

inhibitors provided excellent suppression of dollar spot on creeping bentgrass. Reduced-rate fungicide applications may also reduce selection pressure for the development of resistance. Fungicides can also have direct and indirect effects on nontarget microorganisms (Smiley & Craven 1979) and these effects may influence the development of diseases such as dollar spot. Significant increases in disease severity following the termination of fungicide treatments may be related to such effects (Melzer & Boland 1998).

1.2.5.3 Biological Control

Several biological control strategies for management of dollar spot have been investigated and, in general, these strategies fall into two approaches. One approach is through the application of nutrients and organic amendments to stimulate naturally-occurring populations of microorganisms in the phyllosphere. The second approach is the more typical use of inundative applications to turfgrass of specific bacteria and fungi known to suppress disease. Most biological control strategies evaluated to date were less effective than fungicides, but several merit further investigation.

1.2.5.3.1. Nutrients and Organic Amendments

Several studies compared the influence of organic amendments, such as commercially available organic fertilizers, composts, and sludges, on suppression of dollar spot (Hoyland & Landschoot 1993, Landschoot & McNitt 1997, Liu et al. 1995, Nelson & Craft 1991a). Several composts suppressed dollar spot severity but there was substantial variation in their efficacy. In one study, the organic fertilizer Ringer Compost Plus (Ringer CP) (Ringer Corporation, Minneapolis, MN) provided substantially greater disease suppression than treatment with iprodione (Nelson & Craft 1991a). Effective disease

suppression was evident up to one month following application of Ringer CP and the organic fertilizer Ringer Greens Restore (Nelson & Craft 1992). However, one month after a second application, propiconazole was more effective than the organic fertilizers. Some disease suppression resulted from applications of sludge composts and of Sustane turkey litter compost (Sustane Corporation, Cannon Falls, MN) (Nelson & Craft 1992, Soika & Sanders 1991). In contrast, Hoyland and Landschoot (1993) found little suppression of disease by Ringer CP, with effects comparable to applications of a high rate of Sustane. The organic fertilizers, Harmony/KLM and Ringer Commercial Greens Super, and the synthetic fertilizers, urea and Nitroform, provided similar levels of disease suppression (Hoyland & Landschoot 1993). Landschoot and McNitt (1997) demonstrated that organic fertilizers, such as Ringer Commercial Greens Super, Ringer CP, Sustane, Milorganite and Harmony, were not superior in dollar spot suppression when compared with synthetic N sources such as urea. Further studies are required to define whether reduction in dollar spot is attributed to a microbial effect on the pathogen or increased vigor of the plant in response to N amendments (Landschoot & McNitt 1997).

Despite the efficacy of several of these organic amendments, no information was provided on mechanisms of action. Markland et al. (1969) found no correlations between disease reduction and microbial activity, soil pH, or fertility. Liu et al. (1995), however, established that the ability of selected organic amendments to suppress disease was correlated with increased populations of naturally-occurring microorganisms in turfgrass. For example, treatment of a creeping bentgrass putting green with Ringer Turf Restore and Ringer Greens Super substantially suppressed dollar spot incidence (Liu et al. 1995) and significantly increased populations of soil microorganisms. Plots of creeping bentgrass

treated with Ringer amendments or ammonium nitrate had higher fungal and bacterial populations on grass, thatch and soil than plots treated with other organic amendments or the untreated control. According to Liu et al. (1995), these higher microbial populations resulted from increased plant growth in response to the fertilizer N content, the introduction of microorganisms in the treatments, the stimulation of native microbe populations, or combinations of these factors.

Topdressings of other organic materials, such as autoclaved grain previously colonized by *Fusarium heterosporum* Nees:Fr. (teleomorph: *Gibberella gordonii* Booth) (Goodman & Burpee 1991), wheat-bran (Schumann & Reuter 1993), autoclaved sand-oatmeal and sodium alginate-oatmeal formulations (Zhou & Boland 1998b), suppressed dollar spot when included as experimental controls in evaluations of biological control agents (BCAs). In each of these experiments, the authors reported reduced disease severity in plots treated with organic substrates that did not contain live organisms (e.g., formulation control) in comparison with plots receiving no treatments whatsoever (i.e., experimental control). Results such as these support the suggestion that stimulation of naturally occurring microbial populations by sterile amendments is related to disease suppression. Alternative mechanisms may also contribute to this effect, such as turfgrass growth stimulation by nutrients in organic materials or the presence of antimicrobial metabolites in heat-killed, colonized substrates.

1.2.5.3.2 Microbial Antagonists

Inundative applications of bacteria and fungi known to suppress dollar spot have been investigated, with varying degrees of success. Topdressings prepared from cornmeal-sand mixtures inoculated with strains of *Enterobacter cloacae* (Jordan) Hormaeche and Edwards were used to introduce this bacterial antagonist into creeping bentgrass putting greens

naturally-infested with *S. homoeocarpa* (Nelson & Craft 1991b). In the first year, monthly applications of isolate EcCT-501 were as effective as curative rates of propiconazole in reducing disease severity. However, the following year, the same isolate provided a lower and less persistent suppression of disease. Another isolate provided variable disease suppression in both years, but was the only treatment to provide significant suppression on the second rating date in the second year. The mechanisms of action responsible for disease suppression were not determined, but this test was performed on 60-year-old swards believed to possess a diverse and well-established microflora that restricted the activity of *E. cloacae*. The authors suggested that this antagonist could be more effective on newer putting greens with a less diverse microflora, as competition would be reduced between the naturally-occurring and artificially-introduced microbial populations.

Topdressings of wheat-bran fortified with a *Streptomyces* sp. were evaluated for their ability to suppress dollar spot on a creeping bentgrass putting green, but the results were inconclusive (Schumann & Reuter 1993). There was no correlation between total actinomycete population and application of sterile bran or *Streptomyces*-infested bran. Either the application method did not increase the isolate population, or the method of sampling turfgrass and soil cores did not provide an accurate assessment of the populations present.

Several microorganisms have been identified that are capable of producing antifungal compounds that suppress development of *S. homoeocarpa*. Strains of *Pseudomonas fluorescens* Migula and one strain of *P. lindbergii* (ATCC 31099) suppressed dollar spot on Kentucky bluegrass (*Poa pratensis* L.) under controlled conditions (Hodges et al. 1994, Rodriguez & Pfender 1997). Antagonism to *S. homoeocarpa* in creeping bentgrass, bluegrass turfgrass and grass clippings by *P. fluorescens* was associated with pyrrolnitrin,

an antibiotic produced by the bacterium (Rodriguez & Pfender 1997). In a two-year field study, *F. heterosporum* suppressed dollar spot symptoms by up to 93 % (Goodman & Burpee 1991). Treated plots continued to show residual effects of the BCA treatments into the following summer. Disease suppression by the BCA topdressings was attributed to inhibition of pathogen growth by antibiosis. This view was supported by topdressing with heat-killed *F. heterosporum*, which provided disease suppression comparable with that of topdressings that were not heat-killed. In addition, in vitro tests confirmed that inhibitory or toxic compounds were released by *F. heterosporum*.

The efficacy of *F. heterosporum* in suppressing dollar spot of turfgrass was compared in a two-year field study with several other fungal antagonists of *Sclerotinia* spp., such as *Alternaria* sp., *Cladosporium* sp., and *Epicoccum* sp. (Boland & Smith 2000). None of these fungi were as effective as the fungicide iprodione, nor were they effective under high disease pressure. *Fusarium heterosporum* was the most effective BCA and suppressed disease by 32 to 49 %. The efficacy of this BCA could be increased by modifications in formulation, concentration of the BCA applied, the frequency of application, and the use of weather-predicted applications.

Other microorganisms that were identified as antagonists of *S. homoeocarpa* include *Rhizoctonia* sp., *Acremonium* sp. (Goodman & Burpee 1991), *Gliocladium virens* Miller, Giddens and Foster (Haygood & Mazur 1990), *Trichoderma hamatum* (Bonord.) Banier strain 382, and *Flavobacterium balustinum* Harrison strain 299r₂ (Grebis et al. 1995). Although applications of these BCAs provided good dollar spot suppression, studies that included a fungicide treatment established that greater control was achieved by the fungicides.

Trichoderma harzianum Rifai is a bio-protectant, that was tested against numerous fungal plant pathogens (Lo et al. 1996, 1997). *Trichoderma harzianum* strain T22 (KRL-AG2) was recently registered with the U.S. Environmental Protection Agency as a biological fungicide for control of fungal diseases of turfgrass, including dollar spot (BioWorks, Inc., Geneva, NY). Marketed as BIO-TREK (Wilbur-Ellis, Fresno, CA), this bio-protectant was the first biological fungicide approved for turfgrass in the United States. When introduced into turfgrass, strain T-22B rapidly colonizes the roots of plants, protecting them through competition and mycoparasitism of the pathogen. However, this product is only effective when applied as a preventative measure. Once a pathogen penetrates the plants and disease symptoms are apparent, an appropriate fungicide must be applied. Efficacy studies of *T. harzianum* have yielded conflicting results (Lo et al. 1996, 1997, Melzer & Boland 1998, Vincelli & Doney 1997, Vincelli et al. 1996); therefore, further research is required to improve the consistency of this BCA.

Hypovirulence refers to the reduced ability of a pathogen to infect, colonize, kill, and reproduce on susceptible host tissue (Elliston 1982). Hypovirulence often is associated with the presence of double-stranded RNA (dsRNA) that is transmissible between infected and healthy isolates of the pathogen. The use of “transmissible hypovirulence” to suppress plant disease was reported for several plant pathogens (Anagnostakis 1982, Elliston 1982, Herr 1995) and transmissible hypovirulence associated with dsRNA also was reported from *S. homoeocarpa* (Zhou & Boland 1997, 1998b). In growth room conditions, three hypovirulent isolates of *S. homoeocarpa* suppressed disease by 51 to 90 % (Zhou & Boland 1998b). In artificially-infested field plots, the hypovirulent isolate suppressed disease by up to 80 % and disease suppression was significant for one year post-inoculation. In naturally-infested field

plots, the hypovirulent isolate suppressed disease by up to 58 % and, in most plots, disease suppression was equivalent to treatment with chlorothalonil. Although further epidemiological and efficacy studies need to be conducted, this study demonstrated that hypovirulent isolates have potential as an effective management tool for the control of dollar spot and other *Sclerotinia*-incited diseases (Zhou & Boland 1998a).

The current status of biological control of turfgrass diseases can best be summarized as an emerging technology. Biological control agents are currently in various stages of research and development, but the registration of BIO-TREK was an encouraging sign that such products are becoming more commercially viable and available. Integration of BCAs into integrated disease management programs will provide the turfgrass industry with alternatives to chemical fungicides, and perhaps an effective tool to prevent the development and spread of fungicide-resistant strains

1.2.5.4 Disease Forecasting

At present, fungicide and biological control treatments for dollar spot are applied to turfgrass at regular intervals. In some crops, diseases are forecasted using disease prediction systems that correlate weather conditions with sporulation, infection or symptom expression (Gillespie & Sutton 1979, Gleason et al. 1995, Madden et al. 1978). In general, environmental variables such as leaf wetness duration, relative humidity, rainfall, temperature, and solar radiation are monitored until conditions are conducive to pathogen development, and a fungicide is then applied. Fungicide use is minimized because treatments are applied only when conditions are favorable for disease. Additionally, forecasting systems would be applicable for weather-timed applications of microbial

antagonists (Boland & Smith 2000, Goodman & Burpee 1991). Turfgrass disease forecasters were designed for anthracnose [*Colletotrichum graminicola* (Ces.) G. W. Wils.] of annual bluegrass (Danneberger et al. 1984); Rhizoctonia blight (*R. solani*) of creeping bentgrass (Schumann et al. 1994); brown patch (*R. solani*) of perennial ryegrass (*Lolium perenne* L.) (Fidanza et al. 1996); and Pythium blight on turfgrass (Nutter et al. 1983).

Disease forecasting systems for dollar spot were proposed by Mills and Rothwell (1982) and Hall (1984). In the Mills and Rothwell (M&R) system, a fungicide application was recommended when maximum air temperature was ≥ 25 °C and maximum relative humidity was ≥ 90 % during any 3 days of a 7-day period. In the Hall system, a fungicide application was recommended after 2 consecutive days of rainfall and daily mean air temperature of ≥ 22 °C, or 3 consecutive days of rainfall and daily mean air temperature of ≥ 15 °C. The accuracy of these two disease forecasters was compared in a two-year study, but both models failed to predict weather-related increases of dollar spot (Burpee & Goultly 1986). The M&R model predicted too many infection periods, and disease suppression resulted more from the high frequency of prediction-based fungicide applications and not the accuracy of the model. The Hall system failed to predict sufficient infection periods, resulting in poor disease control. Boland and Smith (2000) used a preliminary dollar spot forecasting model in the EnviroCaster (NEOGEN Corporation, Lansing, MI) to time applications of biological agents. The model did not predict any infection periods during 1991, despite high levels of disease in all plots.

The M&R and Hall models used easily measured variables to predict disease. The Hall system used rainfall and air temperature data recorded at a weather station located 2.2 km from the research station and did not include irrigation inputs. Neither model accounted

for leaf wetness caused by dew and guttation, both of which are important contributors to water on the leaf surface (Williams et al. 1998). Although humidity and rainfall are factors in dew formation, actual leaf wetness duration is a more accurate indicator of available free water. Today, data are available from commercial weather stations capable of measuring numerous turfgrass microclimate variables. With this new technology in place, disease prediction models can use more microclimate variables to accurately forecast dollar spot increases.

1.3 ELECTRONIC LEAF WETNESS SENSORS

1.3.1 Disease and Leaf Wetness

An important aspect of plant disease management is leaf wetness duration because many pathogens require a film of water on the leaf surface for spores to germinate and for mycelia to infect the host (Sutton et al. 1988, Van der Wal 1978). Disease severity on turfgrass has been associated with leaf wetness, particularly dew duration (Danneberger et al. 1984, Fidanza et al. 1996, Hall 1984, Mills & Rothwell 1982, Nutter et al. 1983, Schumann et al. 1994, Williams et al. 1996). Leaf wetness results from dew, fog, rainfall, and irrigation (Huband & Butler 1984, Monteith 1963, Noffsinger 1965, Van der Wal 1978, Williams et al. 1996) and will persist depending on solar radiation, wind velocity, canopy structure, soil moisture, atmospheric radiation, and temperature (Pedro & Gillespie 1982). While precipitation and irrigation can be monitored by a rain gauge, dew must be monitored by an alternative instrument.

Dew can result from three processes; condensation, distillation, and guttation. Grass

absorbs, reflects and emits radiation, and at night there is a net heat loss. When grass temperature falls below the dew point temperature of the adjacent air, the saturation vapor pressure is reached and water condenses on the leaf surface. Condensation usually occurs on clear nights because there are no clouds to absorb and re-emit radiation back towards the earth's surface. The rate of condensation is also dependant on the leaf's boundary layer. The boundary layer is an unstirred layer of air next to the leaf where temperature, vapor pressure and velocity of air is influenced by only the leaf (Salisbury & Ross 1992). Water molecules must pass through this layer by molecular diffusion because the boundary layer does not mix with the adjacent laminar or turbulent flow layer. A leaf boundary layer is thickest when wind velocity is low, at which time the gradient between leaf temperature and atmospheric temperature produces condensation on the leaf surface. Thus, dew formation generally begins at sunset when turbulent airflow and temperature often decrease. The quantity of dew generally increases until a maximum is reached just before sunrise (Van der Wal 1978, Williams et al. 1998). Distillation is the same as condensation except the atmospheric water is the product of evaporation from moist, warm soil below the foliage. Plant transpiration decreases at night but roots continue to absorb water that is transported to the leaves. Water collects as guttation fluid at hydathodes and cut leaf tips, and does not evaporate because the air is near the saturation vapor pressure.

Turfgrass has a unique surface, its blades of grass provide many foci for droplets of water to form directly at the tips of individual leaves. Not only does the grass offer a large surface area for condensation, but approximately one third of dew on grass is composed of exudates from cut leaf tips and hydathodes (Williams et al. 1998). These exudates are important in disease development because they contains a wide range of compounds that

increase fungal growth, such as vitamins, amino acids, sugars, pectic substances and sugar alcohols (Couch 1995, Williams et al. 1998). Large water droplets may drip into the underlying "canopy" not exposed to wind and solar radiation, thus prolonging leaf wetness.

1.3.2 Measurement of Leaf Wetness

Three modern methods to determine leaf wetness duration include: estimation of leaf wetness duration through the relation of weather parameters (Atzema 1992, Deshpande et al. 1995, Gleason et al. 1994, Huber & Gillespie 1992, Pedro & Gillespie 1982, Severini et al. 1984), beta-ray attenuation wetness meter (Armstrong et al. 1993, Barthakur 1985), and electronic leaf wetness sensors (Fernandes et al. 1991, Giesler et al. 1996, Gillespie & Kidd 1978, Gleason et al. 1998, Smith & Gilpatrick 1980, Weiss & Lukens 1981).

Electronic leaf wetness sensors (LWS) or impedance grids are composed of two non-contacting adjacent metal electrodes independently connected to a datalogger via a two-wire shielded cable. Wires are energized with a momentary potential difference of 2.5 to 5.0 V, and an electrical current flows between electrodes when water bridges the gap. Changes in impedance are recorded by the datalogger: as the sensor becomes wet, the impedance decreases; as the sensor dries, the impedance increases. Based on the change in impedance, the duration and amount of free water on the leaf surface is estimated (Howard & Gillespie 1985).

There are several characteristics required in a leaf wetness sensor. The sensor should have a short time constant (Fritschen & Gay 1979), meaning it quickly responds to changes in free water on the leaf surface. In the case of LWS, the time constant represents the sensor's accuracy, that is, the relation between measured and "true value" (Fritschen & Gay

1979). An accurate sensor will respond simultaneously with dew formation on the foliage. Precision, the variability observed among numerous measurements, is also important (Fritschen & Gay 1979). A sensor that lacks accuracy, but is precise, can still be of value as an environmental instrument because it will give a consistent response that can be adjusted to mimic the true leaf wetness condition. Sensors must be durable to not lose precision or accuracy when repositioned or during extended field use. Sensors must not be hygroscopic, meaning they should only respond to free water and not respond to high relative humidity.

Weiss (1990) provided a comprehensive review of instrumentation available for sensing leaf wetness and described modifications to improve sensor accuracy. Accurate measurement of leaf wetness was possible when sensors were positioned in the canopy at similar angle and height to the leaves of the crop (Gillespie & Kidd 1978). Several sensors were used to estimate average leaf wetness duration within a diverse canopy. Sensors of different shapes, sizes, and surface characteristics were designed to mimic drying properties of fruits or leaves within crops (Sutton et al. 1988). For example, a spherical sensor was used to mimic the apple's large volume to surface area ratio which dictated its unique thermal qualities. A painted, cylindrical LWS placed vertically imitated an onion leaf (Howard & Gillespie 1985). Wire grids on these three-dimensional sensors were formed by wrapping nickel wire around an object shaped like the target plant structure. Green latex paint applied to sensors (Sutton et al. 1988, Gillespie & Kidd 1978) improved response to initial wetness and distribution of water across the surface, and better simulated the leaf radiative properties. Weiss & Lukens (1981) anchored alternating wires on a plastic frame and wove a piece of thin cotton cloth between the wires. As the cloth became saturated with dew it formed a continuum between the wires, thus reducing the impedance. The appropriate

cloth had similar wetting and drying characteristics to the leaf. Some sensors were not designed to mimic the plant surface, instead the wires were attached directly to a plant structure. Sutton et al. (1984) made use of plastic clothes pins that supported two nickel wire electrodes which rested gently against the leaf surface. Occasionally, sensors caused leaf damage or became disconnected from the surface because of wind or rain; therefore, sensors were checked daily to assure proper positioning.

There are two leaf wetness sensors available for turfgrass; the site-specific tall fescue LWS (Giesler et al. 1996) and the Model 237 LWS (Campbell Scientific, Inc., Logan, UT). The tall fescue LWS was a reliable instrument for measuring free water on tall fescue; however, its design does not permit use on closely-mown turfgrass used for golf course greens. The anthracnose and brown patch disease forecasting systems (Danneberger et al. 1984, Fidanza et al. 1996) were developed using data from the Model 237 LWS. There was no evaluation or modification of the Model 237 LWS to confirm its ability to measure leaf wetness on turfgrass and, as a result, its suitability for use in a disease forecasting system. A putting green LWS must respond to free water that accumulates as a result of guttation, distillation and condensation but should not restrict the drying effects of solar radiation and wind. The Model 237 impedance grid was printed on only one side of the sensor, thus, it could monitor condensation but could not respond to guttation fluid exuded from the leaf tips. Also, the sensor could potentially interfere with radiative heating, cooling, and evaporation because it covered the turfgrass. The Model 237 LWS was an unpainted impedance grid, and studies have shown that a coating of green latex paint improves the sensor response time (Gillespie & Kidd 1978).

1.4 CONCLUSIONS

In summary, dollar spot is one of the most common and destructive diseases of warm and cool season turfgrasses in North America. Symptoms of disease include 5- to 7-cm diameter spots of straw-colored turfgrass on closely mown putting greens and tees, or large coalescing spots on lawns and turfgrass maintained at higher mowing heights. Although the pathogen is commonly referred to as *S. homoeocarpa*, the taxonomic status of this fungus requires clarification. The epidemiology of disease is associated with warm temperatures and heavy, prolonged periods of dew in summer and early fall, but an improved understanding of environmental and physical factors contributing to dollar spot will contribute to better disease management strategies.

Currently, disease is primarily controlled through fungicides and by cultural practices. With the onset of fungicide-resistance in populations of the pathogens and heightened public awareness concerning the use of pesticides, there is increasing need for alternative pest management practices and technologies. Biological control of dollar spot has shown promising results in laboratory, greenhouse and field evaluations. However, biological control is still largely an emerging technology, despite the recent registration of a strain of *T. harzianum* in the United States for managing several turfgrass diseases. Turfgrass amendments, containing microorganisms and N, have shown potential for management of dollar spot; however, the contribution of microbes or N, or both, to disease suppression requires clarification.

Previous disease prediction models did not accurately predict the need for fungicide applications. Therefore, a more accurate disease prediction model is needed. The

development of an accurate dollar spot forecasting model would contribute to reduced fungicide resistance and costs, and perhaps increase the effectiveness of applied BCAs. Information about the suitability of LWS for use on turfgrass is required before these sensors can be relied upon in disease forecasting systems. Further research on the biology, epidemiology, taxonomy, and potential microbial antagonists of *S. homoeocarpa* is required to achieve cost-effective and environmentally-sound management of dollar spot.

1.5 RESEARCH OBJECTIVES

The objectives of the research presented in the following dissertation are listed below.

1. To identify the temperature and leaf wetness conditions required for infection of turfgrass by *Sclerotinia homoeocarpa* in controlled environmental conditions.
2. To monitor natural epidemics of dollar spot in turfgrass and correlate disease increase with environmental conditions.
3. To combine the results from controlled environment and field studies into a preliminary disease prediction model.
4. To characterize the symptom development and epidemiology of dollar spot.
5. To evaluate existing sensors used to monitor leaf wetness on turfgrass.
6. To modify these sensors to improve accuracy and suitability for use in the turfgrass industry.

2. DEVELOPMENT AND EVALUATION OF IMPEDANCE LEAF WETNESS SENSORS FOR MEASUREMENT OF DEW DURATION ON CREEPING BENTGRASS GREENS

2.1 ABSTRACT

Leaf wetness duration (LWD) is a variable measured by turfgrass disease forecasting models (Danneberger et al. 1984, Fidanza et al. 1996). In a two-year study, ten impedance leaf wetness sensors were evaluated for accuracy and precision of monitoring dew onset and dissipation on creeping bentgrass. Sensors (S) S1 and S2 were Campbell Scientific, Inc. (Model 237) sensors positioned sensing-side up on a creeping bentgrass green (S1) and Kentucky bluegrass sward (S2). S3 through S6 were one-half of Model 237 sensors coated with green latex paint. S3, S4 and S5 were not evaluated because they malfunctioned >25 % of the time. S6 was positioned facing north at 45° to the horizontal at a height of 1.5 m. S7 was constructed from plastic tubing (1 cm diameter) wound with two wires, coated with green latex paint, and positioned horizontally on the green. S8 and S9 were made of plexiglass frames (10 × 4 × 1.3 cm and 10 × 4 × 0.6 cm, respectively) with two wires stretched across the long axis of each frame, and string wound around the wires to wick dew droplets. S10 was constructed from two plastic golf tees joined by two wires and positioned flush with the creeping bentgrass green. The onset and dissipation of dew was determined by tactile observations of creeping bentgrass wetness and compared to sensor output. Least Squares Mean (LSM) for the response time of each sensor defined accuracy while standard deviation (SD) indicated precision. The absolute value of LSM plus the SD was used to rank sensors in order of ability to monitor dew onset and dissipation. For dew onset, S1 was most

accurate with LSM of 11.7 minutes (min) and S6 was most precise with SD of 44.7 min. S2 responded within 65.7 min of actual dew onset. For dew dissipation, S2 had the smallest LSM and SD of -0.9 min and 39.7 min, respectively, thus S2 responded within 40.6 min of actual dew dissipation. S2 estimated, on average, within 1.7 h of the actual entire dew duration. Therefore, the Model 237 sensor, when placed within a Kentucky bluegrass canopy maintained at fairway height, was the optimum sensor evaluated for monitoring LWD on creeping bentgrass greens.

2.2 INTRODUCTION

An important aspect of plant disease management is leaf wetness duration (LWD) because many pathogens require a film of water on the leaf surface for spores to germinate and for mycelia to infect the host (Sutton et al. 1988, Van der Wal 1978). Leaf wetness results from dew, fog, rainfall, and irrigation (Huband & Butler 1984, Monteith 1963, Noffsinger 1965, Van der Wal 1978, Williams et al. 1996) and will persist depending on solar radiation, wind velocity, canopy structure, soil moisture, atmospheric radiation, and temperature (T) (Pedro & Gillespie 1982). Dew can result from three processes; condensation, distillation, and guttation. While precipitation and irrigation can be monitored by a rain gauge, dew must be monitored by an alternative instrument.

Three modern methods to determine LWD include: estimation of LWD through the relation of weather parameters (Atzema 1992, Deshpande et al. 1995, Gleason et al. 1994, Huber & Gillespie 1992, Pedro & Gillespie 1982, Severini et al. 1984), beta-ray attenuation wetness meter (Armstrong et al. 1993, Barthakur 1985), and electronic leaf wetness sensors (Fernandes et al. 1991, Giesler et al. 1996, Gillespie & Kidd 1978, Gleason et al. 1998, Smith & Gilpatrick 1980, Weiss & Lukens 1981). An electronic leaf wetness sensor (LWS) or impedance grid, the topic of the present study, is composed of two non-contacting adjacent metal electrodes independently connected to a datalogger via a two-wire shielded cable. Wires are energized with a momentary potential difference of 2.5 to 5.0 V, and an electrical current flows between electrodes when water bridges the gap. Changes in impedance are recorded by the datalogger: as the sensor becomes wet, the impedance decreases; as the sensor dries, the impedance increases. Based on the change in impedance,

the duration and amount of free water on the leaf surface is estimated (Howard & Gillespie 1985).

There are several characteristics required in a leaf wetness sensor. The sensor should have a short time-constant (Fritschen & Gay 1979), meaning it quickly responds to changes in free water on the leaf surface. In the case of a LWS, the time constant represents the sensor's accuracy, that is, the closeness of the measured to the "true value" (Fritschen & Gay 1979). An accurate sensor will respond simultaneously with dew formation on the foliage. Precision, the variability observed among numerous measurements, is also important (Fritschen & Gay 1979). A sensor that lacks accuracy, but is precise, may still be of value as an environmental instrument because it will give a consistent response that can be adjusted to mimic the true leaf wetness condition. Sensors must be durable so they do not lose precision or accuracy when repositioned or during extended field use. Sensors must not be hygroscopic, meaning they should only respond to free water and not respond to atmospheric humidity.

Disease severity on turfgrass is associated with LWD, particularly dew duration (Danneberger et al. 1984, Fidanza et al. 1996, Nutter et al. 1983, Schumann et al. 1994, Williams et al. 1996). Turfgrass has a unique surface, its blades of grass provide many foci for droplets of water to form directly at the tips of individual plants. Not only does the grass offer a large surface area for condensation, but approximately one third of dew on grass is composed of exudates from cut leaf tips and hydathodes (Williams et al. 1998). Large water droplets may drip into the underlying "canopy" not exposed to wind and solar radiation, thus prolonging leaf wetness.

In other pathosystems, T and LWD within the microclimate of the host affect

pathogen development and disease severity (Van der Wal 1978, Sutton et al. 1988). Disease forecasting systems for a variety of crops were designed using knowledge of specific environmental factors that influence disease development. Forecasting systems for turfgrass diseases such as brown patch on perennial ryegrass (*Lolium perenne* L.) (Fidanza et al. 1996) and anthracnose on annual bluegrass [*Poa annua* var. *reptans* (Hauskins Timm.)] (Danneberger et al. 1984) used LWD and T to predict disease. Nutter et al. (1983) and Schumann et al. (1994) used T and relative humidity variables in disease forecasters for Rhizoctonia blight (*R. solani*) on creeping bentgrass (*Agrostis palustris* Huds.) and pythium blight (*Pythium* spp.), respectively. Dollar spot on creeping bentgrass was predicted by Hall (1984) and Mills and Rothwell (1982) using T, rainfall and relative humidity. Relative humidity (RH) exceeding 90 % has been used as an estimator of leaf wetness (Sutton et al. 1984).

There are two leaf wetness sensors available for turfgrass; the site-specific tall fescue LWS (Giesler et al. 1996) and the Model 237 LWS (Campbell Scientific, Inc., Logan, UT). The tall fescue sensor is a reliable instrument for measuring free water on tall fescue; however, its design does not permit use on closely-mown turfgrass used for golf course greens. The anthracnose and brown patch systems (Danneberger et al. 1984, Fidanza et al. 1996) were developed using data from the Model 237 LWS. There was no evaluation or modification of the Model 237 LWS to confirm its ability to measure leaf wetness on turfgrass and, as a result, its suitability for use in a disease forecasting system. A putting green LWS must respond to free water that accumulates as a result of guttation, distillation and condensation but should not restrict grass to the drying effects of solar radiation and wind. The Model 237 impedance grid was printed on only one side of the sensor, thus, it

could monitor condensation but it could not respond to guttation fluid exuded from the leaf tips. Also, the sensor interfered with radiative heating, cooling, and evaporation because it covered the turfgrass. The Model 237 LWS was an unpainted impedance grid, and studies have shown that a coating of green latex paint improves the sensor response time (Gillespie & Kidd 1978).

The objective of this study was to test the accuracy, precision, and durability of ten impedance LWSs for use on closely-mown creeping bentgrass greens. Sensors were evaluated for their ability to respond to dew onset and dissipation but not the quantity of dew accumulation, because LWD is believed to be more important than dew quantity (Burrage 1972, Danneberger et al. 1984, Fidanza et al. 1996). Information gathered about turfgrass leaf wetness sensors will be used in a preliminary disease forecasting system for dollar spot in southern Ontario.

2.3 MATERIALS AND METHODS

2.3.1 Field Site

All trials were conducted on the native sand green and pathology green at the Guelph Turfgrass Institute, Guelph, ON, Canada, 43°32.97 N, 80°12.90 W. During the 1996 season, both the native sand and pathology greens were used for testing; however, only the native sand green was used during 1997. Both greens were maintained with similar irrigation, fertility and mowing regimes. Greens differed in microclimate as a result of tree cover, the pathology green having substantially more shade during the morning that influenced dew dissipation. This dissimilarity in microclimate was of no consequence because sensor response and tactile observations of dew were independently recorded for each green.

Sensors were placed on creeping bentgrass (cultivar 'Penncross') greens maintained at 6.5 mm mowing height or in the adjacent Kentucky bluegrass (*Poa pratensis* L.) maintained at 5.0 cm mowing height.

2.3.2 Sensor Design and Positioning on Turfgrass

Electronic LWSs were constructed with two non-contacting adjacent metal wires independently connected to a datalogger via a two-wire shielded cable (Figure 2.1). Wires were energized with a momentary 2.5 or 5.0 V potential difference and current flowed between wires when water droplets closed the circuit. An alternating current was used to avoid polarization of water and to minimize heating of the sensor (Weiss & Lukens 1981). A Campbell Scientific 21X datalogger (Campbell Scientific, Inc., Logan, UT), programmed with an AC half-bridge command, recorded the change in impedance used to estimate duration and amount of liquid water on the leaf surface. The Model 237 LWS was a printed circuit on one side of a hard epoxy-fibreglass board measuring 6 × 8 cm (Sensors 1 and 2). Sensor 1 was positioned horizontally on the creeping bentgrass with the sensing side up (Figure 2.2). Sensor 2 was positioned facing north approximately 30° to the horizontal, with the sensing side up, in the canopy of 5-cm-high Kentucky bluegrass located 5 m north of the test area (Figure 2.3). Although this sensor was placed in Kentucky bluegrass, the response of sensor 2 was correlated to dew duration on the creeping bentgrass green. Sensors 3, 4, 5, and 6 were made from Model 237 LWSs cut in half (3 × 8 cm) and painted with green latex paint (Pittsburgh P-88-6) (Gillespie & Kidd 1978, Sutton et al. 1988) to improve their response to initial wetness by distributing water across the surface and simulating the thermal properties of turfgrass (Figure 2.4). Sensors 3 and 4 were positioned on creeping bentgrass,

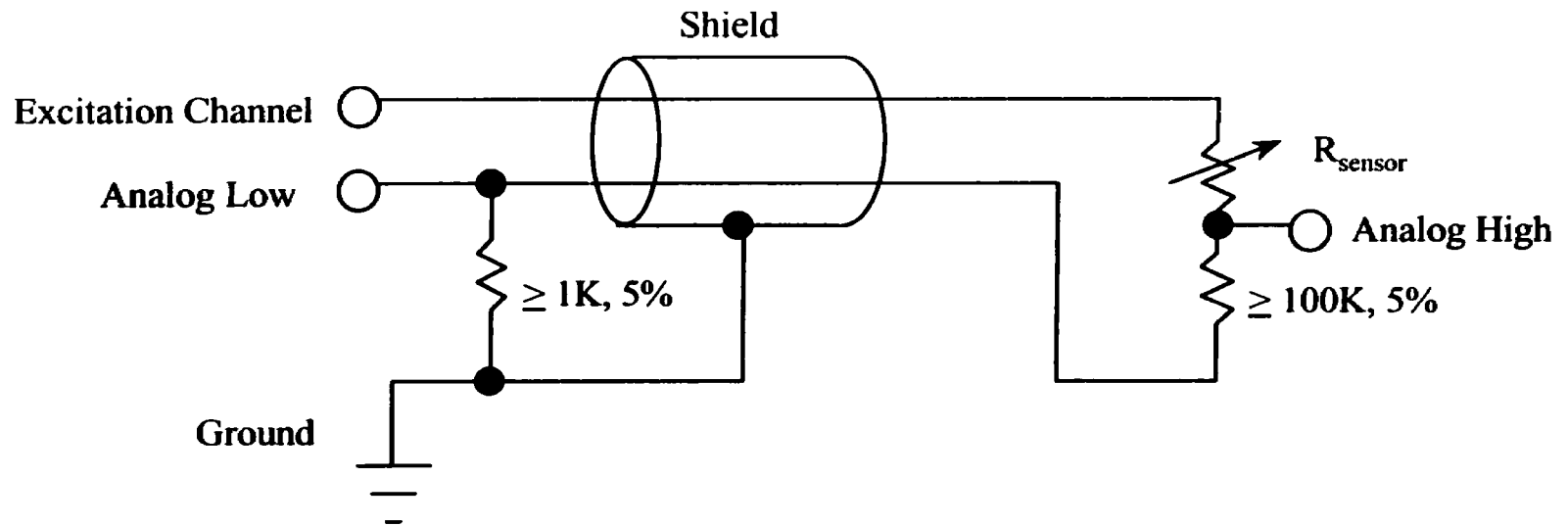


Figure 2.1 General Schematic for impedance leaf wetness sensors.

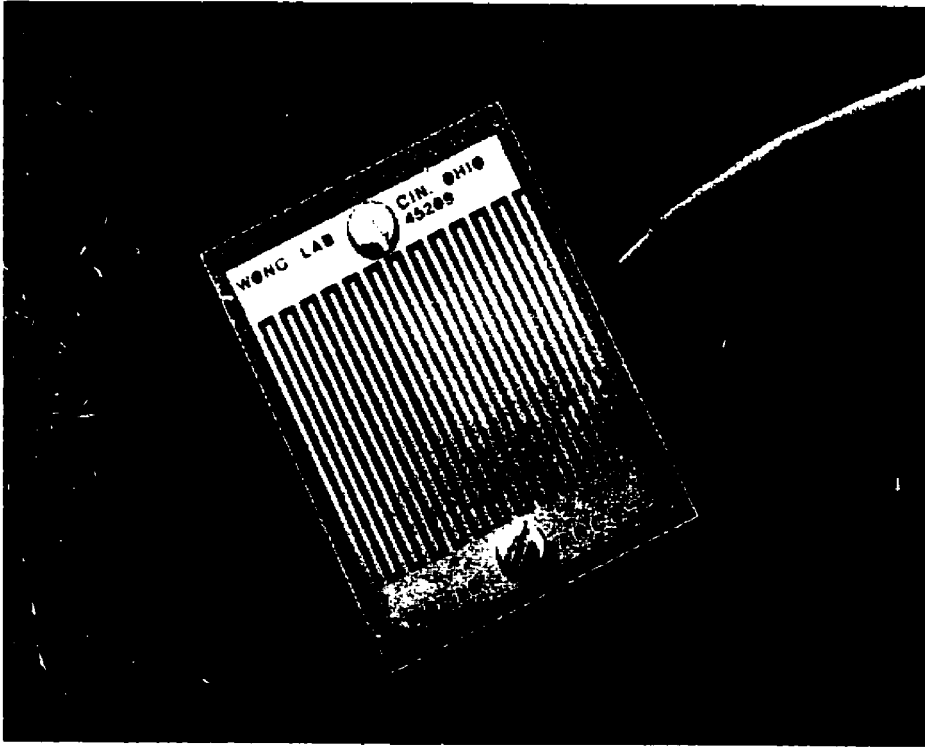


Figure 2.2 Campbell Scientific, Inc. Model 237 leaf wetness sensor (Sensor 1) positioned on a creeping bentgrass green.

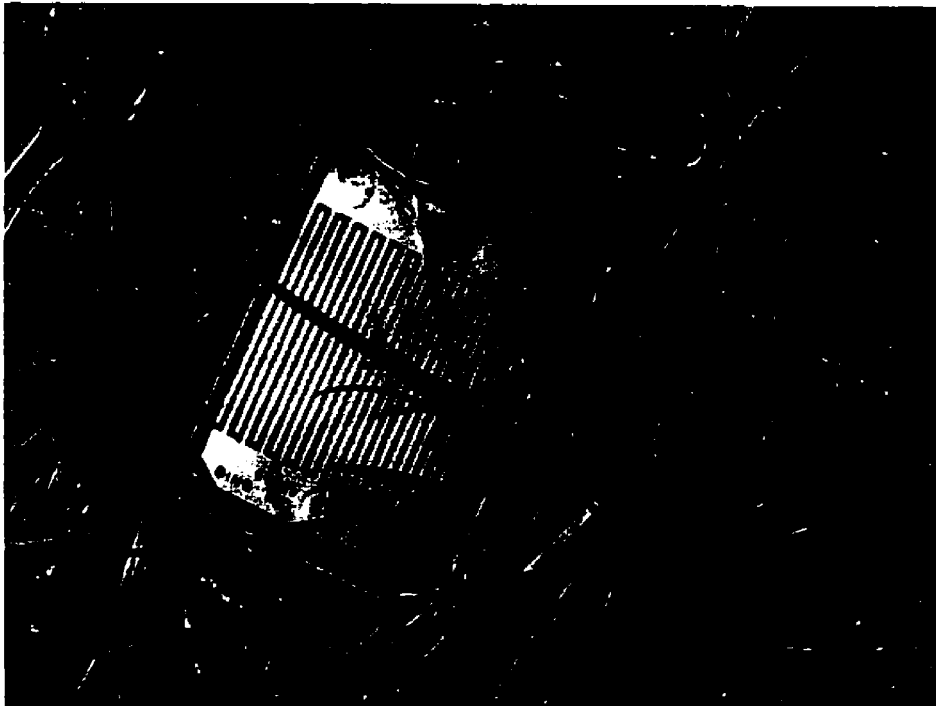


Figure 2.3 Campbell Scientific, Inc. Model 237 leaf wetness sensor (Sensor 2) positioned on Kentucky bluegrass.

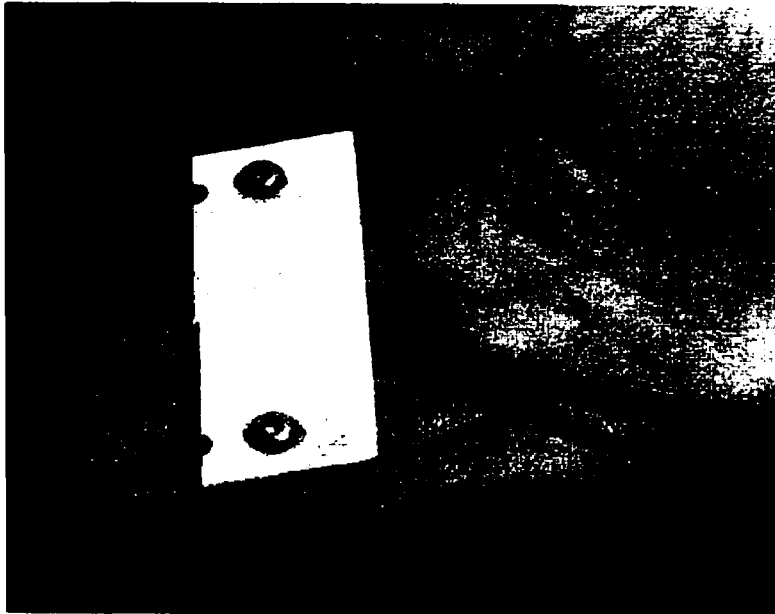


Figure 2.4 Modified and painted Campbell Scientific, Inc. Model 237 leaf wetness sensor (Sensor 6) positioned 1.5 m above a creeping bentgrass green and angled to the north at 45° to the horizontal.

with the sensing side up, at 0° and 45° to the horizontal facing N, respectively. Sensors 5 and 6 were tipped about their short axis toward the north at 45° to the horizontal at 30 cm and 150 cm above the turfgrass surface, respectively. Sensor 7 was made from two nickel wires wound parallel to each other around a 9-cm-long plexiglass tube (1 cm diameter) then painted (Figure 2.5). Sensor 7 was placed horizontally on creeping bentgrass and anchored to maintain contact with leaf tissue. Sensors 8 and 9 (Figure 2.6 and 2.7) were each comprised of a Plexiglass frame (10×4 cm) with two wires (nickel and stainless steel, respectively) stretched across the long axis of the frame. A piece of nylon string was woven in a zig-zag fashion around the wires to act as a wick for dew droplets. The thinner frame thickness of sensor 9 (0.63 cm) compared to sensor 8 (1.25 cm) allowed the wires to have more contact with the creeping bentgrass surface. Sensor 10 was made from two stainless steel wires stretched between two golf tees with string woven through the wires (Figure 2.8). The golf tees were pushed into the ground until the tops of the tees and wires were flush with the creeping bentgrass surface. Sensors 8, 9, and 10 were designed to function on the creeping bentgrass surface without influencing the microclimate. Also, these sensors could measure the water that originated above and below the instrument, a characteristic not achieved with the one-sided impedance grids (sensors 1 to 6).

2.3.3 Calibration

Each sensor was calibrated at the beginning of 1996 and 1997 field seasons, and were periodically recalibrated and checked for hygroscopy. To calibrate, each sensor was placed on the creeping bentgrass green and the voltage received was recorded when the sensor was dry (V_D) and when the sensor was misted with deionized water (V_W). The multiplier and

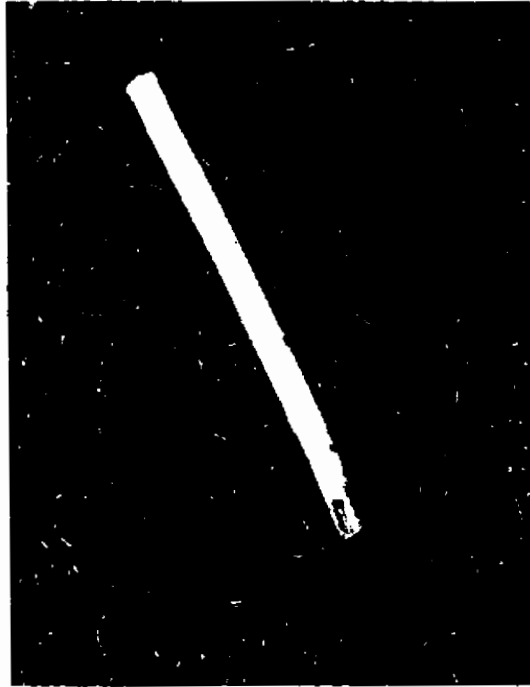


Figure 2.5 Cylindrical, painted leaf wetness sensor (Sensor 7) positioned on a creeping bentgrass green.

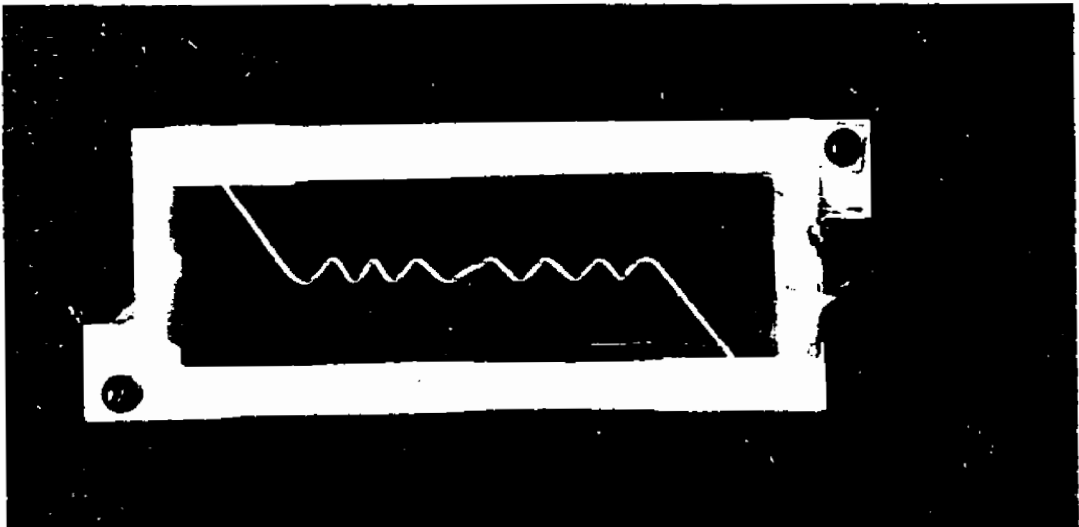


Figure 2.6 Stainless steel wire leaf wetness sensor with frame depth of 1.25 cm (Sensor 8) positioned on a creeping bentgrass green.

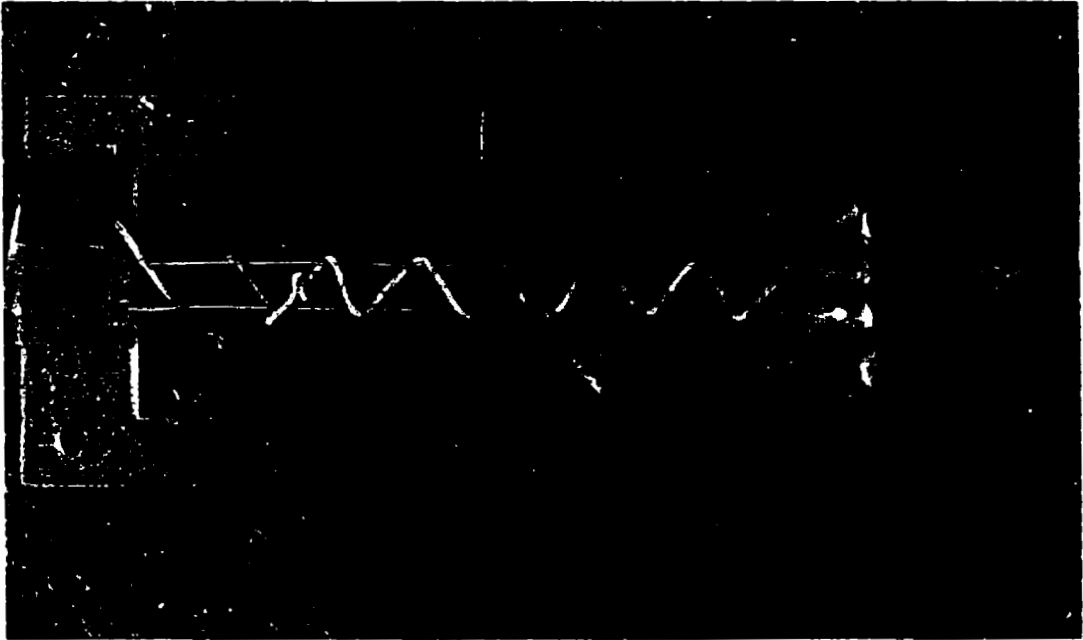


Figure 2.7 Stainless steel wire leaf wetness sensor with frame depth of 0.63 cm (Sensor 9) positioned on a creeping bentgrass green.



Figure 2.8 Golf tee and stainless steel wire leaf wetness sensor (Sensor 10) positioned on a creeping bentgrass green.

offset values used in the datalogger program were calculated using the following formulae (Campbell Scientific (Canada) Corporation 1996):

$$\text{Multiplier} = 100 \bullet [1 / (V_w - V_D)] \quad (2.1)$$

$$\text{Offset} = \text{Multiplier} \bullet V_D \quad (2.2)$$

The datalogger program converted voltage received from each sensor into a percent wetness value, 0 % indicated a dry sensor while 100 % indicated a wet sensor. Each sensor was tested for hygroscopy by sealing it in a plastic bag that contained a beaker of cold water. Condensation on the sensor was not possible since the sensor was warmer than adjacent water; therefore, a voltage increase registered if the sensor was responding to water vapor. Hygroscopic sensors were washed in sodium dodecyl sulphate (10 %) to remove the contaminants responsible for hygroscopy, then were retested to confirm they were no longer responding to atmospheric vapor.

2.3.4 Assessment of Dew Onset and Dissipation

Leaf wetness sensors were evaluated June to September during 1996 and 1997. Sensors were positioned on creeping bentgrass or Kentucky bluegrass after the greens were mowed and dew had dried (at approximately 15:00 h). Irrigation was not applied during the 24 h experimental period. Data were discarded if rain started the leaf wetness event but data were used if rain occurred after dew onset and before dew dissipation. Sensor output was sampled every minute and then averaged over ten minutes.

Observations for dew onset began 30 minutes prior to sunset. Grass was touched with the back of the hand (Giesler et al. 1996) at ten minute intervals while sensor readings were being recorded. A positive tactile observation of wet grass was recorded as the time

of dew onset. Once dew had set on the bentgrass green, it did not evaporate until after sunrise, also observed by Giesler et al. (1996) and Williams et al. (1998). The next morning, the creeping bentgrass was visually observed for droplets of water on turfgrass foliage. When droplets were no longer visible, tactile assessment of dew presence commenced. The time of dew dissipation was recorded when creeping bentgrass was completely dry to the touch. Assessment of dew presence was conducted on turfgrass 50 cm west of the LWS to not disturb sensor microclimate.

2.3.5 Statistical Analysis and Sensor Evaluation

Ideally, sensor output would be zero when completely dry, and 100 when completely wet. However, this scale was offset in some cases because certain sensors were difficult to calibrate. Therefore, for each dew observation, a response threshold for each sensor was calculated by adding the average output during a 3 hour dry period (usually between 15:00 h and 18:00 h) with 2.5 % of the maximum output during the wet period. When the sensor output was above or below this threshold the sensor was deemed wet or dry, respectively (Figure 2.9). The response threshold was calculated for each sensor using the following formula:

$$\text{response threshold} = \text{lower limit} + 0.025 \bullet \text{upper limit} \quad (2.3)$$

Giesler et al. (1996) encountered the same difficulty in determining a threshold for dew onset or dissipation on the leaf wetness sensor. They calculated a response threshold by adding three standard deviations to the lower limit. The Giesler et al. (1996) method was explored in this study; however, the Model 237 LWS had standard deviations of zero which rendered this method unusable. Comparisons were made between the actual time of dew onset or dew

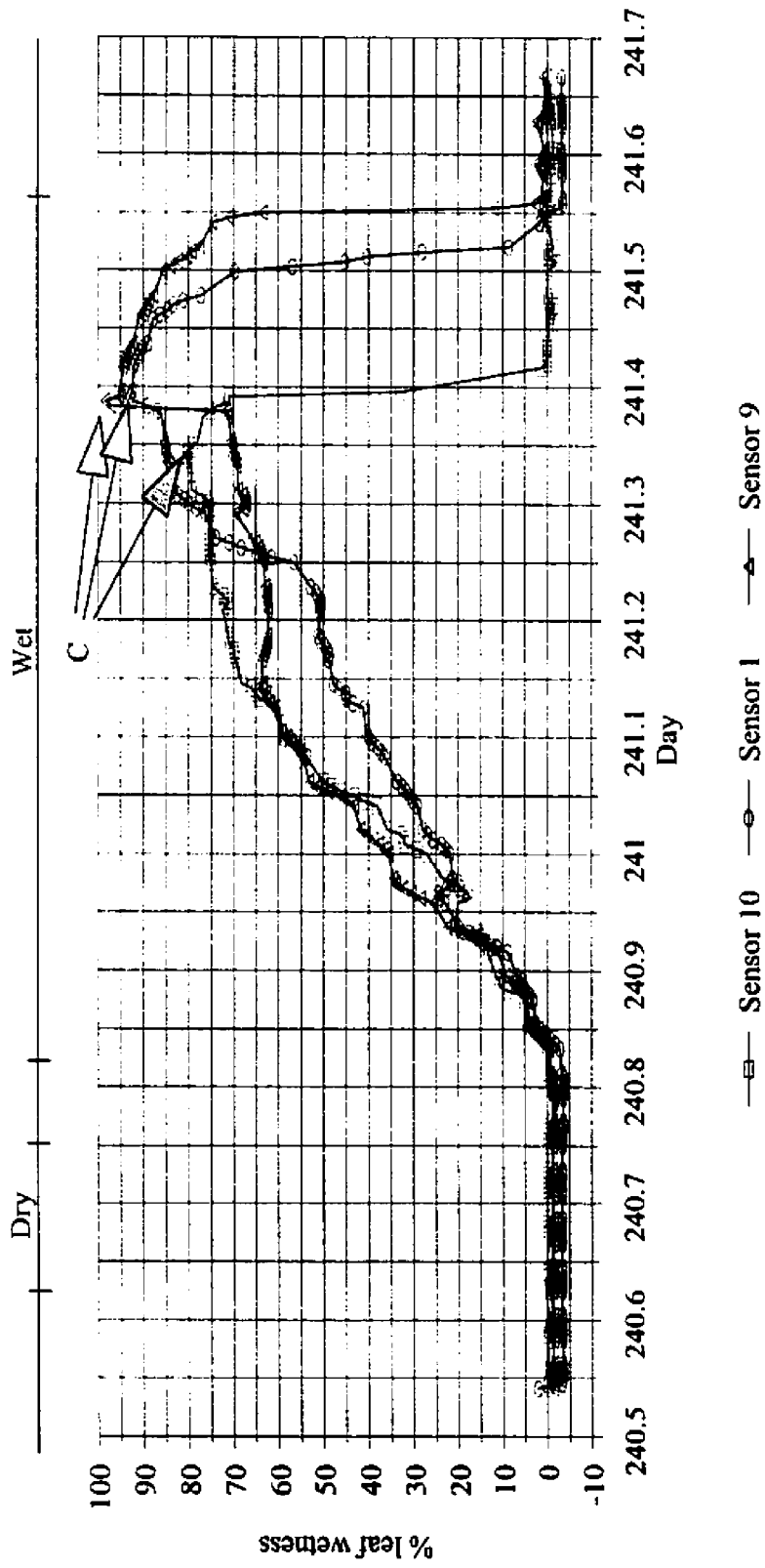


Figure 2.9 Typical sensor output for a dew event. Response threshold for each sensor was calculated by adding the average output during a 3 hour dry period (DRY) with 2.5 % of the maximum output (C) during the wet period (WET). When the sensor output was above or below this threshold the sensor was deemed wet or dry.

dissipation and the time at which each sensor registered a change in water presence. A positive value represented a response time (minutes) that was in advance of actual dew onset or dissipation, while a negative value represented a late response.

Sensor response time was tested using a two factor, incomplete design with year as the first factor and sensor as the second factor. Data were checked for normality and equal variances using the Shapiro-Wilk *W*-test statistic for normality (Royston 1995) and an informal test for examining residual plots that tested for homogeneity (Kuehl 1994). PROC GLM (SAS Institute, Inc. 1996) and Least Squares Means (LSM) Tukey-Kramer multiple comparisons ($\alpha = 0.05$) were used to compare sensors and test the null hypothesis of no difference between the tactile observation and sensor response time. Sensors with LSM closest to zero were considered the most accurate. The sensor with the most consistent response had the smallest standard deviation (SD), thus was considered the most precise instrument. The absolute LSM and SD for each sensor were summed ($|\text{LSM}| + \text{SD}$), then the value was used to assign an overall ranking of the sensors. The sensor with the smallest $|\text{LSM}| + \text{SD}$ was deemed most appropriate for field use. In section 2.4, ranking of sensors for precision and accuracy was listed in descending order beginning with the best sensor. Some sensors did not surpass the threshold value, or fluctuated sporadically throughout the observation period because of incorrect calibration or physical damage. This was classified as a malfunction and, those sensors that malfunctioned >25 % of the time were assumed faulty and were excluded from statistical analysis and subsequent evaluations.

2.4 RESULTS AND DISCUSSION

2.4.1 Sensor Response to Dew Onset

Data were normal with equal variances. Sensors 3, 4, and 5 malfunctioned on >25 % of test days; therefore, they were not considered for accuracy or precision evaluations and were excluded from statistical analysis. Analysis of variance showed a difference in sensor (S) response times (Table 2.1) for $n = 126$ during 1996 and 1997. The instruments with LSM closest to zero sensed dew onset most accurately, and were S1, S9, S2, and S10, respectively (Table 2.2, Figure 2.10). These four sensors did not significantly differ ($\alpha = 0.05$) from the tactile observation. Sensor 1 and S2 tended to register dew before it actually set by 11.7 and 15.3 minutes (mean response time), respectively. Sensor 9 and S10 responded after dew onset by 13.9 and 38.6 minutes (mean response time), respectively. Sensors with the smallest SD were considered the most precise for monitoring dew onset, and were S6, S2, S7, and S1, respectively (Table 2.2, Figure 2.11). Sensors were ranked based on the absolute LSM plus SD ($|LSM| + SD$) (Table 2.2); therefore, the most reliable sensors for measuring dew onset were S2, S1, S9, and S6, respectively. Sensor 2 responded to dew onset within 65.7 minutes of actual dew onset when the LSM and SD were combined. This response time is a resolution sufficient for existing turfgrass disease forecasting models (Danneberger et al. 1984, Fidanza et al. 1996).

Dew accumulation on turfgrass originates from three sources: condensation, distillation and guttation. In this study, sensors were evaluated on response time to dew formation. The most accurate sensor, the Campbell Model 237 LWS positioned in 5-cm Kentucky bluegrass (S2), registered water that accumulated on top of the sensor facing away

Table 2.1 Analysis of variance (ANOVA) for response time (minutes) of sensors (S1, S2, S6 - S10) to dew onset for 25 observations during June to September, 1996 and 1997

Source	DF	SS	MS	F	P-value > F
Corrected total	125	1307232.54			
Year	1	64387.74	64387.74	10.03	0.00196
Sensor	6	522853.36	87142.23	13.57	1.0 E-11
Error	118	757493.27	6419.43		

Table 2.2 Summarized 1996 - 1997 data for accuracy, precision and overall ranking of leaf wetness sensor response to dew onset

Response Time ^a	Sensor Number									
	1	2	3	4	5	6	7	8	9	10
Maximum	200	60	0	540	0	110	260	140	60	120
Minimum	-150	-100	-230	-260	-160	-50	-30	-280	-270	-240
LSM ^b	11.7*	15.3*	-114.7	-116.1	-41.4	52.6	78.7	-122.0	-13.9*	-38.6*
Pr > T Ho: LSM=0 ^c	0.477	0.549	-	-	-	0.041	1.0E-05	4.0E-11	0.452	0.059
SD ^d	80.9	50.4	73.3	263.9	83.3	44.7	77.4	112.3	83.1	99.9
LSM + SD	92.6	65.7	188.0	380.0	124.7	97.3	156.1	234.3	97.0	138.5
Sample Size	24	11	7	8	3	11	22	24	19	16
Malfunctions	1	1	5	4	9	1	2	1	3	5
Ranking ^e	2	1	-	-	-	4	7	5	3	6

a Time in minutes. Positive values represent premature response to dew onset, negative represent delayed response.

b Least Squares Means (LSM) used to assess accuracy.

c Probability that LSM response time is equal to the tactile assessment.

d Standard deviation (SD) used to assess precision.

e Ranked in order of smallest |LSM| + SD value.

***** LSM was not significantly different from tactile assessment ($\alpha = 0.05$).

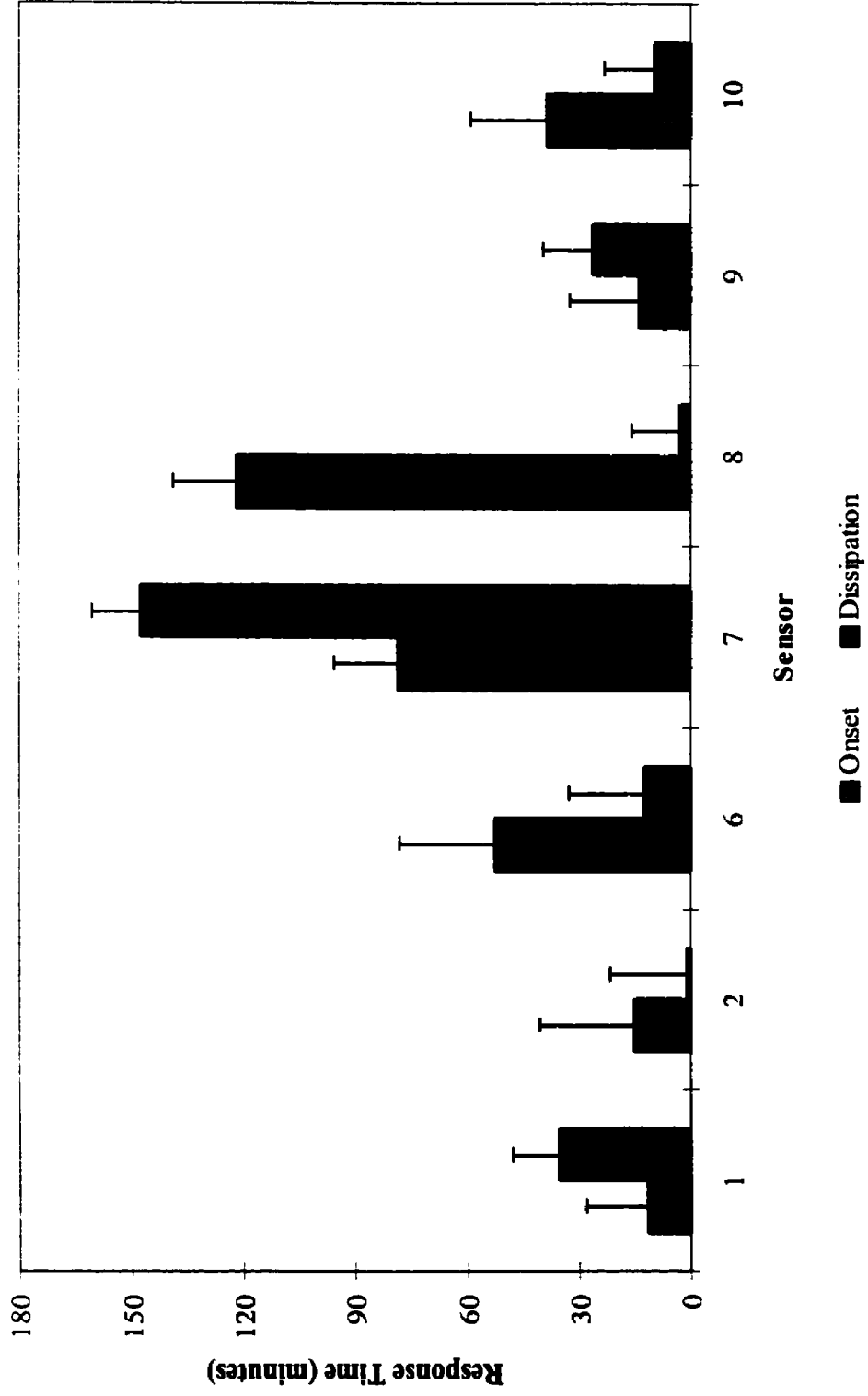


Figure 2.10 Least squared means with standard error bars for sensor response time to dew onset and dissipation.

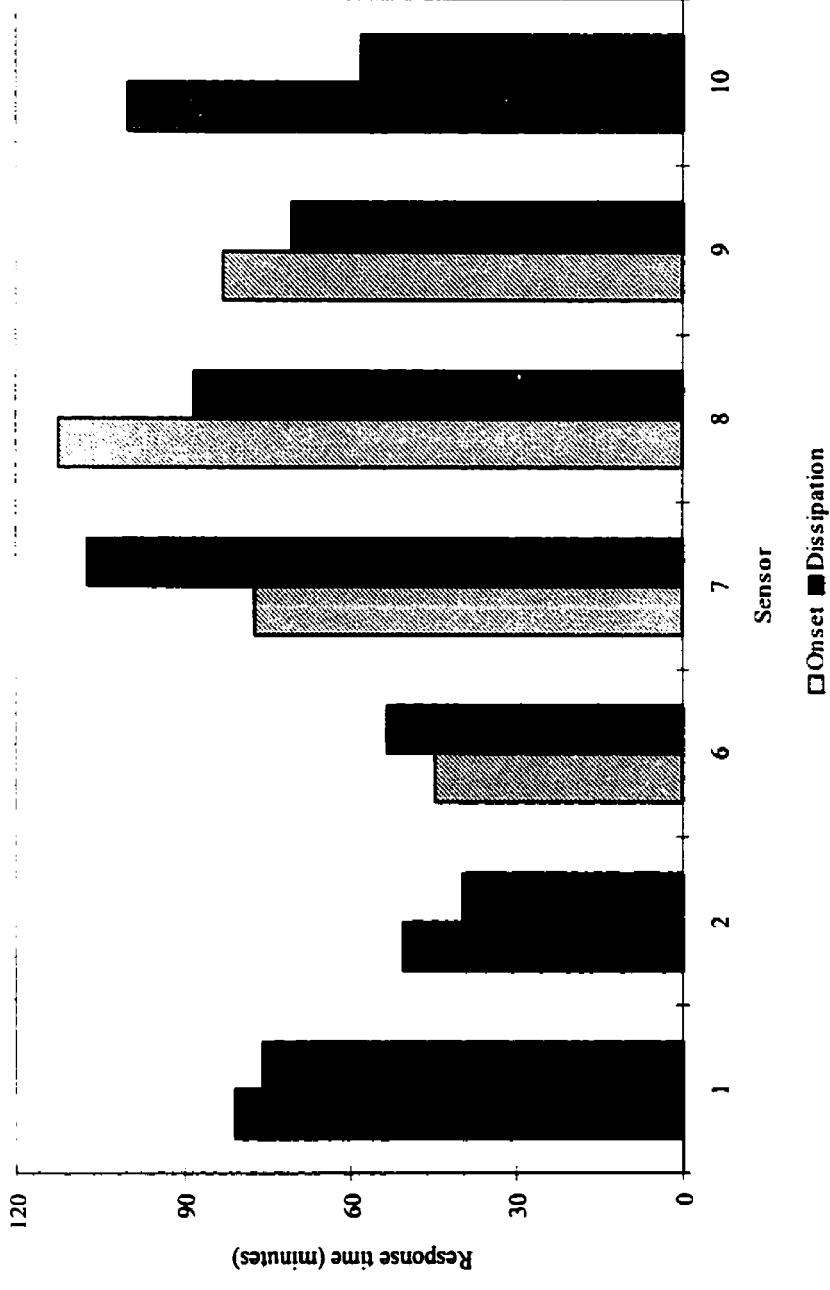


Figure 2.11 Standard deviation for sensor response time to dew onset and dissipation.

from the turfgrass. Therefore, S2 did not respond to guttation fluid, but did respond to water originating in the atmosphere. Whether this water vapor was, in part, from distillation could not be determined. The original belief was this sensor would be inaccurate because it did not measure all sources of dew. However, enhanced condensation may have occurred because of its substantial boundary layer. Boundary layer thickness is greater in large objects compared to small objects (Salsbury & Ross 1992). Condensation would occur faster on the sensor compared to the creeping bentgrass if the sensor boundary layer was large enough to create and retain a steep T gradient. Furthermore, its positioning in the Kentucky bluegrass canopy set it within the turfgrass microclimate. This microclimate likely contained a great deal of water vapor due to transpiration, creating a vapor pressure gradient between the sensor boundary layer and ambient air. Diffusion of water molecules into the sensor boundary layer would result from the vapor pressure gradient. Also, less turbulent air within the canopy would enhance the boundary layer resistance and not introduce warm air into the vicinity of the sensor boundary layer cooled by emission of infrared radiation. To support this fact, the identical sensor placed on top of creeping bentgrass (S1) responded to dew onset on average 3.6 minutes slower than the sensor in Kentucky bluegrass. The variation in Model 237 LWS response time was reduced by 30 minutes by placing it in the Kentucky bluegrass instead of on top of the creeping bentgrass. Results suggest the microclimate within the Kentucky bluegrass canopy was less variable than ambient air above the creeping bentgrass green.

The leaf cools at night because it emits long wave radiation into the atmosphere. Sensor 9, made from wire and string stretched across a frame, was designed to monitor water that originated from above and within the sward while not interfering with radiative cooling

of the grass. The only difference between S8 and S9 was the wire height above the creeping bentgrass surface, which was a function of frame thickness. Sensor 9 had a shorter response interval than S8 (108.1 minutes) because S9 was closer to the turfgrass. Sensor 10 had a similar design, but was placed flush with the turfgrass and responded relatively quickly to dew onset, 24.7 minutes later than S9.

S6 performed differently than anticipated with regards to accuracy. Condensation on an object at a height of 1.5 m should be less than on an object ground level because wind velocity increases with height. However, S6 responded, on average, 52.6 minutes before dew set on creeping bentgrass 150 cm below. Hygroscoopy is a reasonable explanation; at the end of the field season the sensor was responding to atmospheric water vapor. No comparative conclusions can be made about this sensor because identical instruments were removed from the study due to malfunctions. S6 was fixed above the creeping bentgrass, uninfluenced by the dynamic condition of the turfgrass or the soil beneath it, which explains its precision of 44.7 minutes.

2.4.2 Sensor Response to Dew Dissipation

Data were normal with equal variances. Sensor response to dew dissipation differed from dew onset. Sensors 3, 4, and 5 malfunctioned on >25 % of test days, therefore, were not considered for accuracy or precision evaluations and were excluded from statistical evaluations. Analysis of variance showed there was a difference in sensor response times (Table 2.3) for $n = 182$ during 1996 and 1997. The most accurate sensors were S2, S8, S10, and S6, respectively (Table 2.4, Figure 2.10). Sensor 2 and S6 tended to register as dry prior to actual dew dissipation by 0.9 and 12.4 minutes (mean response time), respectively, while

Table 2.3 Analysis of variance for response time (minutes) of sensors (S1, S2, S6 - S10) to dew dissipation for 36 observation nights during June, July, August and September in 1996 and 1997.

Source	DF	SS	MS	F	P-value > F
Corrected total	181	1715430.77			
Year	1	70093.14	70093.14	13.46	0.00032
Sensor	6	692171.73	115361.95	22.16	3.0 E-19
Error	174	905928.12	5206.79		

Table 2.4 Summarized 1996 - 1997 data for accuracy, precision and overall ranking of leaf wetness sensor response to dew dissipation

Response Time ^a	Sensor Number									
	1	2	3	4	5	6	7	8	9	10
Maximum	210	90	160	180	100	90	170	190	120	160
Minimum	-100	-30	0	0	-20	-90	-350	-220	-130	-140
LSM ^b	35.6	-0.9*	72.6	58.9	17.4	-12.4*	-147.6	2.9*	26.4	9.5*
Pr > T Ho: LSM=0 ^c	0.005	0.966	0.540	-	-	-	5.0E-22	0.821	0.046	0.481
SD ^d	76.0	39.7	50.0	59.6	48.5	53.3	107.4	88.2	70.6	57.9
LSM + SD	111.6	40.6	122.6	118.5	65.9	65.7	255.0	91.1	97.0	67.4
Sample Size	35	13	9	10	7	14	31	32	30	29
Malfunctions	2	2	6	5	8	1	4	3	1	2
Ranking ^e	6	1	-	-	-	2	7	4	5	3

a Time in minutes. Positive values represent premature response to dew dissipation, negative represent delayed response.

b Least Squares Means (LSM) used to assess accuracy.

c Probability that LSM response time is equal to the tactile assessment.

d Standard deviation (SD) used to assess precision.

e Ranked in order of smallest |LSM| + SD value.

***** LSM was not significantly different from tactile assessment ($\alpha = 0.05$).

S8 and S10 responded after dew dissipation by 2.9 and 9.5 minutes (mean response time), respectively. The most precise sensors were S2, S6, S10, and S9, respectively (Table 2.4, Figure 2.11). Based on the absolute LSM response time plus SD ($|LSM| + SD$), the best sensors for measuring dew dissipation were S2, S6, S10 and S8, respectively (Table 2.4). LSM comparisons showed these top ranked sensors did not significantly differ from the tactile observation ($\alpha = 0.05$). Sensor 2 measured dew dissipation within 40.6 minutes of the actual condition when the LSM and SD were combined. Once again, hygroscopic behavior is offered as an explanation for unexpected failure to show a stronger tendency toward early drying and therefore the high ranking response of sensor 6.

One concern when using the Model 237 sensor for monitoring LWD on creeping bentgrass is its dissimilar thermal properties compared to turfgrass. In the morning sun, S1 heated faster than adjacent creeping bentgrass because the grass could regulate its T through transpiration. Also, the three dimensional structure of the creeping bentgrass canopy provided more surface area for dew formation, and shaded adjacent blades of grass from solar radiation. As a result, dew dried more quickly from the sensor surface compared to creeping bentgrass. The Kentucky bluegrass canopy slightly shaded S2, thus reduced evaporation from the sensor surface due to latent heat loss, resulting in a delay of 34.7 minutes when compared to the same sensor positioned on the creeping bentgrass. Sensor 2 had a consistent response (SD of 39.7 minutes) because it was a component of an on-site weather station and was rarely moved or adjusted during the course of the season. Sensor 10, composed of two wires stretched between golf tees, rested on top of creeping bentgrass to directly monitor free water. It did not interfere with creeping bentgrass evaporative properties and was in close proximity with dew droplets; therefore, S10 responded much like

the turfgrass and dried only 9.5 minutes after dew dissipation. The golf tee sensor (S10) was a delicate instrument that required skilled placement. Ideally, the cable to the datalogger would be buried so the mower could pass above the instrument and the instrument could remain in place for the season. Elimination of the need to reposition S10 and modifications in the design to improve rigidity of the sensor may result in greater precision. In its current design, this sensor would be able to estimate dew dissipation within 67.4 minutes (mean response time including standard deviation) of the actual time, a resolution acceptable by currently available turfgrass disease forecasting models because L is measured in one-hour units (Danneberger et al. 1984, Fidanza et al. 1996).

2.4.3 Sensor Response to Leaf Wetness Duration

Sensors 3, 4, and 5 malfunctioned during >25 % of the observation dates, therefore, were not considered for accuracy or precision evaluations and were excluded in statistical evaluations. Data were not normal and did not have equal variances when the onset and dissipation responses were pooled for analysis using PROC GLM (SAS Institute, Inc. 1996). A square root transformation made data normal but transformed results were incompatible to test the hypothesis of no significant difference between tactile assessment and sensor response. Therefore, a different strategy was employed to determine the best sensor for monitoring the entire dew duration. Absolute LSM for dew onset and dissipation were summed to give overall accuracy (Table 2.5). The most accurate sensors were S2, S9, S1, and S10, respectively. Standard deviations for dew onset and dissipation were also summed, which revealed the most precise sensors were S2, S6, S9, and S1, respectively. When the total LSM and total SD were added together, the results showed the best sensors for

Table 2.5 Summarized 1996 - 1997 data and accuracy, precision and overall ranking of leaf wetness sensor response to dew duration

Criteria	Sensor Number									
	1	2	3	4	5	6	7	8	9	10
Total LSM ^a	47.3	16.2	187.3	175.0	58.8	65.0	226.3	124.9	40.3	48.1
Total SD ^b	156.9	90.1	123.3	323.5	131.8	98.0	184.8	200.5	153.7	157.8
Total (LSM + SD) ^c	204.2	106.3	310.6	498.5	190.6	163.0	411.1	325.4	194.0	205.9
Overall Ranking ^d	4	1				2	6	7	3	5

a The sum of absolute Least Squares Means (LSM) for dew onset and dissipation response.

b The sum of the Standard Deviation (SD) for dew onset and dissipation response.

c The sum of absolute Least Squares Means plus Standard Deviation for dew onset and dissipation response.

d Overall ranking of sensors for monitoring dew duration.

monitoring an entire dew event were S2, S6, S9, and S1, respectively (Figure 2.12). Therefore, the Model 237 LWS, when placed within a Kentucky bluegrass canopy maintained at a height of 5 cm, was the best sensor for monitoring dew duration on creeping bentgrass greens. This sensor would determine the dew duration on a creeping bentgrass green within 1.7 hours of the actual dew period (when LSM and SD were combined) that was approximately 15.5 h for 1996-97. S2 does not accurately estimate LWD when compared to the 0.1 h average response estimate of the site-specific sensor used in the tall fescue canopy (Giesler, et al. 1996). However, an alternative LWS had to be investigated because the site-specific sensor could not be used on creeping bentgrass greens. Accuracy of this sensor may be improved by using it in conjunction with other meteorological data. For example, the sensor underestimated the dew duration on days when solar radiation and wind velocity were greater. Sensor response combined with radiometer and anemometer instrumentation may be used in a model to yield more accurate results. Sensor 2 was manually positioned in Kentucky bluegrass following the weekly mowing. Placing the sensor on a support to hold it at a fixed position would improve precision. Several sensors positioned to represent the climatic variation of the green would give an average dew duration for the entire green, or even an entire golf course. The primary purpose of an impedance LWS on turfgrass is its use in forecasting systems for turfgrass diseases. The Model 237 LWS in Kentucky bluegrass had a resolution that was acceptable for use in a forecasting model for use on creeping bentgrass greens because L is measured in one-hour units (Danneberger et al. 1984, Fidanza et al. 1996). The model produced by Danneberger et al. (1984) used LWD, measured in hours, and air T. The average 1.7 h inaccuracy of the S2 sensor, when tested against the model produced by Danneberger et al. (1984), would not

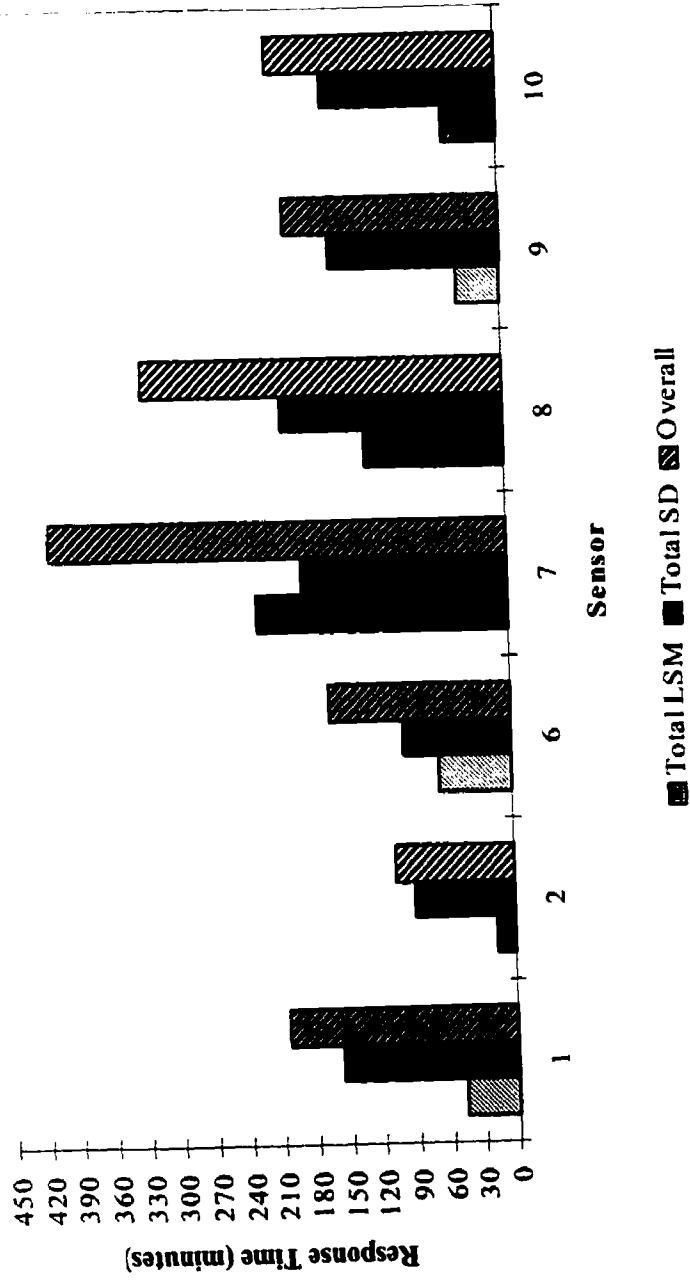


Figure 2.12 Sum of least squared means (LSM), standard deviation (SD) and absolute LSM + SD for sensor response to dew duration.

affect the predicted turfgrass area diseased by anthracnose. For example, if air T was 20 °C and the LWD was 6 h, but the sensor estimated 12 h, the model would predict only 3 % more diseased turfgrass. A difference of 1.7 h would result in only a 1 % over-prediction of disease. Therefore, the Model 237 LWS would provide data that could be used in existing turfgrass disease prediction models.

3. INFLUENCE OF LEAF WETNESS DURATION AND TEMPERATURE ON DEVELOPMENT OF DOLLAR SPOT ON CREEPING BENTGRASS IN A CONTROLLED ENVIRONMENT

3.1 ABSTRACT

The influence of leaf wetness duration (L) and temperature (T) on dollar spot severity in controlled environments was determined by inoculating pots of creeping bentgrass (*Agrostis palustris* 'Penncross') with two 0.25-cm² pieces of autoclaved grass infested with *Sclerotinia homoeocarpa* (isolate Sh48B). Pots were placed in environmental chambers set at 10, 17.5 or 25 °C for 0, 12, 24, 36, or 48 h of L. The diameter of dollar spot foci was measured six days after inoculation. Linear regression analysis was used to develop the model which estimated focus diameter, y (cm), for L and T combinations, where:

$$y = -0.3 + [-1.475 + (2.087 \times 10^{-1})T + (2.497 \times 10^{-2})L + (-4.284 \times 10^{-3})T^2 + (-6.485 \times 10^{-4})TL]^2.$$

This model explained 83.9 % of the variation within the data ($P < 1.0 \times 10^{-38}$, $n = 141$), however, precision was compromised when extrapolated beyond the T and L used to design the model. The estimated minimum T for disease development ranged from 10 to 12 °C with 22 and 12 h L, respectively. The optimum T was 21 to 24 °C, depending on L. Maximum T for disease development was not calculated because it was beyond the T tolerated by the creeping bentgrass. Results support the literature that states dollar spot is active between 15 and 27 °C. The linear response of disease to increases in L resulted in estimated focus diameter increasing as L was increased, at all temperatures tested. This model is the first to quantify the role of L in dollar spot severity. On-site measurements of L and T, used in conjunction with a disease forecasting model to time fungicide applications, may

substantially improve management of dollar spot on creeping bentgrass.

3.2 INTRODUCTION

Little information is available about climatic conditions required for dollar spot development. Controlled temperature (T) experiments established that minimum, optimum, and maximum T for growth of *S. homoeocarpa* on potato dextrose agar (PDA) were 4.5, 26.8, and >32 °C, respectively (32 °C was the highest T tested, at which the fungus still grew) (Endo 1963). *In vivo* cardinal temperatures for dollar spot development are unknown. *Sclerotinia homoeocarpa* is likely to infect and cause disease on turfgrass at ambient air T ranging from 15 and 27 °C but reaches its peak growth rate and maximum pathogenicity when T is between 21 to 27 °C and relative humidity (RH) is >85 % (Couch 1995, Endo 1963, Sears et al. 1996). Although this pathogen does not infect roots directly, *S. homoeocarpa* has been associated with a root damaging mycotoxin produced at T >15.5 °C (Malca & Endo 1965). However, isolates of *S. homoeocarpa* from different locations differ in their growth optima (Bennett 1937, Endo 1963, Fenstermacher 1980).

An important aspect of plant disease management is leaf wetness duration (L) because many pathogens require a film of water on the leaf surface for spores to germinate and for mycelia to infect the host (Sutton et al. 1988, Van der Wal 1978).

In other pathosystems, T and L within the microclimate of the host profoundly affected pathogen development and disease severity (Sutton et al. 1988, Van der Wal 1978). Disease forecasting systems for a variety of crops were designed using knowledge of specific environmental factors that influence disease development or cause increases in disease severity. Growers use these disease forecasting systems to schedule a fungicide or biological control agent application when local climatic conditions are conducive to disease. Successful

forecasting systems reduce the number of fungicide applications, thus minimizing costs to the producer and decreasing pesticide use. Forecasting systems for turfgrass diseases such as brown patch (*Rhizoctonia solani* Kühn) on perennial ryegrass (*Lolium perenne* L.) (Fidanza et al. 1996) and anthracnose (*Colletotrichum graminicola* Ces.) on annual bluegrass [*Poa annua* var. *reptans* (Hauskins Timm.)] (Danneberger et al. 1984) used L and T to predict disease. A fungicide application to control brown patch was recommended when RH was $\geq 95\%$ for ≥ 8 h and mean RH was $\geq 75\%$, L was ≥ 6 h or precipitation was ≥ 2 mm, and minimum air and soil T was ≥ 16 °C during a noon-to-noon interval. Use of this forecasting system provided the same brown patch control as a 14-day calendar-based spray schedule with chlorothalonil, but provided a 29 % reduction in fungicide applications (Fidanza et al. 1996). Multiple regression analysis of natural epidemics revealed that *C. graminicola* infected annual bluegrass when mean daily T was 19.5 to 22 °C with ≤ 24 h L for a 3-day period (Danneberger et al. 1984). Nutter et al. (1983) and Schumann et al. (1994) used T and RH in disease forecasting systems for rhizoctonia blight (*R. solani*) on creeping bentgrass (*Agrostis palustris* Huds.) and pythium blight (*Pythium* spp.), respectively. Relative humidity exceeding 90 % has been used as an estimator of leaf wetness (Sutton et al. 1984). A fungicide application to control rhizoctonia blight was scheduled when minimum and mean air and soil T was 15, 20, 18, and 21 °C, respectively, during the 24 h preceding the tenth consecutive hour of RH $\geq 95\%$. Also, RH $\geq 95\%$ for >36 h, or rainfall ≥ 15 mm in association with required soil T, was also predictive of severe rhizoctonia blight. When a forecasting system and calendar-scheduled applications of propiconazole were compared, there was no significant difference in rhizoctonia blight severity or number of fungicide applications (Nutter et al. 1983). Warm season pythium blight was controlled when

fungicides were only applied when maximum air T was >30 °C and, RH was >90 % for 14 h during which time the minimum air T remained >20 °C (Schumann et al. 1994).

Disease forecasting systems for dollar spot were proposed by Mills and Rothwell (M&R) (1982) and Hall (1984). In the M&R system, a fungicide application was recommended when maximum air T was ≥ 25 °C and maximum RH was ≥ 90 % during any 3 days of a 7 day period. In the Hall system, a fungicide application was recommended after two consecutive days of rainfall and a mean air T of ≥ 22 °C, or three consecutive days of rainfall and a mean air T of ≥ 15 °C. The two disease forecasting systems were compared in a 2-year study, but both models failed to accurately predict weather-related increases of dollar spot (Burpee & Goult 1986). The M&R model predicted too many infection periods, and disease suppression resulted more from the high frequency of prediction-based fungicide applications than model accuracy. The Hall system failed to predict sufficient infection periods, resulting in poor disease control. In this chapter, a controlled environment experiment was used to model dollar spot severity as it related to T and L.

3.3 MATERIALS & METHODS

3.3.1 Creeping Bentgrass Cultivation

Polystyrene pots (10-cm-diameter \times 9 cm) were 1/3 filled with Turface MVP (Applied Industrial Materials, Corp., Buffalo Grove, IL) and then completely filled with ProMix growing medium (Les Tourbieres Premier Ltee., Riviere du Loup, PQ). Growing medium was saturated with water and then placed in impermeable trays containing water. This procedure ensured that soil would remain moist during germination and for four days

following germination. A hand-held shaker was used to seed creeping bentgrass (*Agrostis palustris* Huds. 'Penncross') onto the soil surface at a rate of 2.3 g/m². Fine quartz sand was sprinkled over seeds as a topdressing. Trays were placed on growth room benches and covered with transparent plastic for six days, or until germination occurred. Trays and covers were removed when seedling height was approximately 1.0 cm. Seedlings remained in the growth room with fluorescent light (440 $\mu\text{Em}^{-2}\text{s}^{-1}$) for a 14 h light: 10 h dark cycle at 22 °C and 65 % RH during the day and 18 °C and 75 % RH during the night. Grass was watered with a 20:8:20 N:P:K fertilizer solution as required to provide satisfactory moisture but to avoid undesired *Pythium* spp. colonization. Plants were maintained in the growth room for four weeks. Turfgrass was clipped with hand-held shears (Model GS300-04, Black & Decker Canada Inc., Brockville, ON) every two to three days to maintain a height of approximately 1.5 cm; the final clipping was 12 h prior to inoculation.

3.3.2 Inoculum Preparation

Preliminary studies determined the type of inoculum which effectively caused dollar spot on creeping bentgrass. Agar plugs, mycelial suspensions, a colonized sand:cornmeal mixture, and grass clippings infested with *S. homoeocarpa* were applied to pots of creeping bentgrass and incubated at >95 % RH and 22 °C for 48 h. Mycelial growth was noted and the plants were assessed for disease six days after inoculation. Infested grass inoculum was chosen for subsequent experiments because mycelia readily grew at the start of the leaf wetness period, mycelia reliably grew from inoculum substrate to healthy blades of grass, and inoculum was consistent between batches.

To prepare infested grass inoculum, the first clippings from creeping bentgrass

seedlings were collected. Clippings were placed in a 1-litre beaker, covered with aluminum foil and autoclaved at 121 °C for 15 minutes. This autoclaved grass (3.5 to 4.0 g) was transferred to a sterile Petri plate (9-cm-diameter) and inoculated with five 5 mm-diameter plugs taken from the actively-growing margin of a *S. homoeocarpa* colony on PDA. Isolate Sh48B, used for all experiments, was collected from Cambridge, ON. Inoculated grass was incubated on the laboratory bench at 20 - 22 °C for 14 days and mycelia grew through the grass to form a semi-solid mat that filled the Petri plate. Inoculum was cut into 0.25-cm² pieces, placed in a Petri plate with a moist filter paper, sealed with ParaFilm laboratory film and stored at 20 - 22°C for no more than 12 h prior to experimentation.

3.3.3 Environmental Chambers

Environmental chambers were 50×50×150 cm plexi-glass enclosures that maintained RH at 94 - 96 % through the use of a micrologger-controlled (Model 21X, Campbell Scientific, Inc., Logan, UT) ultrasonic mister (Hannusch et al. 1995). Relative humidity, measured by a model HMP35C probe (Vaisala, Inc., Woburn, MA), provided periods of unbroken leaf wetness without saturating soil within the pots. Enclosures were placed in growth cabinets set at specific T (10, 17.5, and 25 °C). Temperature within each enclosure was monitored at 10 minute intervals using a thermistor (Model 107, Campbell Scientific, Inc.). In one enclosure per replication, a thermacouple was placed in each of the four corners of the enclosure to measure potential spatial differences in T. Incandescent and fluorescent lights in growth cabinets provided 150 $\mu\text{Em}^{-2}\text{s}^{-1}$ of light during the 12 h light: dark cycle.

3.3.4 Creeping Bentgrass Inoculation

Pots of creeping bentgrass were placed into growth cabinets 12 h prior to inoculation to allow soil and grass to equilibrate with cabinet T. Two pieces of inoculum were placed on opposite sides of the turfgrass surface of each pot. Pots were randomly placed within the enclosure and misted with deionized water until droplets formed on blades of grass. The enclosure was sealed and 94.0 to 96.0 % RH was maintained to ensure continuous leaf wetness. At 0, 12, 24, 36 and 48 h after inoculation, treated pots were removed from the enclosure, jostled to shake water from foliage without disturbing the pathogen, and remained within the growth cabinet at the same T until the end of the experiment. All pots were returned to the growth room bench after the 48 h treatment had concluded.

3.3.5 Disease Evaluations

Plants were not clipped during the six days between inoculation and disease assessment. A six-day interval allowed sufficient time for symptom expression but not enough time for grass to recover from damage. Disease was evaluated by measuring the maximum diameter (cm) of the focus produced by mycelial growth from a single inoculum site. Each focus was composed of many individual blades of diseased grass that collectively formed a dollar spot on the turfgrass surface.

3.3.6 Statistical Design and Analysis

Experiments were conducted as a modified central composite rotatable response surface design (Cochran & Cox 1957) as outlined by Allen et al. (1987). This design was utilized to develop a surface response curve of disease severity (y), expressed as focus

diameter, as it relates to L measured in hours and T measured in °C. This was a rotatable design with six design points, or unique L and T combinations, falling at the vertices of a hexagon and with the center point replicated three times. Allen et al. (1987) used incomplete factorial arrangements to estimate the response surface with uniform precision. The center point was the only point that required three repetitions. However, in this study, all treatments were repeated three times to account for growth cabinet variation. The final design was a response surface imposed on a Latin square design of three T replications randomized among three growth cabinets for three repetitions. Within the Latin square, there was a split plot design where T was the whole plot and L was the split plot component (Figure 3.1). Experimental temperatures were chosen based on pathogen and host biology. Fungal growth on pots of creeping bentgrass was not observed in preliminary studies when $T < 10$ °C. The upper limit of 25 °C was chosen to maintain a healthy host because creeping bentgrass is a cool-season grass and was physiologically stressed when maintained at high T for 48 h. The third experimental T was 17.5 °C because the statistical design demanded the median T of the two extremes. Experimental T of 10, 17.5, and 25 °C were each combined with L of 0, 12, 24, 36, and 48 h (not including drying time). Experimental T was not consistently achieved; therefore, mean air T within each cabinet during the leaf wetness periods was used in fitting the surface response curve. Combinations of T and L were tested using two pieces of inoculum on two pots of turfgrass and were repeated three times, providing 12 dollar spot foci for statistical analysis.

Prior to fitting the surface response curve, data were checked to ensure normality and equal variances using the Shapiro-Wilk *W*-test statistic for normality (Royston 1995) and an informal test examining residual plots for homogeneity (Kuehl 1994). Data were

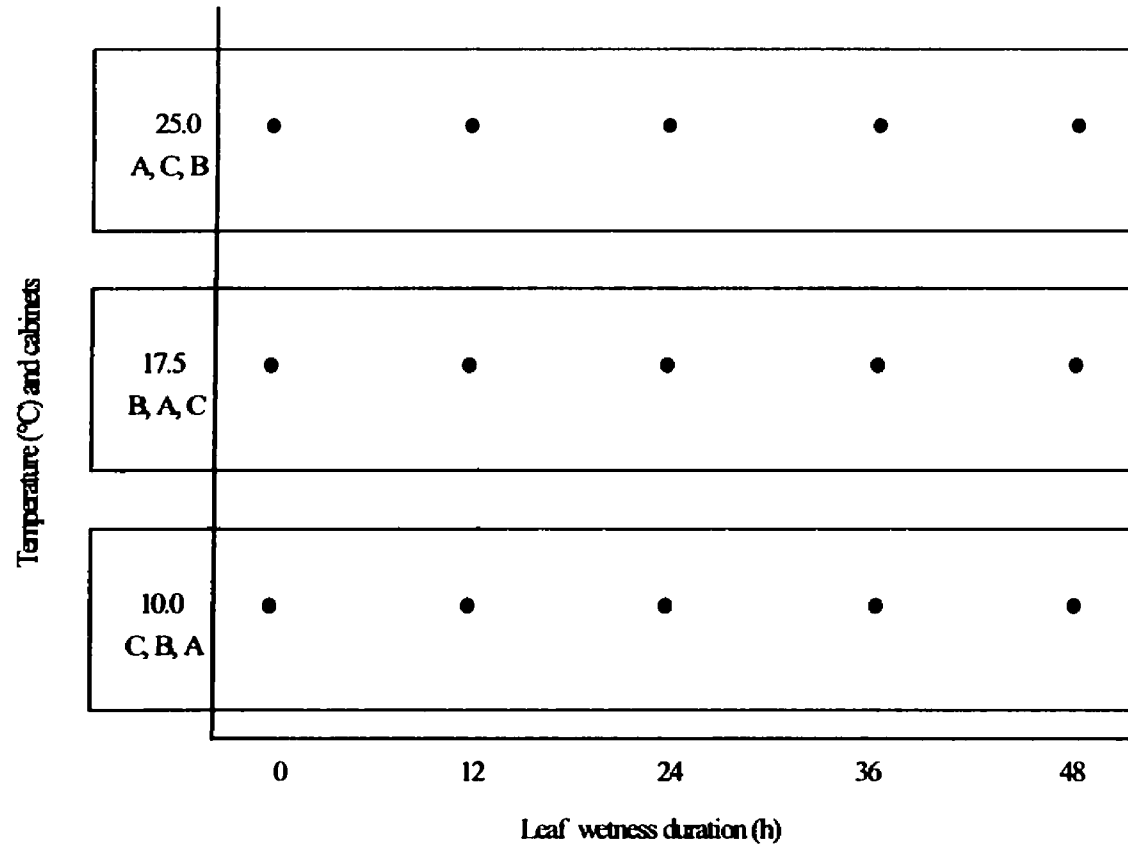


Figure 3.1 Treatment combinations (●) of leaf wetness duration and temperature used to estimate the surface response curve. Treatments were randomized among three growth cabinets (A, B, C) for three repetitions in a Latin square design.

transformed using square root transformation of focus diameter plus 0.3 cm to ensure they were distributed normally. PROC MIXED (SAS Institute Inc., Cary, NC) was used to account for random effects of growth cabinet, repetition, and pot variation; and linear regression analysis was used to model a surface response curve for dollar spot focus diameter in relation to T and L.

3.4 RESULTS

3.4.1 Environmental Chambers

Turfgrass and inoculum were exposed to unbroken periods of L because RH within environmental chambers was maintained at $94.8 \pm 2.8\%$. Actual T within enclosures differed from experimental T, on average, by $\pm 0.7^\circ\text{C}$. Actual T fluctuated, on average, $\pm 1.5^\circ\text{C}$ from the mean, with greatest deviations from the mean of -2.6°C and $+4.0^\circ\text{C}$. Temperature did not differ spatially within each plexiglass enclosure.

3.4.2 Influence of Temperature and Leaf Wetness Duration on Mean Focus Diameter

Dollar spot foci ranged from 0 to 3.0 cm in diameter, the largest focus was produced during 48 h L with a mean T of 17.1°C . Dollar spot foci did not develop on plants that remained dry (L = 0 h). Analysis of variance (ANOVA) (Table 3.1) showed that disease development was not influenced by growth cabinet, therefore, data were pooled and presented in Figure 3.2. Each datum point was the simple mean of two foci diameters from two pots of grass for three growth cabinet repetitions ($n = 12$). The effect of L and the interaction of T \times L were significant ($P = 0.0000$ and $P = 0.0088$, respectively). Mean focus

Table 3.1 Analysis of variance (ANOVA) for simple mean focus diameter of dollar spot (*S. homoeocarpa*) on creeping bentgrass (*A. palustris*) exposed to 0, 12, 24, 36, and 48 h leaf wetness duration at 10, 17.5 and 25°C

Source	DF	SS	MS	F	P
Corrected total	44	95.359			
Whole plots	8	36.446			
Growth cabinet	2	0.487	0.244	0.09	0.9148
Temperature (T) ^a	2	25.254	12.627	4.72	0.0886
Error T	4	10.705	2.676		
Split plots	36	58.913			
L ^b	4	44.341	11.085	39.25	0.0000
L * T	8	7.794	0.974	3.45	0.0088
Error L	24	6.778	0.282		
a	Air temperature (°C)				
b	Leaf wetness duration (h)				

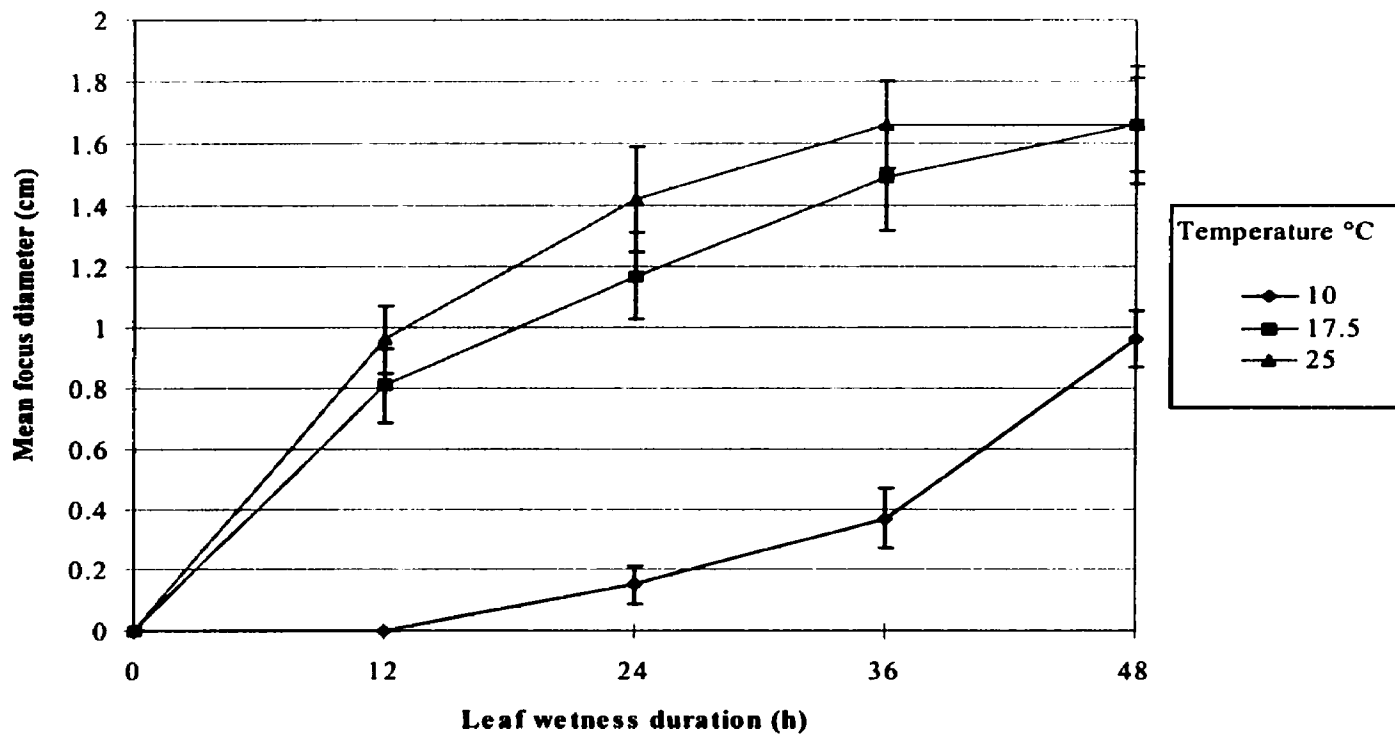


Figure 3.2 Influence of temperature and leaf wetness duration on simple mean focus diameter of dollar spot (*S. homoeocarpa*) on creeping bentgrass (*A. palustris* 'Penncross'). Each datum point (and standard error bar) represents the simple mean focus diameter of 12 foci (two inoculum sites × two pots of creeping bentgrass × three repetitions).

diameter was smallest at 10 °C and increased with T for each L, with the exception of 48 h L at 25 °C. At 48 h L, experimental temperatures of 17.5 and 25 °C produced mean foci diameters of 1.7 cm. When exposed to periods of leaf wetness (L >0), mean focus diameter increased with L at each T with the exception of 10 °C at 12 h L, which did not produce disease at any inoculation sites.

3.4.3 Modeling the Influence of Temperature and Leaf Wetness Duration on Dollar Spot Focus Diameter on Creeping Bentgrass

Sclerotinia homoeocarpa did not grow on, or cause visible symptoms on, plants unexposed to periods of leaf wetness (L = 0 h). This created non-equal variances among treatments, therefore, treatments of L = 0 h were not used in the statistical analysis. A surface response curve for dollar spot focus diameter in relation to T and L was estimated with a second-order model:

$$y = b_0 + b_1T + b_2L + b_{11}T^2 + b_{22}L^2 + b_{12}(T \times L) + e \quad (\text{Equation 3.1})$$

where y is the estimated focus diameter (cm), T is the mean air temperature (°C) during the leaf wetness period, L is the leaf wetness period (h), b_{ii} are estimates of coefficient parameters presented in Table 3.2 and, e is a normally distributed random variable with mean zero and variance σ^2 (SAS Institute, Inc. 1996). The quadratic effect of L was excluded from the model because it was not significant ($P < 0.766$, Table 3.2), resulting in the equation for focus diameter:

$$y = -0.3 + [-1.475 + (2.087 \times 10^{-1})T + (2.497 \times 10^{-2})L + (-4.284 \times 10^{-3})T^2 + (-6.485 \times 10^{-4})T \times L]^2$$

(Equation 3.2)

Table 3.2 Estimated values, standard error, percentage of standard error (%), degrees of freedom (DF), and *P*-values for coefficient parameters in surface response model for the relationship of temperature (*T*) and leaf wetness duration (*L*) to dollar spot (*S. homoeocarpa*) focus diameter (*y*) on creeping bentgrass (*A. palustris* cultivar ‘Penncross’) inoculated with isolate Sh48B

Parameter	Estimated value ^a	Standard error	% ^b	DF	two tailed <i>P</i> -value
b_0	-1.475	2.450×10^{-01}	88.81	2	2.65×10^{-02}
b_1	2.087×10^{-01}	2.291×10^{-02}	8.30	68	2.14×10^{-13}
b_2	2.497×10^{-02}	7.093×10^{-03}	2.57	68	7.73×10^{-04}
b_{11}	-4.284×10^{-03}	6.031×10^{-04}	0.22	68	9.26×10^{-10}
b_{22}	-3.007×10^{-05}	1.006×10^{-04}	0.04	68	7.66×10^{-01}
b_{12}	-6.485×10^{-04}	1.882×10^{-04}	0.07	68	9.80×10^{-04}

- a Parameters of surface response model: $(y + 0.3)^{1/2} = b_0 + b_1T + b_2L + b_{11}T^2 + b_{22}L^2 + b_{12}(T \times L) + e$; where *y* is the estimated value for focus diameter (cm), *T* is the mean temperature (°C) during the leaf wetness period (*L*) (h).
- b Standard error as a percentage of total standard error (0.2755884).

The fitted surface response curve explained 83.9 % ($P < 1.0 \times 10^{-38}$, $n = 141$) of the variation within the transformed data.

The *in vivo* cardinal temperatures for disease development were estimated using the model presented in Equation 3.2. The smallest focus diameter, estimated at -0.27 cm, was produced at 10 °C with 0 h L. Obviously, a negative focus diameter was impossible, and this result was an artifact of removal of the L = 0 treatment from the statistical model. The surface response curve in Figure 3.3 was modified by changing all negative values to zero and restricting the leaf wetness parameter to 12 to 48 h L. The minimum T for disease development ranged from 10 °C with 22 h L to 12 °C with 12 h L. Estimated focus diameter increased with T until maximum disease was predicted between 21 and 24 °C depending on L. The largest estimated focus diameter (2.14 cm) was produced with 48 h L at 21°C. Estimated focus diameter decreased as T progressed beyond the optimal T. The maximum T was not calculated because precision of the model was compromised when extrapolated beyond the test points (10 to 25 °C for 12 to 48 h). Leaf wetness duration caused a linear response in estimated focus diameter that increased as L increased.

3.5 DISCUSSION

The study quantified the relationship of T and L to dollar spot development on creeping bentgrass. The initial analysis of simple means established that L, T, and the interaction of L×T had an effect on dollar spot foci diameter. Subsequent regression analysis was used to model focus diameter in relation to L and T.

Observations cited by Couch (1995), Sears et al. (1996) and Smiley (1992) indicated

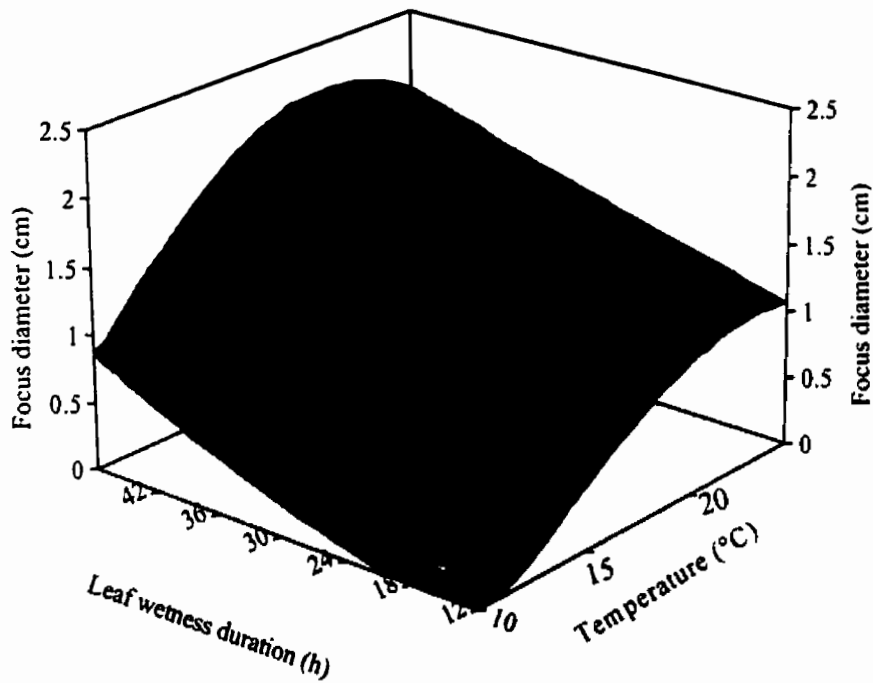


Figure 3.3 Effect of leaf wetness duration and temperature on dollar spot (*S. homoeocarpa*) on creeping bentgrass (*A. palustris* 'Penncross'). Estimated from model equation $y = -0.3 + [-1.475 + (2.087 \times 10^{-1})T + (2.497 \times 10^{-2})L + (-4.284 \times 10^{-3})T^2 + (-6.485 \times 10^{-4})T \times L]^2$ where y is the estimated value for focus diameter (cm), T is the mean temperature (°C) during the leaf wetness duration (L) (h).

that disease symptoms arose when mean daily T was >15 °C. Mean daily T is the net sum of turfgrass temperature when cool and wet (e.g., during the night) and when warm and dry (e.g., during the day). Results of this study indicated L was a limiting factor in dollar spot development; therefore, T during the night-time dew period is of primary importance to disease development. Simple means showed that *S. homoeocarpa* did not cause disease at 10 °C when leaf tissue was wet for 12 h, but did at 17.5 °C with the same L, which indicates a threshold T between 10 and 17.5 °C. The model estimated dollar spot activity began between 10 and 12 °C when foci diameters were 0.05 and 0.08 cm, respectively. These were small foci that may not be noticed in field conditions but, if an arbitrary focus diameter of 0.5 cm was selected as a visual threshold, the model predicted that dollar spot activity began when T was 15 °C with 16 h of L. Predictions by the model support the statement that symptoms are expressed when $T >15$ °C (Couch 1995, Sears et al. 1996, Smiley 1992). Couch (1995), Sears et al. (1996), and Smiley (1992) also reported that dollar spot reached maximum severity when mean daily T >21 °C. The model developed in the present study estimated maximum focus diameter when T was between 21 and 24 °C, regardless of L.

Endo (1963) found minimum, optimum and maximum T for growth of *S. homoeocarpa* on PDA were 4.5, 26.8, and >32 °C, respectively (Endo, 1963). Temperatures of 4.5 and 32 °C were not included in the T range of this study and the model was not extrapolated because of loss of precision. The optimum T of 26.8 °C for growth on PDA was greater than the optimum T of 21 to 24 °C estimated by the model. The discrepancy could be a function of *S. homoeocarpa* growth on PDA compared to its pathogenicity on creeping bentgrass. Also, isolates from different geographical regions may differ in cardinal T. This study did not establish an upper T limit for disease development because potted plants

exposed to $T > 25$ °C and $RH > 94$ % for 48 h did not survive. Field epidemics may provide information about the maximum T for pathogenicity of *S. homoeocarpa*.

Prolonged duration of dew and high RH were associated with increased dollar spot severity, thus indicating that leaf wetness was important to disease development (Couch 1995, Sears et al. 1996, Smiley et al. 1992, Williams et al. 1996). This study is the first to quantify the relation of L and dollar spot severity. Regardless of T , disease did not develop when $L = 0$ h, yet the model predicted disease. A lack of L can be assumed to eliminate the possibility of disease development when using the equation to estimate disease based on climatic conditions. In the predictive model, L was a linear response because the quadratic effect was excluded from the model due to insignificance and because of the relatively small contribution of $T \times L$ in the model. This linear response predicted that dollar spot focus diameter will increase indefinitely; thus, the limiting factor is the probability of an uninterrupted leaf wetness period.

Dollar spot forecasting systems were designed by Hall (1984) and Mills and Rothwell (1982). In the M&R system, a fungicide application was recommended when maximum air T was ≥ 25 °C and maximum RH was ≥ 90 % during any 3 days of a 7 day period. The M&R system did not account for mean daily T , a variable that would be more applicable based on the data gathered in this study. Hence, no comparisons were made to the M&R system. In the Hall system, a fungicide application was recommended after two consecutive days of rainfall and a mean air T of ≥ 22 °C, or three consecutive days of rainfall and a mean air T of ≥ 15 °C. The model estimated that maximum focus diameter resulted when mean T was 22 °C with 48 h L and, the model predicted disease when T was 15 °C with 16 h L . The Hall system would not suggest a spray application for the 15 °C condition, although it may have

been deemed necessary by the growth room model. This was supported by a two year study which found the Hall system underestimated dollar spot incidence, and fungicide applications scheduled with the Hall system did not control disease (Burpee & Goultly 1986).

The model to estimate focus diameter was designed with data from controlled environment experiments where T remained relatively constant during each L. Temperature tends to steadily decline between sunset and sunrise in the natural environment, at the same time turfgrass that is wet from dew and favorable for fungal infection. Temperature may vary when weather systems change during the night, when parcels of air move through the microclimatic site, or when radiative cooling of the grass is altered due to changes in cloud cover. Whatever the cause of T variation, the model may be erroneous in estimating dollar spot severity because of the lack of a constant T. Hourly air T during the leaf wetness period, sampled as close to the grass surface as possible, is recommended to be used as T in Equation 3.2.

On golf course greens and fairways, once dew condenses in the evening, L usually remains unbroken until dew finally dissipates in the morning (Chapter 2, Williams et al. 1996); therefore, the value for L can be used directly. Guttation was not mimicked in growth cabinets because L was generated with condensation that originated from vaporized water. The lack of guttation fluid may have resulted in underestimation of disease severity because these exudates contain nutrients which increase fungal growth (Williams et al. 1998).

4. ETIOLOGY AND EPIDEMIOLOGY OF DOLLAR SPOT IN SOUTHERN ONTARIO

4.1 ABSTRACT

Etiology and epidemiology studies were conducted during 1996 and 1997 at the Guelph Turfgrass Institute (GTI) and in 1998 at the Cutten Club and Victoria West Golf Course. Dollar spot epidemics began on 14 June 1996, 17 June 1997, and 22 May 1998 at the GTI. During 1997 and 1998, epidemics began when irises and peonies were in bloom and when lilacs were in full- to late-bloom. Symptoms of dollar spot began when mycelia in contact with green foliage caused a water-soaked appearance. The tissue was greyish-green without a reddish-brown lesion border. Grass exhibiting these symptoms was marked with golf tees in the morning and, by the next day, water-soaked lesions had desiccated to form typical bleached tissue of dollar spot. Spores and colonies that arose from isolates cultured from diseased tissue were *S. homoeocarpa*, *Fusarium* spp., *Bipolaris* spp. and *Drechslera eruthrospila*; however, dollar spot was the only disease to develop on the turfgrass. Smiley et al. (1992) stated that mycelia of *S. homoeocarpa* could be confused with spiderweb on the surface of turfgrass. Sampling and culturing of mycelia and spiderwebs confirmed that mycelia of *S. homoeocarpa* only grew from diseased foliage. Mycelia of *S. homoeocarpa* was robust and white, and did not collect free water on its surface. In contrast, spiderwebs were not associated with diseased foliage, were fine and clear in color, and collected dew on the filaments. Disease progress curves were best fit to exponential or logistic models, both of which describe compound interest diseases. Dollar spots would

reappear in the same location when temperature became favorable for disease. This three-year study supported the hypothesis of Britton (1969), Couch (1995), Smiley et al. (1992) and Fenstermacher (1980) that stroma and/or mycelia of *S. homoeocarpa* survive locally in turfgrass crowns or foliage and are the initial inoculum for the disease. Each piece of mycelium (in grass clippings or individually) is a potential colony forming unit of secondary inoculum that leads to new infections. If the initial inoculum is controlled, the start of disease progress will be delayed and will not reach the magnitude of disease of an untreated site. Use of control measures during the epidemic will reduce the secondary inoculum and thus reduce the rate of disease progress.

4.2 INTRODUCTION

Dollar spot is a fungal disease of turfgrass caused by the pathogen, *Sclerotinia homoeocarpa*. Dollar spot is prevalent in North America, Central America, Australia, New Zealand, Japan, the British Isles and continental Europe, and all commonly cultivated turfgrass species are susceptible (Couch 1995, Fenstermacher 1980, Vargas 1994). Except for western Canada and the Pacific northwest region of the United States, dollar spot is the most common disease of turfgrass in North America (Couch 1995).

Sclerotinia homoeocarpa is believed to overwinter as darkly-pigmented stromata remaining on margins of dollar spot lesions from previous epidemics (Britton 1969, Couch 1995, Smiley et al. 1992). However, Fenstermacher (1980) suggested that *S. homoeocarpa* more likely survives as dormant mycelia in infected grass crowns and tissues. The pathogen primarily infects leaves via mycelial growth into cut leaf tips and stomata, but direct penetration into the leaves also occurs (Endo 1966, Monteith & Dahl 1932). Ascospores are assumed to be of minor importance to the spread of disease because these propagules are rarely observed in nature. However, fertile apothecia in turfgrass swards were reported in Britain, suggesting that ascospores may be a potential source of initial inoculum in the spring (Baldwin & Newell 1992). Local distribution of dollar spot occurs when mycelium grows from a diseased leaf to a healthy leaf in close proximity. The pathogen is suspected to be distributed over larger areas by physical displacement of the fungus. Humans can transport infested and diseased material, such as grass clippings, on the bottoms of golf shoes, on golf cart tires, and on maintenance equipment such as mowers, sprayers, and irrigation hoses (Smith 1955). On individual blades of grass, the first symptom of disease is yellow green

blotches that progress to a water-soaked appearance (Smith 1955). As disease advances, infected tissue bleaches to straw-colored tan lesions with reddish-brown borders, and these lesions often enlarge across the entire leaf (Smiley et al. 1992, Smith 1955). Reddish-brown lesion borders do not typically occur on annual bluegrass (*Poa annua* L.) (Couch 1995, Vargas 1994). Usually, entire leaves are blighted but, in some cases, only portions of a leaf become necrotic (Couch 1995, Smiley et al. 1992).

Symptoms of dollar spot on turfgrass swards vary according to turfgrass species and management practices. On closely-mown turfgrass, such as that on golf course putting greens, sunken, circular, straw-colored patches develop that range in size from a few blades of grass to spots the size of a silver dollar (5.0 to 7.5 cm diameter) (Smith 1955). Necrotic patches stand out because they contrast sharply with adjacent healthy turfgrass. If disease progresses, these necrotic patches coalesce to form larger areas of necrotic, straw-colored turfgrass (Couch 1995, Smith 1955). As mowing height is increased on golf course fairways, parks, and home lawns, patches of blighted grass range from 6 to 12 cm in diameter, but may coalesce to form large, irregular shaped areas of injured turfgrass (Couch 1995).

In addition to symptoms of disease, mycelial growth of the pathogen also can be observed. When dew is present on leaves, or during extended periods of high relative humidity, white cobweb-like aerial mycelia of the pathogen can be seen growing on the surface of turfgrass and extending from leaf to leaf (Monteith & Dahl 1932, Smith 1955). However, mycelia are sometimes confused with spiderwebs, downy seed tufts of cottonwood trees (*Populus* spp.) (Smiley 1983), and mycelia of *Pythium*, *Rhizoctonia* and *Nigrospora* spp. (Smiley et al. 1992). Therefore, correct diagnosis of disease is dependent on additional information, such as weather conditions, history of the site, management practices and

isolation of the pathogen. The objective of the following research was to describe symptom expression of dollar spot and model epidemics of dollar spot in southern Ontario.

4.3 MATERIALS & METHODS

4.3.1 1996 and 1997 Field Sites

Etiology and epidemiology studies were conducted at the Guelph Turfgrass Institute (GTI), Guelph, ON, Canada, 43°32.97 N, 80°12.90 W. Plots of 25 m² with 2 m borders were established on two greens. The first green, the pathology green, was a 1022 m² United States Golf Association (USGA) green built in 1993 using salicaceous soil. The pathology green was primarily used for turfgrass disease research because it was sheltered from morning sunlight and wind and therefore promoted long dew periods (Figure 4.1). The second green, the native green, was an undrained non-USGA green constructed with sand available on site (Figure 4.2). Dew duration was less than that of the pathology green because tree cover less dense. Greens were seeded in 1994 with creeping bentgrass (*Agrostis palustris* 'Penncross') stands (Anonymous 1994). Grass was maintained at 4.8 mm, was mowed 3 - 4 days per week and was irrigated to avoid drought stress. Plots were fertilized with half-rate nitrogen (N) (125 kg N/ ha/ year) to promote natural epidemics because N applications are associated with dollar spot recovery (Freeman 1969, Hoyland & Landschoot 1993, Landschoot & McNitt 1997, Markland et al. 1969, Smiley et al. 1992). Artificial inoculations during previous years established populations of *S. homoeocarpa* on both greens. There was increased disease pressure on the pathology green because it had an established history of more severe disease than the native sand green. No fungicides or herbicides were applied



Figure 4.1 Pathology research green at the Guelph Turfgrass Institute.

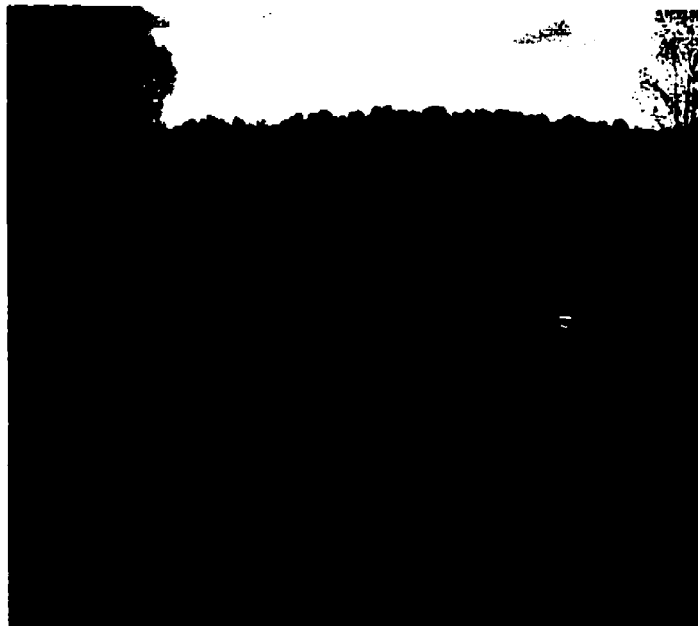


Figure 4.2 Native sand research green at the Guelph Turfgrass Institute.

during the epidemics; however, applications of pentachloronitrobenzene at 360 g of formulated product/100 m² (The Scotts Company, Marysville, OH) were applied in November of 1996 and 1997 to control pink snow mold (*Microdochium nivale*).

4.3.2 1998 Field Sites

Plots were first established at the GTI, but on 15 June 1998 were relocated to the Cutten Club, Guelph, ON, Canada, 43° 31.78 N 080° 13.37 W, and Victoria West Golf Course, Guelph, ON, Canada, 43° 31.80 N 080° 11.59 W. Test plots on each golf course were located where there was a history of dollar spot occurrence. At the Cutten Club, the 2.5 × 10 m (25 m²) plot with a 2 m border was positioned on the north side of the ninth fairway (Figure 4.3). This was a mature turfgrass sward of approximately 70 % creeping bentgrass and 30 % annual bluegrass. The management regime was typical of industry standards; and included mowing three days per week to a height of 6.5 mm, clippings not removed, fertilization at regular intervals, irrigation to avoid drought stress, and morning dew removal. Iprodione (Rovral, Rhone-Poulenc Ag Company, Research Triangle Park, NC) fungicide was applied to the adjacent fairway to control dollar spot but the fungicide did not contact the plot area.

The Victoria West Golf Course site was a 25 m² plot positioned on the north side of a 2-year-old creeping bentgrass nursery green (Figure 4.4). The management regime for the entire green included: mowing two days per week to a height of 6.5 mm (clippings removed), fertilization with half rate of N, irrigation to avoid drought stress and no fungicide.

4.3.3 Disease Assessments

Scouting for disease began prior to dollar spot appearance during the spring of 1996,

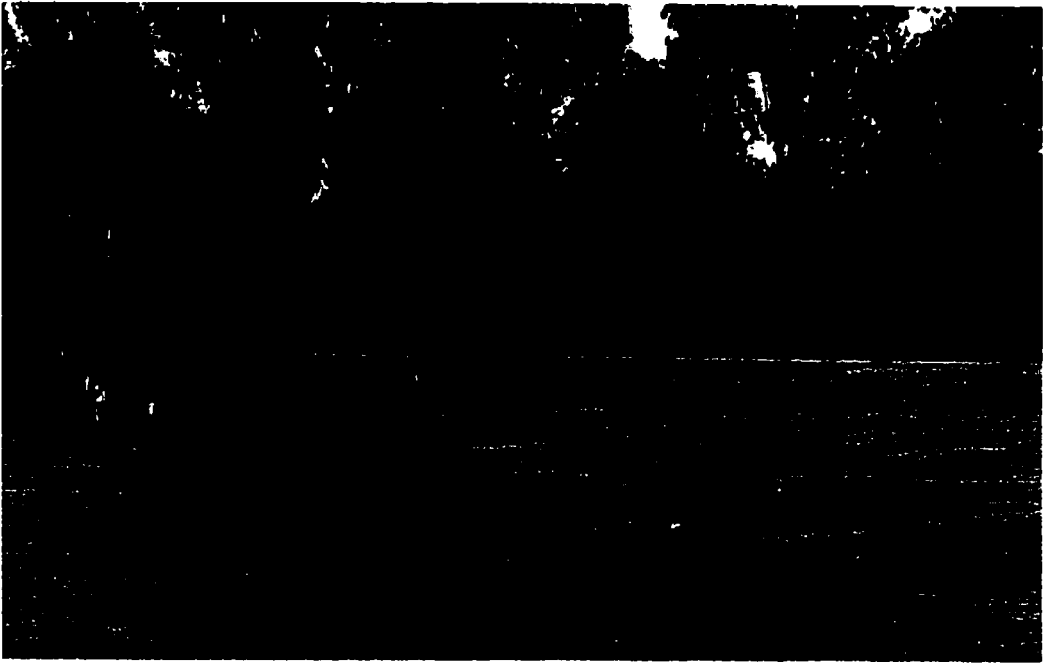


Figure 4.3 1998 research plot on the 9th fairway of the Cutten Club.



Figure 4.4 1998 research plot on the nursery green at Victoria West Golf Course.

1997, and 1998. Diseased tissue was sampled from the first dollar spot foci noticed in 1997 and 1998 to confirm *S. homoeocarpa* activity at the GTI. Blades of grass were surface sterilized for one minute with each of 70 % ethanol, 10 % bleach, and then rinsed with sterile water. Grass was placed on potato dextrose agar (PDA) or a selective medium of PDA augmented with 100 ppm streptomycin, 100 ppm penicillin, 25 ppm rose bengal, and 50 ppm bromophenol blue (SPDA). Mycelial growth was subcultured after 24 h and incubated for two weeks at 22 °C.

Observations of actively growing mycelia of *S. homoeocarpa* were conducted on 65 mornings during the summers of 1996, 1997 and 1998. Smiley et al. (1992) stated the mycelia of *S. homoeocarpa* were often confused with spiderwebs that collected dew on the surface of golf greens. Structures that resembled fungal mycelia, whether or not they were growing in association with diseased turfgrass, were sampled to determine if they were mycelia or spiderwebs. Samples were collected by entangling strands of web or mycelia around an autoclaved dissecting pin or pipe-cleaner. These samples were plated on SPDA and incubated at approximately 22 °C for 24 h, then the leading edge of mycelial growth was subcultured and assessed for presence of *S. homoeocarpa* six to ten days later. On 31 July 1997, the spiderweb-like structures were collected on sterilized glass slides.

Daily observations for new dollar spot foci were made at 15:00h during 1996 and 1997. In 1998, daily observations were made before 09:00h to avoid interference with golfers. Plots were divided into 25 - one m² subplots (10 - 2.5 m² subplots at the Cutten Club) to facilitate counting of dollar spot foci. New foci were counted and marked with golf tees to avoid recounting the spots. Golf tees were pushed into the ground until flush with the turfgrass surface so the mower could pass over them. Lesions located at the Cutten Club site

were not marked with golf tees because they would interfere with play on the fairway. Greens were assessed for dollar spot until disease diminished in September or October. Data gathered from the daily counting of new foci were used to characterize the epidemics.

4.3.4 Disease Progress Curves and Epidemic Modeling

Epidemics were presented as disease progress curves of cumulative foci over the season. Curves were analyzed using EPIMODEL (Nutter & Parker 1997) to fit five temporal population growth models used commonly in the analysis of plant disease epidemics (i.e., monomolecular, exponential, logistic, Gompertz and linear). To format the data for the software, epidemics from each year and test site were condensed into 18 - 20 time points of equal intervals. The number of foci observed at an individual time point was divided by the total cumulative foci for that epidemic, and was presented as percent of final disease. Percent of final disease was used to model each progress curve because turfgrass diseases differ from other pathosystems. Susceptible tissue on a normal leaf is limited by the size of the leaf and leaf area can be assessed for disease. Turfgrass is different because disease affects many blades of grass at one time but individual blades are not assessed independently and the diseased tissue is replaced by new foliage or removed through mowing. Dollar spot was not rated for severity, a function of area infected compared to the total area that could have been infected. To plot the disease progress in a manner that accommodated the analysis software, the total cumulative spots for each green was used as 100 % infection, representing 100 % disease severity. Then, each day during the epidemic was expressed as percent of final disease and used to model the progress curve. The model with the highest coefficient of determination (R^2) was considered the best fit for the disease progress curve. Actual,

predicted and linearized curves were graphed.

4.4 RESULTS

4.4.1 Symptom Expression

Six epidemics over three years were used to characterize symptom development of dollar spot on turfgrass in southern Ontario. Dollar spot epidemics began on 14 June 1996, 17 June 1997, and 22 May 1998 at the GTI. During 1997 and 1998, the epidemic began when irises and peonies were in bloom and when lilacs were in full- to late-bloom. The first symptoms noted were two or more blades of grass with white, necrotic tissue usually at the leaf tip, but occasionally on the leaf surface. The bleached lesion was commonly surrounded with a reddish-brown border. A small amount of mycelia was present on, or growing from, initial diseased tissue on 17 June 1997 and 26 May 1998. Blades of grass exhibiting dollar spot symptoms were sampled during 1997 and 1998. Mycelia developing from surface sterilized tissue on PDA were white and floccose, and quickly grew across the Petri dish. After 10 - 14 days, a flat, black, stromal layer developed throughout the medium. Cultures matched descriptions in the literature and of previously collected *S. homoeocarpa* isolates. Fruiting structures and spores were not observed because *S. homoeocarpa* rarely sporulates; therefore, the morphological characteristics of the colony were used to conclude that diseased tissue was caused by *S. homoeocarpa*. Additional organisms cultured from the diseased grass samples included *Bipolaris* sp., a genus common in the phylloplane, and the causal agent of *Helminthosporium* sp. leaf spot or melting-out (Couch 1995, Smiley 1992). *Helminthosporium* sp. was not deemed the causal agent of blighted turfgrass because

symptoms of melting-out did not develop. *Drechslera eruthrospila* (red leaf spot) was cultured from a tan-colored lesion on a blade of grass. While red leaf spot and dollar spot lesions were of similar appearance, the sampling sites exhibited only dollar spot symptoms and red leaf spot did not develop. Fusarium patch did not affect the greens, though two samples were colonized with *Fusarium* spp. Dollar spot foci three cm in diameter were apparent six days after samples were collected on 16 June 1997. More isolates were collected from dollar spot foci on 15 July 1997, all of which yielded fungal colonies with typical *S. homoeocarpa* morphology. During the three years of study, no apothecia of any kind were observed in, or on, turfgrass or thatch near dollar spots. Cores of turfgrass infected by *S. homoeocarpa* were collected when mycelia was actively growing, were placed in plastic bags to increase humidity and incubated in the laboratory at approximately 22 °C. No apothecia were present and no apothecia developed during incubation.

Turfgrass was assessed for the presence of mycelia on dollar spot foci on 65 summer mornings during 1996, 1997 and 1998. Mycelia was actively growing on 37 days, whereas mycelia was not observed on 28 days. Often, mycelia were present after rain or periods of high relative humidity (e.g., RH \geq 85 %). Upon close analysis, mycelia almost always originated from diseased tissue and grew aeriually to healthy blades of turfgrass. One exception was mycelial growth on Kentucky bluegrass (*Poa pratensis*) with no visible necrotic tissue, but when cultured produced *S. homoeocarpa*. Typical dollar spot symptoms developed on the Kentucky bluegrass a few days later (Figure 4.5). On 6 September 1996, mycelia in contact with green foliage caused a water-soaked appearance, as described by Couch (1995), Smiley et al. (1992), and Smith (1955). The presumed infected tissue was greyish-green without a reddish-brown lesion border. Mycelial growth causing water-soaked

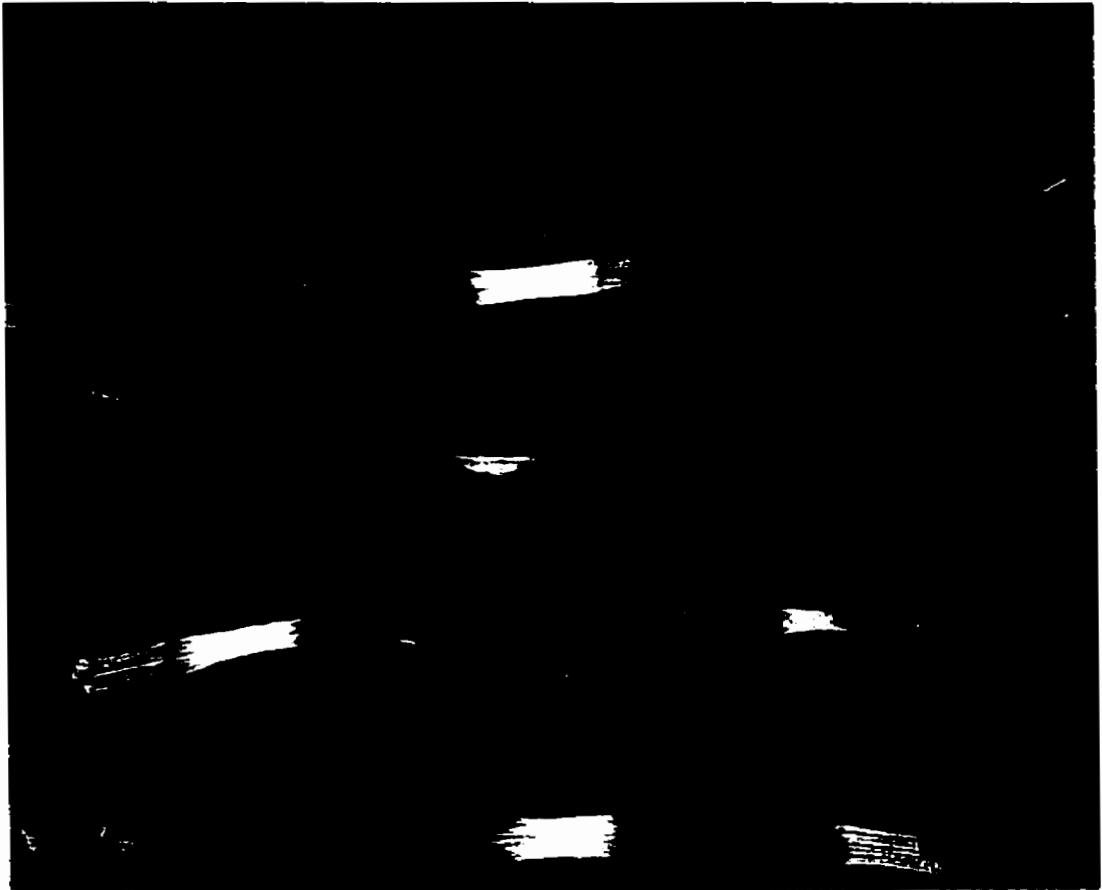


Figure 4.5 Symptoms of dollar spot on Kentucky bluegrass.

lesions was also noted on 9 July 1998. Grass exhibiting these symptoms was marked with golf tees in the morning. The day was sunny and warm and the next day, the water-soaked lesions had desiccated to form typical bleached tissue of dollar spot. During 1996, 1997 and 1998, the location of mycelia growing on the turfgrass surface was noted in the morning following a warm, humid night. By the afternoon, water-soaked lesions had developed and the next day, typical dollar spot lesions were evident on the blades of grass. Therefore, a one-day lag period was assumed between the appearance of water-soaked tissue and dollar spot lesions.

At the Cutten Club in 1998, dollar spots were more common following periods of warm and humid or rainy weather conducive to disease development. The turfgrass foliage usually exhibited symptoms at the tips of the leaf blade. When daily temperature cooled and rainfall and relative humidity were low, the conditions favored turfgrass growth rather than the disease. Diseased turfgrass appeared to recover from disease and no longer showed dollar spot symptoms when the plant grew and dead leaf tips were removed by mowing. The influence of mowing on disease progression was most obvious after weekends because greens were not mowed and diseased tissue remained intact until Monday morning. Dollar spots would appear in the same section of the subplots when weather conditions once again favored disease. Diseased tissue and associated inoculum at the GTI and Victoria West Golf Course were forced into the soil when golf tees were pushed into the newly formed dollar spot focus; therefore, only new spots were visible and enlargement of foci was not recorded. Reappearing foci were evident at the Cutten Club because golf tees were not used for marking diseased foliage. Dollar spots at the Cutten Club were allowed to grow naturally resulting in foci approximately 3 - 5 cm in diameter that developed throughout the summer.

Smiley et al. (1992) stated that mycelia of *S. homoeocarpa* were often confused with spiderwebs that collected dew on the surface of golf greens. Two types of “mycelial” growth were observed on the heavily dewed turfgrass on 19 June 1997. The difference was that mycelium growing from the dollar spot was <0.5 cm in length, was floccose and white in color, and obviously originated from diseased turfgrass. The other type (true spiderwebs) was not associated with diseased tissue, was finer and extended to a diameter of >2 cm. To discriminate between mycelium and spiderwebs, samples were collected from both types of structures on 15, 16, 31 July, and 12 and 27 August, 1997. The samples were collected by entangling strands of web or mycelia with an autoclaved dissecting pin or pipe-cleaner. The spiderweb-like structures, when plated to selective media or viewed under the microscope, only contained airborne spores of *Penicillium* spp., *Bipolaris* spp. and *Alternaria* spp. On 24 August 1997, there was a spider crawling on the spiderweb. Mycelia sampled from diseased tissue always resulted in cultures of *S. homoeocarpa* and only mycelia sampled from dollar spot foci gave rise to *S. homoeocarpa* cultures. The strands of spiderweb and mycelia were different in appearance: both were white in color, however, spiderwebs were much finer than mycelia of *S. homoeocarpa* and dew collected on the spiderwebs, whereas no free water was apparent on the surface of mycelia.

4.4.2 Disease Progress Curves and Epidemic Modeling

Assessments for dollar spot began on 4 July 1996 at which time the spots were >2 cm in diameter indicating that the epidemic began earlier. The Turfgrass Specialist at the GTI noticed spots on 14 June 1996 (P. Charbonneau, *personal communication*). Disease progressed on the pathology green until 22 August, at which time the 5th replication of the

test plot was 100% diseased and no new spots were distinguishable. However, the epidemic remained active outside the test plot until after 4 September. The native green epidemic began on 3 July 1996 and dollar spot activity had not subsided upon the conclusion of the study on 20 September. Disease incidence was greater on the pathology green than on the native green; cumulative foci totaled 7,987 on the pathology green on 4 September compared to cumulative spots of 1,728 on the native green on the same date. The disease progress curves for both the pathology and native greens in 1996 are presented in Figure 4.6.

Dollar spots were first noticed on the plot on 17 June 1997. The number of foci on the native green was 319 by 22 August, at which time this epidemic was no longer monitored because the turfgrass was physiologically stressed due to poor management. Cumulative foci on the pathology green increased to 2,187 by 3 October. The disease progress curves for both the pathology and native greens in 1997 are presented in Figure 4.7.

The dollar spot epidemic began at the GTI on 22 May 1998. During April and May, temperature was high with little precipitation. The GTI received only 76 mm of rainfall from 4 April to 20 June and greens were not irrigated until the later part of May causing the pathology green to die. As a result, the dollar spot epidemic did not progress on test plots, and the experiment was relocated to the Cutten Club and Victoria West Golf Course. The disease progress curves for both the Cutten Club and Victoria West Golf Course in 1998 are presented in Figure 4.8. Disease progress curves were analyzed using EPIMODEL to fit five temporal population growth models used commonly in the analysis of plant disease epidemics (i.e., monomolecular, exponential, logistic, Gompertz and linear). The model with the highest coefficient of determination (R^2) was accepted as the best of fit to the epidemic disease progress curve (Table 4.1). Epidemics at the GTI during 1996 (Figure 4.9) and

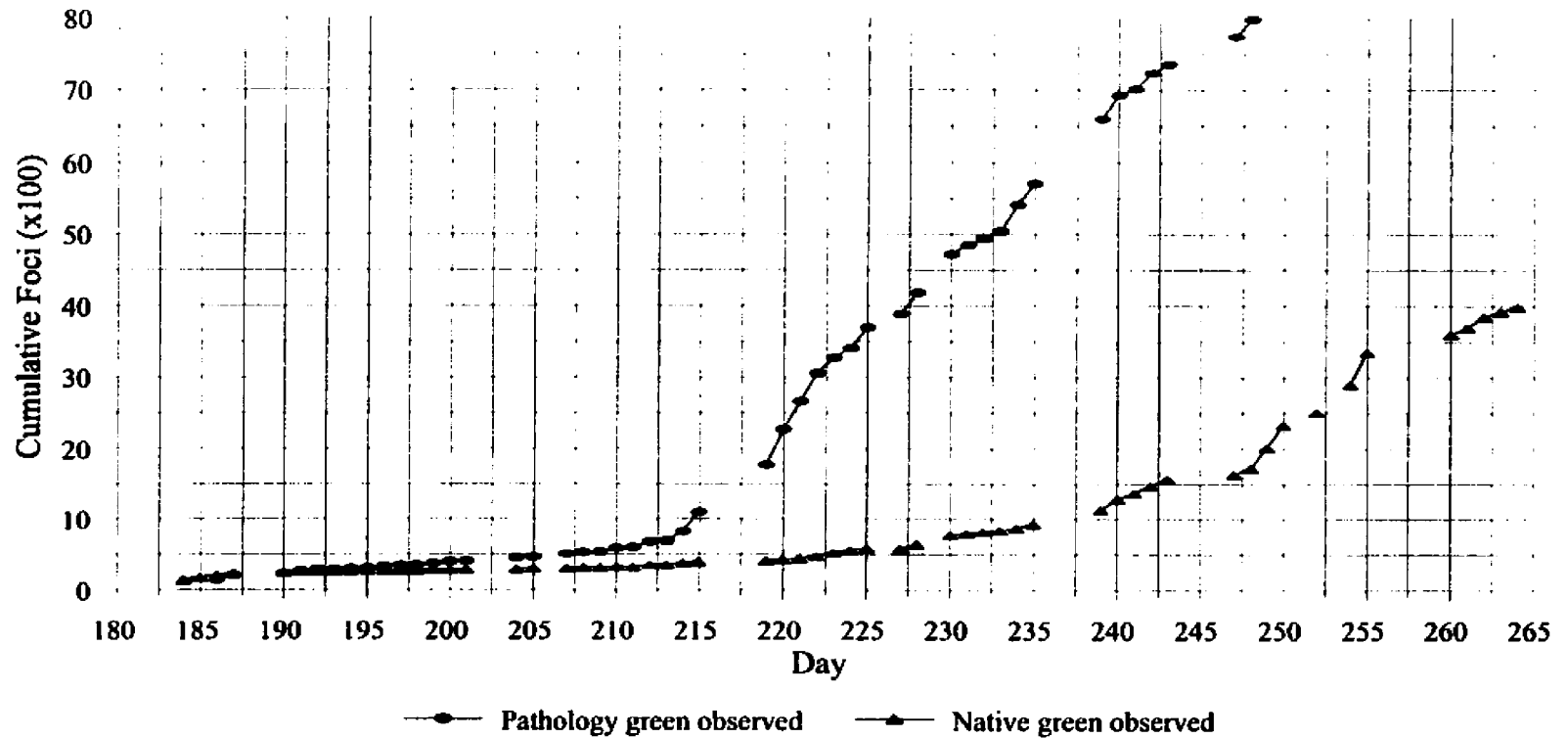


Figure 4.6 Disease progress curves for dollar spot of creeping bentgrass on the pathology and native greens at the Guelph Turfgrass Institute during 1996. Disease is measured as cumulative dollar spot foci, days with no observations are blank points of the progress curve, and day represents Julian date (e.g., Day 185 = 3 July).

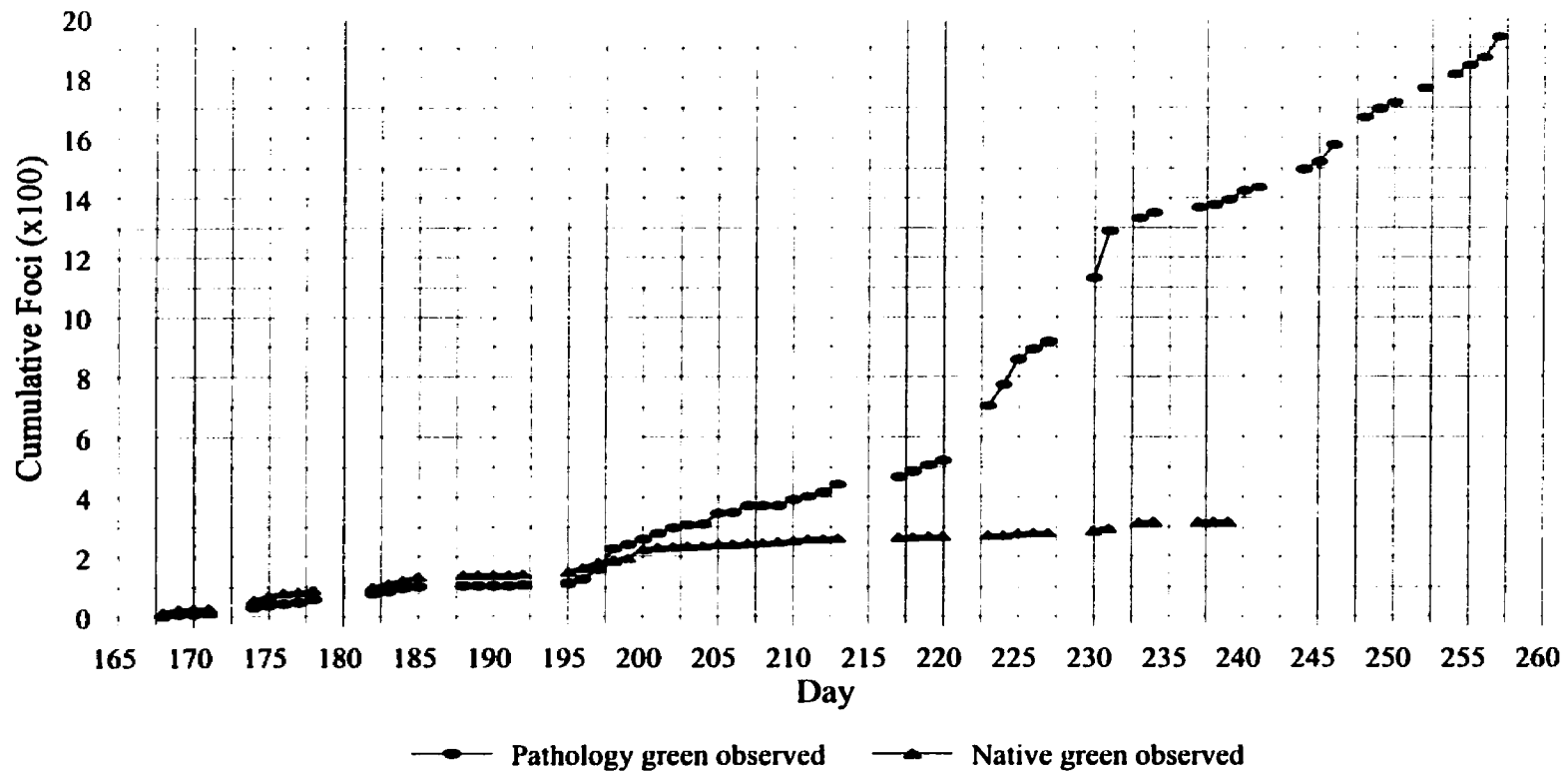


Figure 4.7 Disease progress curves for dollar spot of creeping bentgrass on the pathology and native greens at the Guelph Turfgrass Institute during 1997. Disease is measured as cumulative dollar spot foci, days with no observations are blank points of the progress curve, and day represents Julian date (e.g., Day 168 = 17 June).

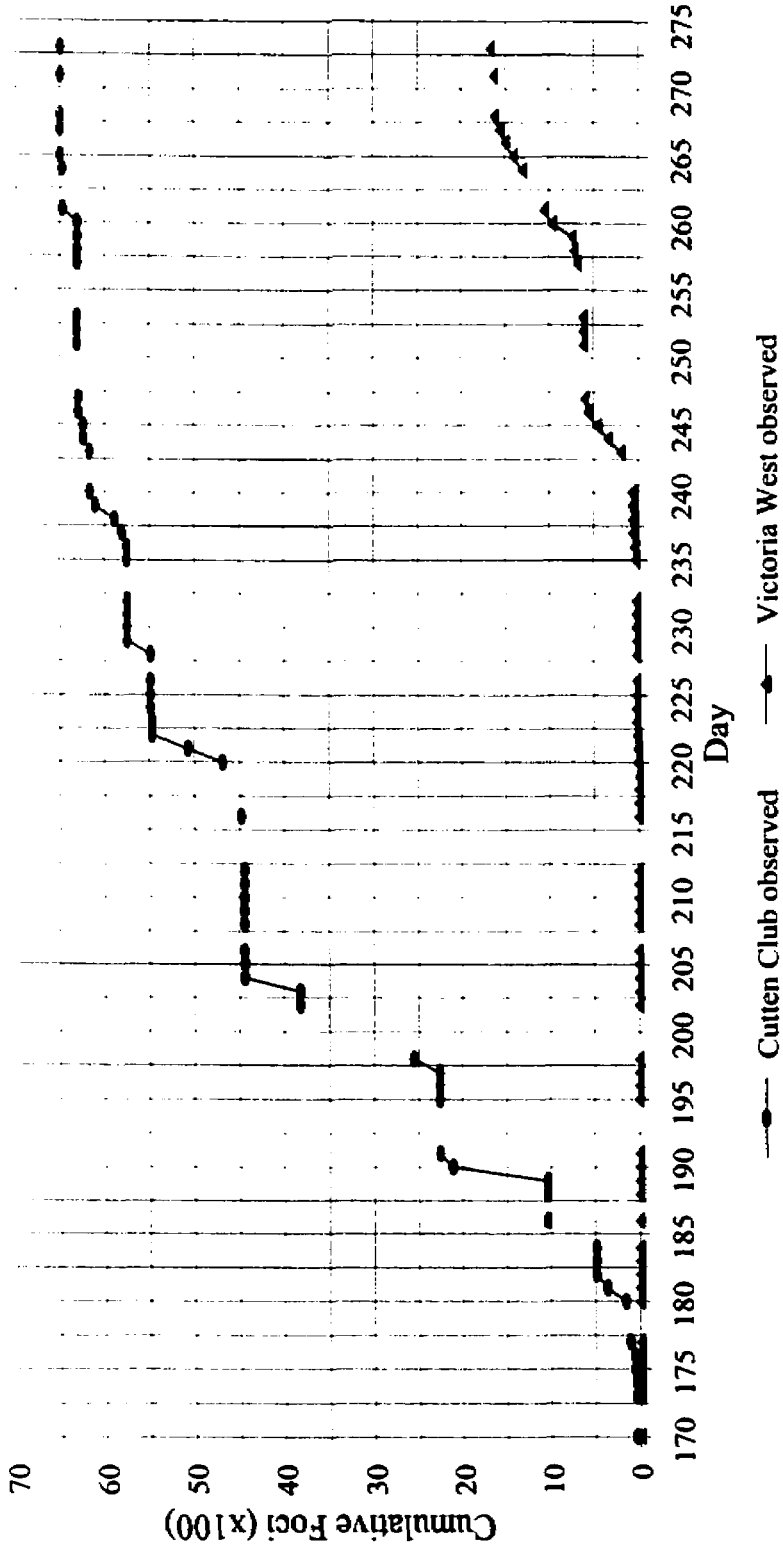


Figure 4.8 Disease progress curves for dollar spot at Victoria West Golf Course and the Cutten Club during 1998. Disease is measured as cumulative dollar spot foci, days with no observations are blank points of the progress curve, and day represents Julian date (e.g., Day 170 = 19 June).

Table 4.1 1996 - 1998 cumulative foci, number of assessments included in the progress curve, R^2 values and mean square error for disease progress curves of *Sclerotinia homoeocarpa* compared to population growth models

	1996				1997				1998			
	Pathology Green		Native Green		Pathology Green		Native Green		Victoria	Cuttan		
Final Disease	7743		3994		1938		319		1671	6496		
Non-zero points	20		20		19		18		19	20		
Model	R^2 *	\sqrt{MSE}	R^2	\sqrt{MSE}	R^2	\sqrt{MSE}	R^2	\sqrt{MSE}	R^2	\sqrt{MSE}	R^2	\sqrt{MSE}
Gompertz	0.786	0.829	0.567	1.060	0.796	0.798	0.862	0.584	0.715	1.135	0.979	0.300
Exponential	0.956	0.279	0.954	0.211	0.927	0.429	0.757	0.392	0.948	0.497	0.663	1.006
Logistic	0.907	0.772	0.724	1.119	0.938	0.660	0.907	0.596	0.898	1.099	0.937	0.772
Monomolecular	0.635	0.837	0.423	0.990	0.601	0.850	0.778	0.624	0.521	1.097	0.933	0.417
Linear	0.901	0.116	0.748	0.153	0.917	0.103	0.949	0.069	0.789	0.171	0.899	0.123

* Model with the highest R^2 value was determined the model that best fit the disease progress curve.

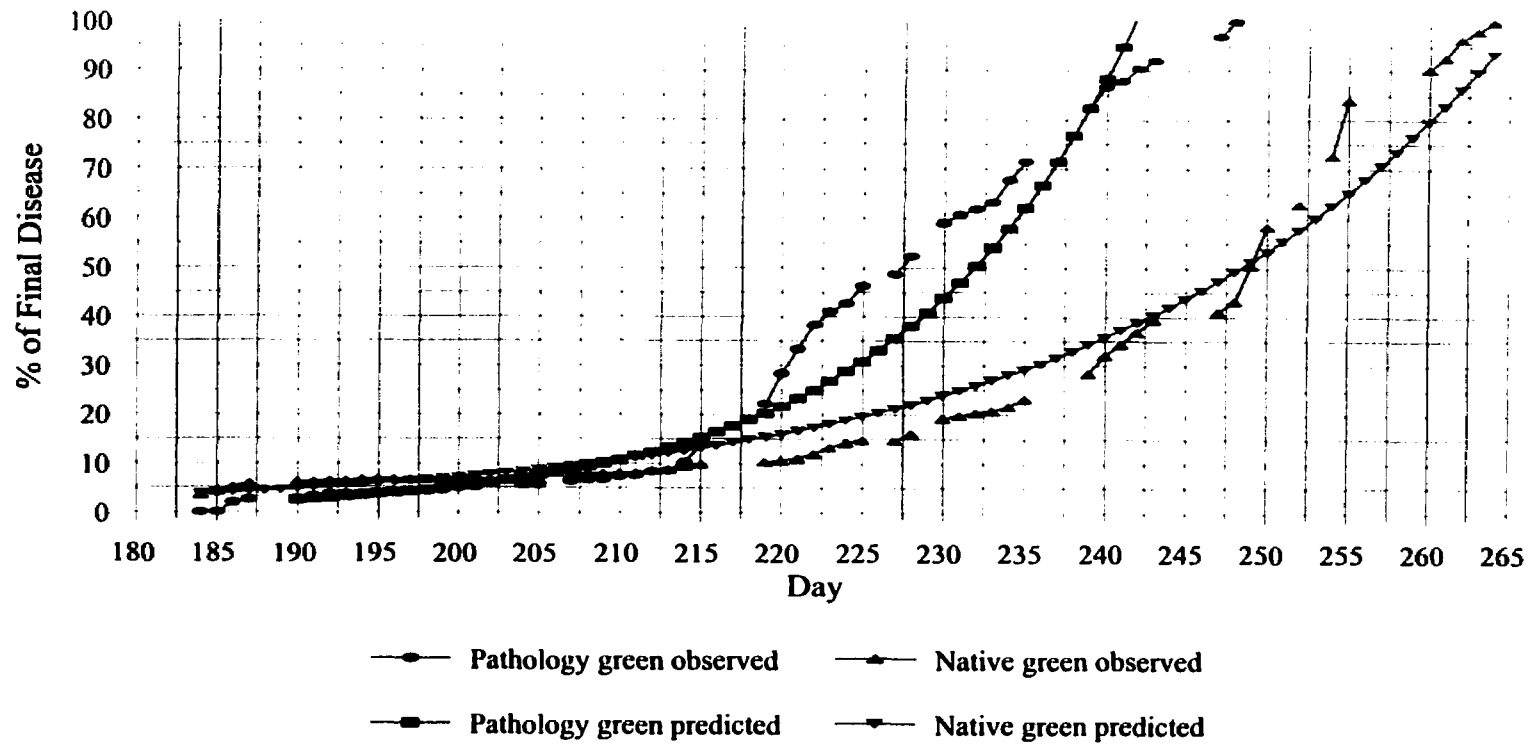


Figure 4.9 1996 percent of final disease for the pathology green and native green. Predicted disease for the pathology green was calculated using the exponential model: percent disease = $y_0 \exp^{rt}$ where y_0 is the initial inoculum (0.0266758), r is the rate of disease progress (0.07) and t is the time, in days, from the start of the epidemic. Predicted disease for the native green was calculated using the same model with $y_0 = 0.0381206$ and $r = 0.04$.

Victoria West Golf Course (Figure 4.11) were best described by the exponential model originally proposed by Vanderplank (1963). In 1997, the native green epidemic was best fit by the linear model while the pathology green was best fit by the logistic model (Figure 4.10). The Cutten Club disease progress curve was best described by the Gompertz model (Figure 4.11). Linearized transformations of disease progress curves and predicted values are illustrated in Figures 4.12 - 4.14. The 1997 native green epidemic was not transformed because it was already linear.

4.5 DISCUSSION

Dollar spot epidemics occurred at each field site during 1996, 1997 and 1998. The disease was first noticed as two or more blades of grass with necrotic, white tissue, usually at cut tips, but occasionally on the leaf surface. Spores and colonies that arose from cultures of diseased samples included *S. homoeocarpa*, *Fusarium* spp., *Bipolaris* spp. and *Drechslera eruthrospila*; however, *S. homoeocarpa* was the only pathogen that caused symptoms.

Spiderwebs on dew-covered turfgrass can be confused with mycelia of *S. homoeocarpa*, leading to misdiagnosis of pathogen presence, disease incidence, and unnecessary fungicide application. Sampling of different mycelia-like structures confirmed that mycelia of *S. homoeocarpa* only grew from diseased foliage, except for one occasion noted on Kentucky bluegrass. *Sclerotinia homoeocarpa* mycelium was robust and white, and did not collect free water on its surface. In contrast, spiderwebs were not associated with diseased foliage, were fine and clear in color, and did collect dew on each filament. Sampling from both spiderwebs and mycelia proved that only mycelia gave rise to cultures

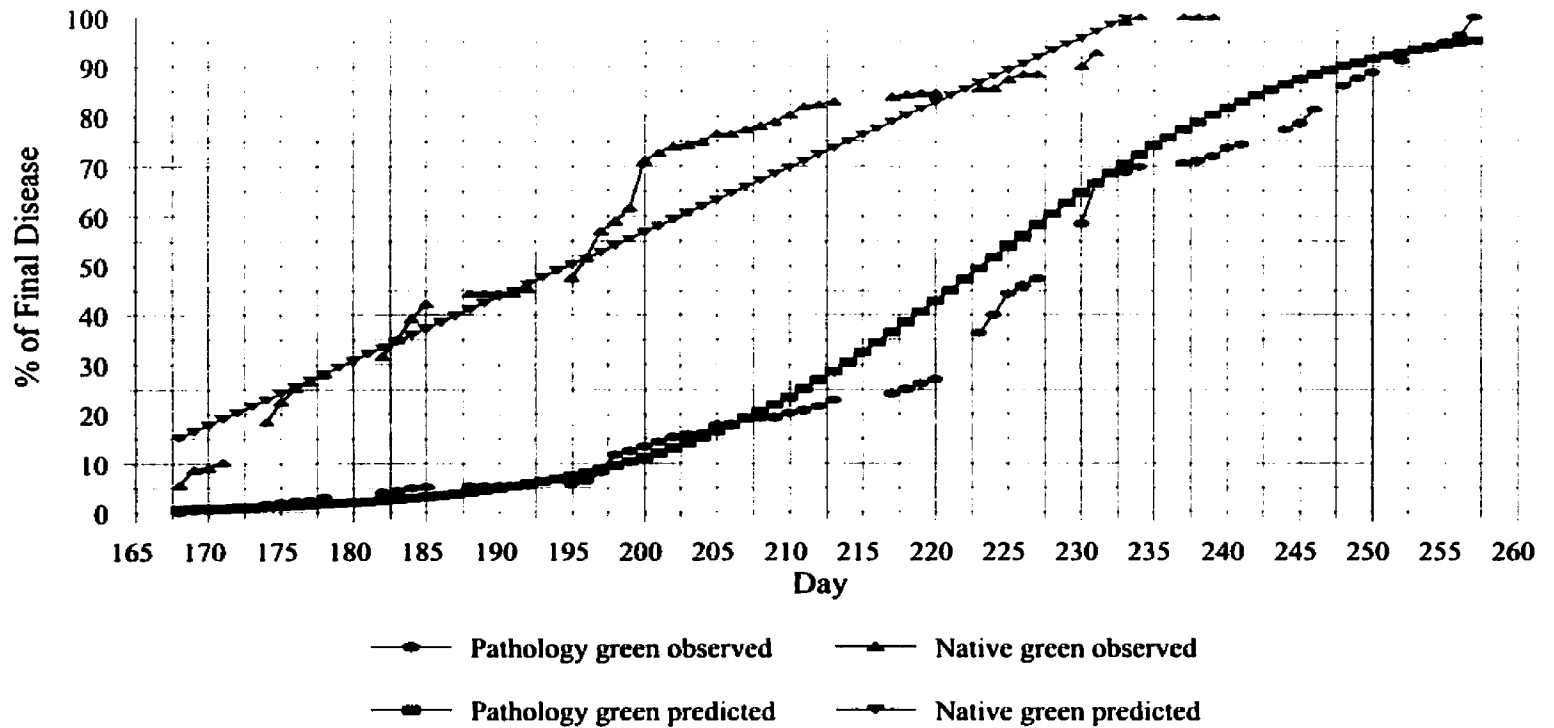


Figure 4.10 1997 percent of final disease for pathology and native greens. Predicted disease for the pathology green was calculated using the logistic model: $\text{percent disease} = 1 / \{1 + [(1 - y_0) / y_0] \exp^{-rt}\}$ where y_0 is the initial inoculum (0.006667), r is the rate of disease progress (0.089) and t is the time, in days, from the start of the epidemic. Predicted disease for the native green was calculated with the linear model: $y = y_0 + rt$, where $y_0 = 0.139$ and $r = 0.013$.

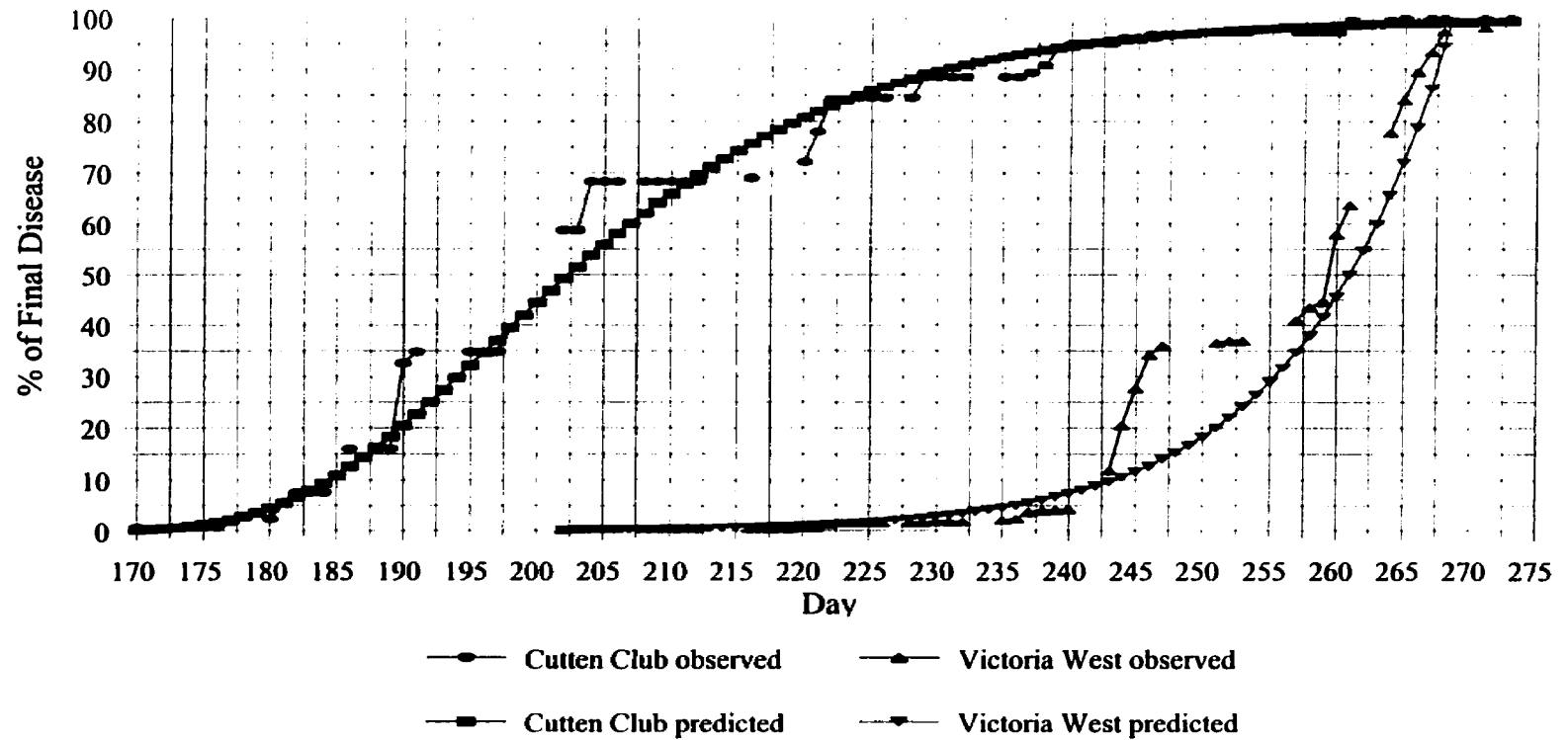


Figure 4.11 1998 percent of final disease for the Cutten Club and Victoria West Golf Course. Predicted disease for the Cutten Club was calculated using the Gompertz model: percent disease = $\exp \{[\ln(y_0)] \exp^{-rt}\}$ where y_0 is the initial inoculum (0.0009975), r is the rate of disease increase (0.067) and t is the time, in days, from the start of the epidemic. Predicted disease for Victoria West Golf Course was calculated with the exponential model: percent disease = $y_0 \exp^{rt}$, where $y_0 = 0.0021292$ and $r = 0.091$.

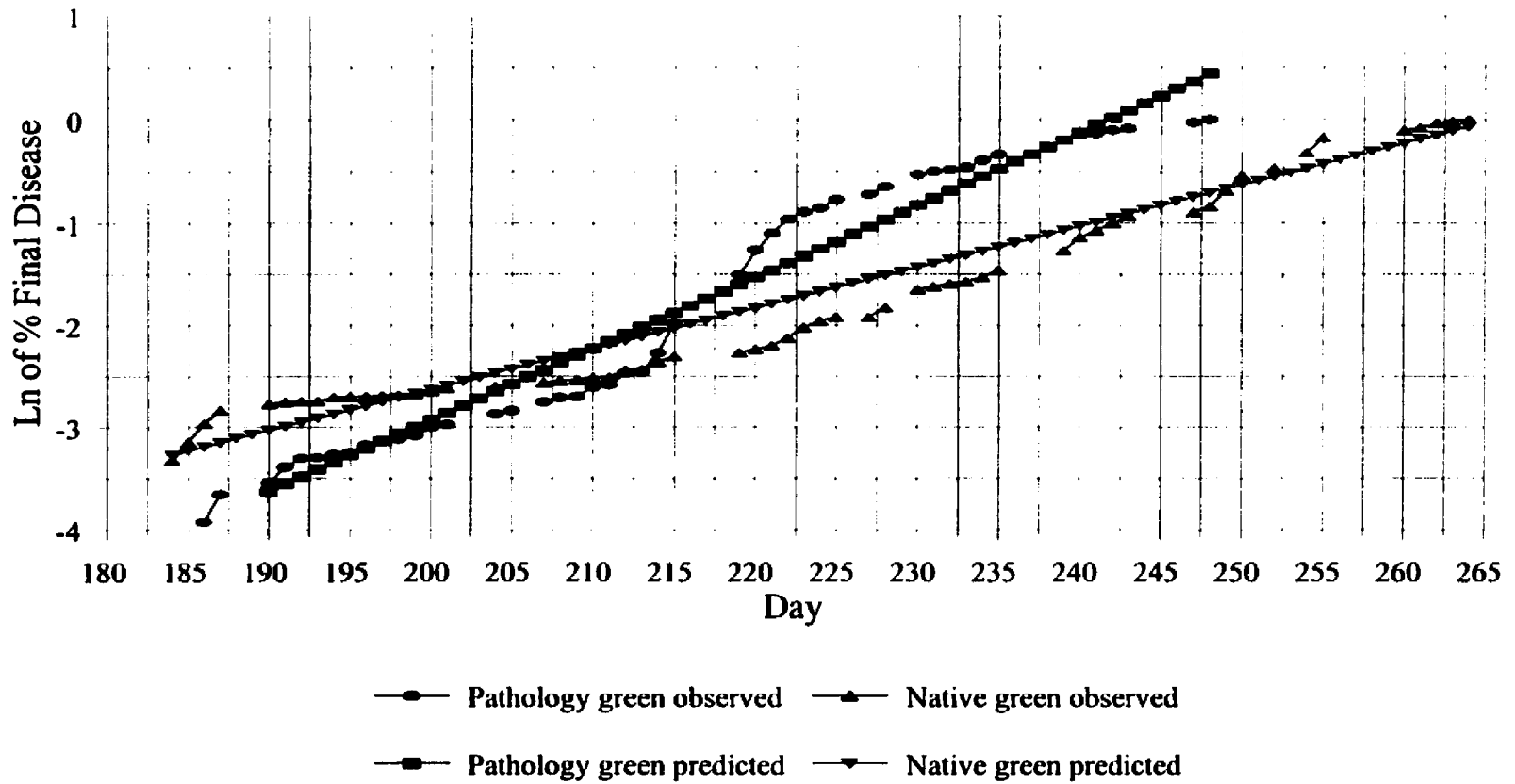


Figure 4.12 Linearized disease progress curves for dollar spot on creeping bentgrass for the pathology and native green epidemics during 1996.

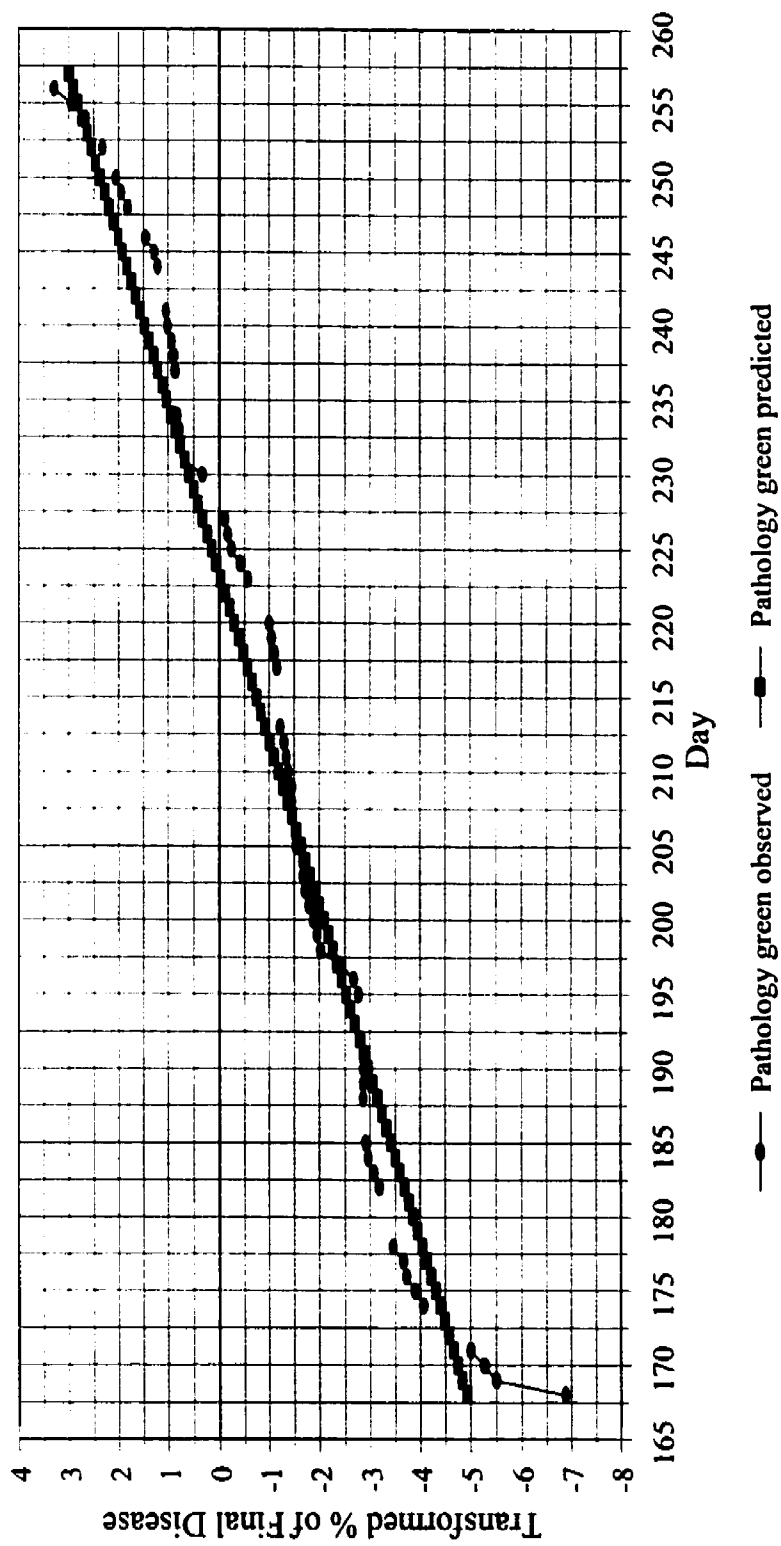


Figure 4.13 Linearized disease progress curve for dollar spot on creeping bentgrass for the pathology green epidemic during 1997.

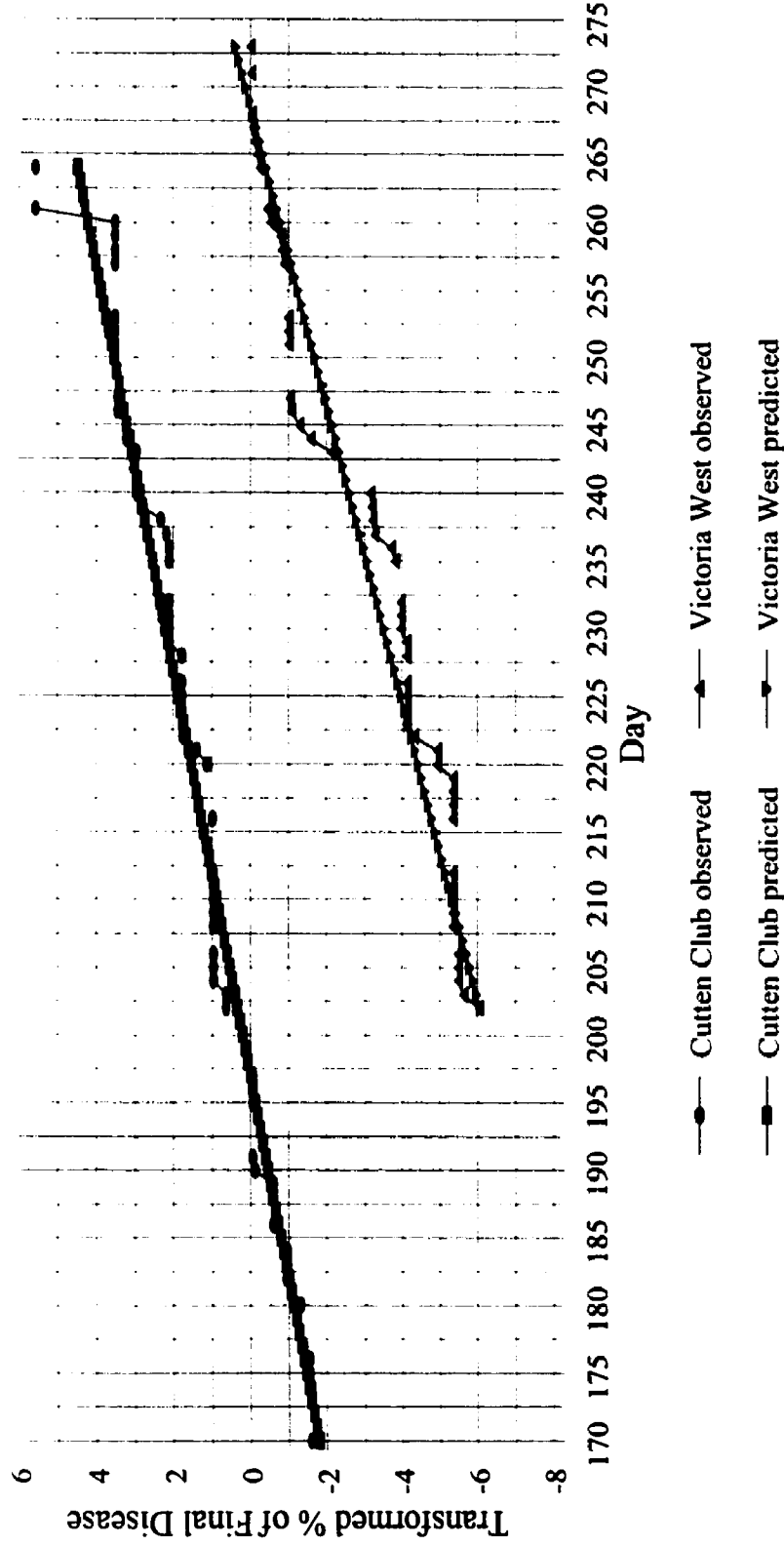


Figure 4.14 Linearized disease progress curves for dollar spot on turfgrass for the Cutten Club and Victoria West Golf Course epidemics during 1998.

of *S. homoeocarpa*, whereas the spiderwebs gave rise to colonies of commonly occurring, sporulating fungi.

Dollar spot symptoms progressed at Guelph sites in the same manner described by Smiley et al. (1992) and Smith (1955). The first symptoms of disease were water-soaked lesions on mornings when mycelia were present. Then, the lesions turned white and usually developed a reddish-brown border as the dew dried and sunlight heated the turfgrass during the day. Mycelia likely grew during the night when weather conditions were favorable for dew formation, especially since no mycelium was noted on the foliage during the previous day. Water-soaked tissue appeared to be the result of mycelia in contact with the foliage, which was presumed to have infected the tissue. Therefore, a one-day lag period was assumed between infection and the development of white, dollar spot lesions. There was a relationship between mycelia growth and disease because dollar spots arose in the same location where mycelia were observed previously. However, new dollar spots were noted on days when mycelia were not apparent, which indicated aerial mycelia were not critical to symptom development.

Turfgrass is a unique host because it is perennial and because diseased foliage at the tips of the plant are removed by mowing. Disease was more apparent on Monday mornings and during September when the grass was mown less frequently. This three-year study supported the hypothesis of Britton (1969), Couch (1995), Smiley et al. (1992) and Fenstermacher (1980) that stroma and/or mycelia of *S. homoeocarpa* survive locally in turfgrass crowns or foliage. Dollar spots appeared in the same subplots as they were noted during the epidemic, and in previous years, as if the fungus was residing at the base of the plant until it caused symptoms on the entire plant. There were no means to quantify initial

inoculum because stroma were not observed in association with these reappearing spots. Inoculum may reside in close proximity to the perennial host, ready to infect at the beginning of the season. Dollar spots multiplied quickly, and consequently, the disease was very destructive to the turfgrass, as seen on five subplots of the pathology green in 1996 where new foci were indistinguishable because the area was consumed by disease. At the Cutten Club, spots likely disappeared as the result of weather conditions favoring the plant rather than the pathogen, and because the diseased tissue was mowed away. The spots reappeared when weather conditions changed to warm, moist weather that favored fungus growth and infection. The observations suggest that turfgrass may be infected or colonized by *S. homoeocarpa* without continuously expressing symptoms.

Disease progress curves for dollar spot epidemics were compared to five temporal population growth models used in plant disease epidemiology. The monomolecular model did not correlate with disease progression. Monomolecular disease progression is associated with simple interest diseases as explained by Vanderplank (1963). Monomolecular diseases have a reservoir of initial inoculum that causes new infections, but these do not produce propagules to cause other infections during the same season. The monomolecular disease progress curve decreases in rate with time. *Sclerotinia homoeocarpa* may have a reservoir of initial inoculum in the turfgrass crowns, thatch or soil. This would be the only source of inoculum for the season if it were a monomolecular or simple interest disease, and the disease would start quickly and the rate would decline over time. The disease progress curves showed the opposite; the rate of disease progress increased through out the epidemic and subsided near the end. However, there were no spores for secondary inoculum associated with *S. homoeocarpa*, therefore, another propagule must be the source for

secondary infection. The mycelia that grow from diseased tissue are most likely the source of secondary inoculum. This secondary inoculum was difficult to quantify because mycelia were not always visible, even when new infections appeared. Also, one mycelium unit may be sliced and distributed by movements on the green resulting in more than one infection. Finally, the initial inoculum in the crowns, thatch or soil could contribute to new infections throughout the growing season, thus exacerbating the dollar spot epidemic.

This was the first study that characterized dollar spot epidemics. The exponential model was most appropriate population growth model to describe the 1996 epidemics at GTI and 1998 epidemic at Victoria West Golf Course. The exponential model fit the compound interest theory proposed by Vanderplank in 1963. In monetary terms, compound interest means the interest earns interest; in dollar spot terms, foci give rise to new dollar spot foci during the same season. Additionally, one infection unit of mycelium may infect more than one blade of grass. The rate of new foci appearance increased for the duration of the epidemic, and there were no apparent limiting factors to stop the increasing rate of infection. Normally, the rate of dollar spot increase is limited because sites to infect become occupied (e.g., the entire leaf is diseased). However, during the 1996 and Victoria West Golf Course epidemics there were factors that allowed the disease epidemic to continue to increase in rate. The turfgrass was able to replenish its foliage through growth and because of mowing of diseased tissue. Foliage on the plots at Victoria West Golf Course remained available for infection because the epidemic started late in the season. Perhaps the symptoms started late at Victoria West Golf Course because the green was young and initial inoculum was not well established. The most influential factor was that plots were not monitored to the completion of the epidemic. Perhaps, if the study was conducted until later in the season, a rate decrease

would have been recorded in the disease progress curve.

The 1997 native green disease progress was linear. Disease progress was interrupted on the native green because the grass was stressed by factors other than disease, thus the study was terminated. A different model might have described the curve if more host tissue was available for infection and the epidemic progressed to the end of the season. The pathology green disease progress curve was best described by the logistic model, and for many of the other epidemics, the logistic model was the second best to describe disease progress curves. The logistic model accounted for the lack of unoccupied sites limiting the rate of infection after half of the host tissue was diseased. The typical S-shaped, sigmoidal curve with an inflection point at 50 % disease associated with the logistic model is evident in Figure 4.10 illustrating the 1997 pathology green epidemic. Rate decrease was noted because the epidemic was studied until later in the season during 1997. The Cutten Club epidemic also had a S-shaped disease progress curve; however, the point of inflection (approximately 37 % disease) was closer to the epidemic start. Thus, the Gompertz model fit best and the logistic was the second best fit. The number and size of dollar spot foci rapidly increased at the Cutten Club and blighted a great deal of turfgrass at the start of the epidemic, between 16 June and 23 June (day 197 and 204). The rate of disease then decreased, most likely due to insufficient tissue to infect. Golf tees were not used at the Cutten Club to mark foci because the tees interfered with play, but at all other sites each new focus was marked with a golf tee. New spots were easily distinguished at the GTI and Victoria West Golf Course, but not at the Cutten Club. New foci probably developed at the Cutten Club; however, they were not recorded in the disease progress curve.

As mentioned previously, plant disease epidemics may be classified as simple or

compound interest diseases based on the concept proposed by Vanderplank (1963). A simple interest disease relies on the primary inoculum and has insignificant secondary inoculum. An example of a simple interest disease is white mold of bean caused by *S. sclerotiorum*. Carpogenic germination of sclerotia happens throughout the growing season, but the infection is greatest when the crop canopy closes and bean plant flowers (Boland & Hall 1987, 1988). The primary disease cycle occurs when ascospores infect senescent flowers that drop onto stems and pods to cause lesions. Secondary infections contributed only 10 to 12 % of overall disease (Boland 1984) through plant-to-plant mycelial growth (Abawi & Grogan 1979). Dollar spot, however, appears to be a compound interest disease. Disease progress curves were best fit to exponential or logistic models, both of which describe compound interest diseases. *S. homoeocarpa* rarely produces spores for primary or secondary inoculum. However, dollar spots give rise to new foci because the mycelia produced from lesions are physically moved when the green is mowed or walked on, or the mycelia grows to adjacent turfgrass. Each piece of mycelium or infested host tissue is a potential colony forming unit that leads to new infections. Use of control measures during the epidemic, such as fungicides, will reduce the secondary inoculum and thus reduce the rate of disease progress. If initial inoculum is controlled, the start of disease progress will be delayed and not reach the magnitude of disease of an untreated site. The *S. homoeocarpa* population should be controlled late in the epidemic to reduce the amount of inoculum that over-winters as stroma at the crowns of the turfgrass, ready to infect the plant when environmental conditions are favorable. The disease progress curve for the pathology green in 1997 was compared to the same system if the initial inoculum is reduced, the rate of disease progress is slowed, or a combination of both (Figure 4.15). Dollar spot can be

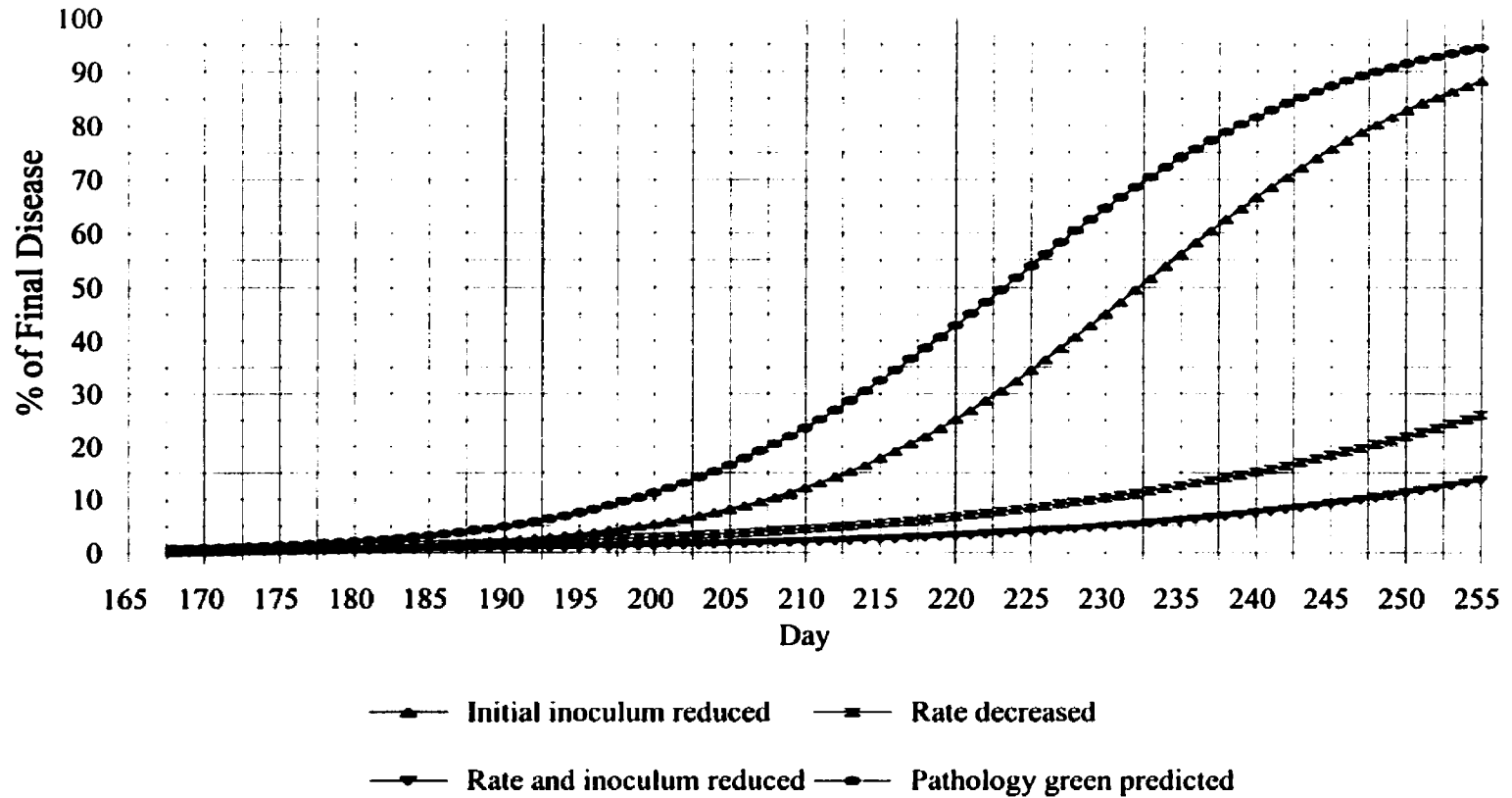


Figure 4.15 Predicted disease progress curve for dollar spot on creeping bentgrass for the pathology green during 1997, the effect of reducing initial inoculum ($y_0 = 0.003$) and/or decreasing the growth rate ($r = 0.0045$).

controlled by two processes: reduction of initial inoculum by compromising the overwintering population and timing the first fungicide application to delay the start of disease and use of control measures to stop secondary spread and slow the rate of disease progress.

5. DEVELOPMENT AND ASSESSMENT OF DOLLAR SPOT PREDICTION MODELS IN SOUTHERN ONTARIO

5.1 ABSTRACT

In this study, several approaches to designing disease prediction models for dollar spot of creeping bentgrass in southern Ontario were developed and assessed. At present, the decision to start fungicide applications for dollar spot is made through scouting of the golf course or is based on a calendar date. Predicting the start of dollar spot appearance on golf courses using this proposed model of accumulation of 9 - 10 days, after May 1, with mean air $T \geq 16$ °C can be easily calculated from local air T data. The proposed model is necessary because diagnosis of dollar spot at the single lesion stage is difficult and it is at this stage, or before, that control measures are most effective in managing epidemic progress. The Mills & Rothwell and Hall models proposed during the 1980s were tested against disease progress curves produced during 1996 - 1998, but these models were not predictive of dollar spot increases. Step-wise multiple regression did not detect that increases in dollar spot during the epidemic were correlated to weather variables. This study supports the literature that there are biotypes of this fungus capable of pathogenicity at a broad temperature range. Receiver Operating Characteristic (ROC) curve analysis was used to establish a decision-threshold for fungicide application. Use of decision-thresholds, based on the models developed in this study, resulted in an over-prediction of disease; thus, disease control would be a function of over-prediction rather than the ability to correctly predict dollar spot increases and treat appropriately. However, this study provided a method to predict the start

of the epidemic, thus allowing for application of fungicides just as the epidemic is beginning, resulting in benefit for the turfgrass industry.

5.2 INTRODUCTION

In southern Ontario, dollar spot is prevalent from June until October, when daily mean air temperature (T) is $>15\text{ }^{\circ}\text{C}$, and reaches maximum pathogenicity when mean air T is $21 - 27\text{ }^{\circ}\text{C}$ (Couch 1995, Endo 1963, Sears et al. 1996). Endo (1963) grew *S. homoeocarpa* on potato dextrose agar (PDA) under controlled temperatures and observed that the fungus reached its maximum growth rate at $26.8\text{ }^{\circ}\text{C}$. Endo and Malca (1965) found that *S. homoeocarpa* produced a root toxin when air T exceeded $15.5\text{ }^{\circ}\text{C}$. Periods of high relative humidity (RH) $\geq 85\%$ increase disease and fungal growth, and RH $\geq 90\%$ has been used as an estimator of leaf wetness (Sutton et al. 1984). Mycelia of *S. homoeocarpa* can be seen growing from diseased tissue to healthy tissue on mornings following warm, humid nights (Smiley et al. 1992). In Chapter 3, a model was designed to predict the effect of T and leaf wetness duration (L) on dollar spot focus diameter in controlled environments. No disease resulted in the absence of leaf wetness or when T was $<10\text{ }^{\circ}\text{C}$ with 12 h of L. The greatest fungal growth and subsequent disease occurred when T was $25\text{ }^{\circ}\text{C}$ for 26 h. The model predicted that optimum T for pathogenicity was between 21 and $24\text{ }^{\circ}\text{C}$.

Dew also plays a role in dollar spot severity. Williams et al. (1996) noted that dollar spot was more prevalent when free water, in the form of dew, was on the leaf surface. Williams et al. (1996) tested several techniques for dew removal, and concluded that dollar spot severity was suppressed when dew was removed early in the morning. A common practice in the turfgrass industry is to pole or syringe dew from golf course greens and fairways. On some golf courses, dew duration is minimized by fans that circulate air above greens and when trees are removed that shade the greens or areas susceptible to dollar spot.

Disease forecasting systems for dollar spot were proposed by Mills and Rothwell (1982) and Hall (1984). In the Mills and Rothwell (M&R) system, a fungicide application was recommended when maximum air T was ≥ 25 °C and maximum relative humidity (RH) was ≥ 90 % during any three days of a seven-day period. In the Hall system, a fungicide application was recommended after two consecutive days of rainfall and a mean air T of ≥ 22 °C, or three consecutive days of rainfall and a mean air T of ≥ 15 °C. The accuracy of these two disease forecasting models was compared in a two-year study, but both models failed to predict weather-related increases of dollar spot (Burpee & Gouly 1986). The M&R model predicted too many infection periods, and disease suppression resulted more from the high frequency of prediction-based fungicide applications and not the accuracy of the model. The Hall system failed to predict sufficient infection periods, resulting in poor disease control. Boland and Smith (2000) used a preliminary dollar spot forecasting model in the EnviroCaster (NEOGEN Corporation, Lansing, MI) to time applications of biological agents. The model did not predict any infection periods during 1991 despite high levels of disease in all plots.

Deciding when to make a fungicide application is a difficult task; therefore, disease forecasting systems are designed to make the decision for the grower. Hughes et al. (1999) made use of the Receiver Operating Characteristic (ROC) curve analysis used by clinicians as a tool to evaluate diagnostic tests for decision making. In clinical practice, patients are separated into “cases” (those patients known to be diseased), and “controls” (those patients known to be free of disease). Then, the diagnostic test is evaluated for ability to correctly identify cases from controls. In most cases, the test has accuracy less than 100%, and does not provide perfect discrimination between cases and controls. There will be some cases that

are true positives, and some that are false negatives (i.e., the test stated the patient was healthy, when in fact the patient was diseased). There will be some controls that are correctly identified as true negatives, but error in the diagnostic test will cause some to be identified as false positives (i.e., the test stated the patient was diseased, when in fact the patient was healthy). The goal is to determine the threshold test score that should be adopted for implementation of treatment in a patient. In the case of a disease forecasting model, a decision- threshold must be established to denote when weather conditions will cause a level of dollar spot increase that warrants a disease management intervention (e.g., fungicide application). In addition, one must calculate the point at which the forecasting model identifies disease increases, with an acceptable level of error for predicting false positive or false negative results. The decision-threshold is calculated by plotting the ROC curve. This plot illustrates on the y-axis the probability of predicted disease above the threshold when the disease is actually above the decision-threshold (True Positive) versus the probability of the predicted disease being above the threshold in the absence of disease increase (False Positive) plotted on the x-axis. The probabilities are calculated over all possible decision-thresholds, and the appropriate decision-threshold with the greatest accuracy will correspond to the point of inflection in the curve. If the line of the ROC runs straight from 0 to 1, this is indicative of a disease forecasting model that does not provide a basis for discriminating between cases and controls. In other words, no decision-threshold can be predicted to indicate whether or not a control measure is required. An efficient model would yield a curve "pushed to the upper left corner" (Yuen et al. 1996). The ROC method for setting a decision-threshold also allows individuals to set their personal decision-threshold based on the amount of risk they can afford, whether that be the level of tolerance towards dollar spots

on greens or the cost of saving a fungicide application. The methods for calculating and plotting ROC curves are outlined in Hughes et al. (1999) and Yuen et al. (1996).

The objective of this study was to describe the effect of weather conditions on natural epidemics of dollar spot in southern Ontario, and to develop a preliminary model to predict the start of dollar spot epidemics and subsequent weather-related increases in disease. A final objective was to calculate a decision-threshold with the best accuracy at predicting dollar spot.

5.3 MATERIALS & METHODS

5.3.1 1996 and 1997 Field Sites

Research was conducted at the Guelph Turfgrass Institute (GTI), Guelph, ON, Canada (43°32.97 N, 80°12.90 W). Plots of 25 m² with 2 m borders were placed on two greens. The pathology green was a 1022 m² United States Golf Association (USGA) green built in 1993 using siliceous soil. The pathology green was primarily used for turfgrass disease research because it was sheltered from morning sunlight and wind which promoted long dew periods. The native sand green was an undrained “push-up” green constructed with sand available on-site (Anonymous 1994). Dew duration was less than on the pathology green because tree cover was less dense. Both greens were established in 1994 with creeping bentgrass (*Agrostis palustris* ‘Penncross’) stands (anonymous 1994). Grass was maintained at 4.8 mm, mowed 3 - 4 days per week, and irrigated to avoid drought stress. Plots were usually fertilized with half-rate nitrogen (N) (125 kg N/ha/year) to promote natural epidemics because N applications are associated with dollar spot recovery (Freeman 1969, Hoyland &

Landschoot 1993, Landschoot & McNitt 1997, Markland et al. 1969, Smiley et al. 1992). A full rate of N (50 kg N/ha) was applied on day 206 of 1996, and fungicides and biological control agents were applied to neighboring plots on day 247. In 1997, both greens were fertilized with full rate of N on days 190, 232 and 280. The native green was erroneously treated with an additional full rate of N on day 182. Artificial inoculations during previous research established populations of *S. homoeocarpa* on both greens. More inoculations of various *S. homoeocarpa* isolates occurred on the pathology green than on the native green, possibly contributing to greater disease pressure on the pathology green. No fungicides or herbicides were applied during the epidemics; however, late-fall applications of pentachloronitrobenzene at 360 g of formulated product / 100 m² (Scotts FFII, The Scotts Company, Marysville, OH) were used to control pink snow mold (*Microdochium nivale*).

5.3.2 1998 Field Sites

Plots were first established at the GTI, but were relocated on 15 June 1998 to the Cutten Club, Guelph, ON, Canada (43° 31.78 N 080° 13.37 W), and Victoria West Golf Course, Guelph, ON, Canada (43° 31.80 N 080° 11.59 W). Test plots on each golf course were located where there was a history of dollar spot occurrence. At the Cutten Club, the 2.5 × 10 m plot (25 m²) with a 2 m border was positioned on the north side of the ninth fairway. This was a mature turfgrass sward of approximately 70 % creeping bentgrass and 30 % annual bluegrass (*Poa annua* L.). The management regime was typical of industry standards: mowing three days per week to a height of 6.5 mm, clippings not removed, fertilization at regular intervals (days 203 and 228), irrigation to avoid drought stress, and morning dew removal. Iprodione (Rovral, Rhone-Poulenc Ag Company, Research Triangle Park, NC)

fungicide was applied to the adjacent fairway but not to the plot area.

The Victoria West Golf Course site was a 5 × 5 m plot (25 m²) positioned on the north side of a two-year-old creeping bentgrass nursery green. The management regime for the green included: mowing two days per week to a height of 6.5 mm (clippings removed), fertilization with half rate N, irrigation to avoid drought stress, and no fungicide.

5.3.3 Environmental Monitoring

Except when otherwise noted, all sensors, gauges, probes and control devices were obtained from Campbell Scientific, Inc. (Logan, UT). On each green, air T and RH were measured with a model HMP35C sensor at a height of one meter above the green surface (Figure 5.1). To reduce radiation affects, the probe was covered with a reflective shield and protected from direct sunlight and rain (Sutton et al. 1988). L was estimated by an electronic impedance grid (Model 237) positioned in the canopy of the adjacent Kentucky bluegrass. The micrologger was programmed to convert the resistance of each sensor to a value between zero and 100, zero being completely dry while 100 was completely wet. A threshold of 2.5% was determined to be the point when the grass was wet (Chapter 2). The model 237 sensor and its positioning in the Kentucky bluegrass was deemed the most accurate and precise method for estimating L on the green (Chapter 2). The leaf wetness sensors were moved only during weekly mowing of the Kentucky bluegrass maintained as rough play area next to the green. Soil T was recorded using a thermistor (Model 107B) buried in the soil 5 cm below the green. In 1996, irrigation and rainfall were recorded with a resolution of 0.5 mm using a tipping bucket rain gauge (Model RG2501) placed next to the native green. During 1997 and 1998, a tipping bucket rain gauge (TE525) with a resolution of 0.1 mm was

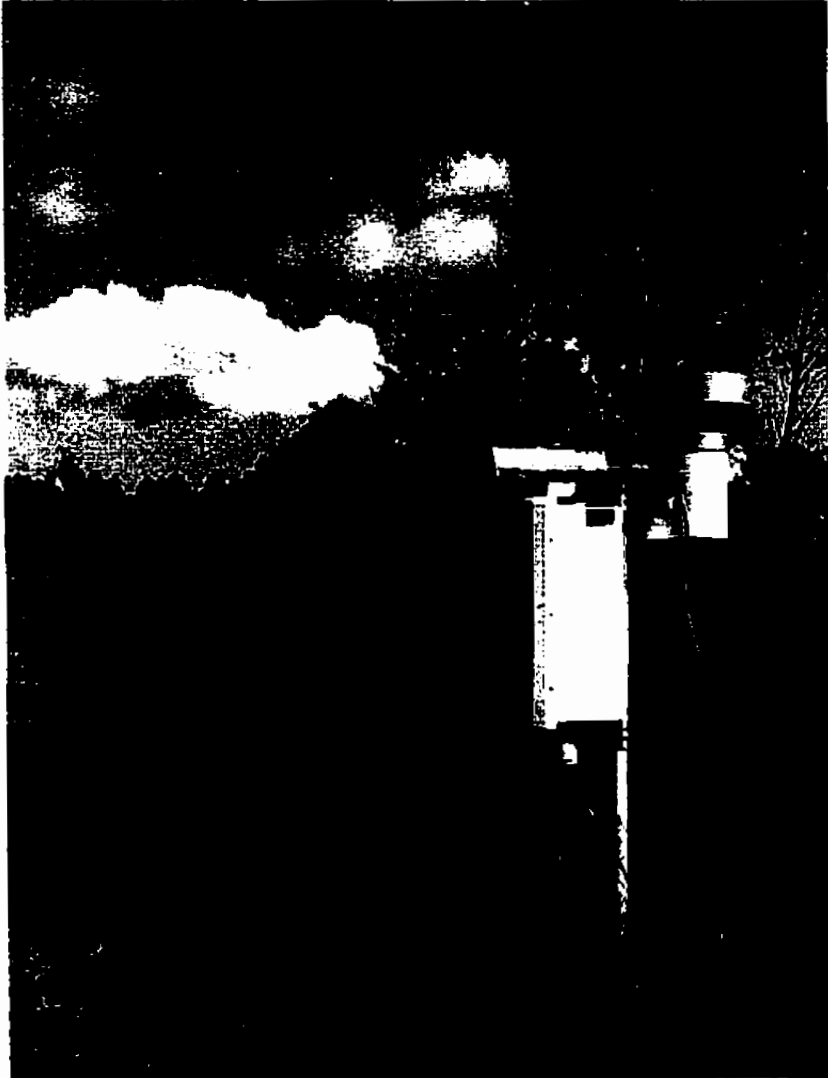


Figure 5.1 Typical weather station configuration used to measure weather conditions during the 1996 - 1998 dollar spot epidemics.

mounted one meter above the native green. Instrumentation was connected to a micrologger (Model 21X), and sampled at one minute (min) intervals, and then averaged (or summed in the case of the rain gauge) over ten minutes. Environmental monitoring equipment was calibrated before installation in the spring of each year and was removed from the field in October.

5.3.4 Disease Assessments

Scouting for disease began prior to dollar spot epidemics during the spring of 1996, 1997, and 1998. Diseased tissue was sampled from the first dollar spot foci of 1997 and 1998 to confirm that *S. homoeocarpa* was the causal agent of dollar spots at the GTI. Fruiting structures or fungal spores were not assessed since there are no reports of apothecia or sporulation of *S. homoeocarpa* in North American. Daily scouting for new spots was made at 15:00h during 1996 and 1997. In 1998, daily observations were made before 09:00h to not interfere with golfers. Plots were divided into 25 - one m² subplots (10 - 2.5 m² subplots at the Cutten Club) to facilitate counting of *S. homoeocarpa* foci. A "new focus" consisted of two or more blades of grass exhibiting typical dollar spot symptoms. Typical symptoms included lesions of straw-colored or white tissue usually at the leaf tip, but occasionally on the leaf surface. Bleached lesions were commonly bordered with reddish-brown necrotic tissue and, following warm evenings or a thunderstorm, white mycelia grew from diseased tissue. New foci at the GTI and Victoria West Golf Course were counted and then marked with a golf tee to avoid recounting of spots. Golf tees were pushed into the ground until flush with the turfgrass surface so a mower could pass over. Foci at the Cutten Club were not marked with golf tees because tees would interfere with play on the fairway.

Greens were assessed for dollar spot 4 - 7 times per week from May (June in 1996) until disease diminished in September or October. Several techniques to graphically present dollar spot epidemics were explored, then two presentations were chosen to indicate incidences of dollar spot increase and disease progress. Epidemics were presented as: 1) disease progress curves of cumulative foci and 2) percent new disease per day calculated by dividing the number of new foci by the total number of foci to date, then multiplying by 100%.

5.3.5 Summary of Weather Data

Daily weather data were summarized on a noon-to-noon basis. This time interval was chosen to include the dew period, a factor proven to affect dollar spot severity (Williams et al. 1996). Relative humidity and temperatures of the air and soil were averaged over this 24 h period, and corresponding minimum and maximum values were calculated. Relative humidity $\geq 85\%$ was used as an indicator of atmospheric vapor high enough to encourage aerial mycelial growth of *S. homoeocarpa* (Smiley et al. 1992). Relative humidity $\geq 90\%$ has been also used as an indicator of leaf wetness (Sutton et al. 1984) but, in this analysis, the threshold was lowered to 85 % to allow for error in instrumentation and the fact that bentgrass leaves wet faster than leaves on other plants because of guttation and distillation. Rainfall and irrigation were summed for each day, and days with >5 mm of precipitation were considered rain days because irrigation was usually applied at 1 - 2 mm per day. L was calculated using continuous hours that the electronic leaf wetness sensors were $>2.5\%$ wet, a threshold that was chosen based on the results of Chapter 2. Minimum and mean air T during the L was also used as a variable. The number of hours of RH $\geq 85\%$, and mean and

minimum air T during this period, was calculated.

5.3.6 Validation of the Mills & Rothwell and Hall Models for Predicting Dollar Spot

Weather data and epidemics of 1996 - 1998 were used to validate the M&R and Hall dollar spot prediction models. The M&R model predicted a disease increase when maximum air T was ≥ 25 °C and RH was ≥ 90 % for any three days in seven. The Hall model predicted a disease increase when there were two days of rain with mean daily air T ≥ 22 °C or when there were three days of rain with mean daily air T of ≥ 15 °C. Criteria of the M&R and Hall models would be incorporated into the preliminary disease forecasting system if these criteria were correlated with dollar spot symptom expression.

5.3.7 Predicting Dollar Spot

Weather conditions were correlated with the start of the dollar spot appearance on any green at the GTI during 1996 - 1999 using an accumulation of days from May 1 that had mean air T above a threshold T. Subsequent increases in disease were modeled through step-wise multiple regression using SAS statistical software (SAS Institute Inc., Cary, NC) to correlate weather variables with percent new disease to date. Some weather variables were collinear; therefore, model assumptions were violated and coefficient of determination (R^2) values could not be relied on to assess the fit of the model. To overcome the collinearity problem, data were separated into two data sets (Bowley 1999). The calibration data set was the combination of the native green and pathology green epidemics from 1996 and 1997 and was used to calculate the regression equation. Data from the Cutten Club and Victoria West Golf Course gathered during 1998 comprised the validation data set used to test the accuracy

of the model. Data were made normal with independent residuals by a $\log(y + 1)$ transformation. Data were checked to ensure normality and equal variances using the Shapiro-Wilk W -test statistic for normality (Royston 1995) and an informal test examining residual plots for homogeneity (Kuehl 1994). Multiple regression was completed using backward step-wise regression with variables being rejected if they were not significant ($P \leq 0.10$). Models were deemed significant at $P = 0.01$ and R^2 values were used to assess the fit of each model, keeping in mind the effects of collinearity of the variables. When the step-wise regression selected collinear variables (i.e., minimum, maximum and average daily air T) for the model, the variable with the highest Type II sum of squares was left in the model and the others were removed, then a new model was created with the limited variables. The effect of the year and green was assessed by selectively removing the specific data from the calibration data set. Three models resulted from the statistical analysis, and all three were tested against the validation data set. The models were first tested against the entire epidemic for each site of 1998, and then the models were reassessed for only the period of the epidemic when the pathogen was causing disease. Deviations between the disease level predicted by the model and the actual disease observed were used to evaluate the suitability of the model for accurate disease prediction. A positive value would indicate the model over predicted the amount of disease, while a negative value would indicate the model under predicted the amount of disease. Ideally, all deviations would be zero, meaning the model could accurately predict disease increases. The decision-threshold to apply a control measure was determined using the ROC curves as described by Yuen et al. (1996). Accuracy of the model at the decision-threshold was calculated from the proportion of decisions that were correct (i.e., true positives or true negatives) in proportion to the cases (days above the

decision-threshold) and controls (days below the decision-threshold) in the data set.

5.4 RESULTS

5.4.1 Dollar Spot Epidemics and Weather Conditions

1996. Assessments of dollar spot began on 4 July 1996 (day 186) when spots were >2 cm in diameter, indicating the epidemic began earlier. The Turfgrass Specialist at the GTI noticed spots on 14 June 1996 (day 166). Disease progressed on the pathology green until 22 August (day 235), at which time the fifth replication of the plot area became totally diseased and no new spots were distinguishable. However, the epidemic remained active outside the plot area until after 4 September. The native green epidemic began on 3 July 1996 (day 185) and dollar spot activity had not subsided upon the conclusion of the study on 20 September (day 264). Disease incidence was greater on the pathology green than on the native green; cumulative foci totaled 7,987 on the pathology green on 4 September (day 249) compared to 1,728 cumulative foci on the native green (Figure 5.2).

Rainfall of 1 - 38 mm occurred on 19 of 80 days monitored during 1996 and greens were irrigated with 1 - 8 mm of water on 16 days. Total rain during the study period was 252 mm and irrigation was 296 mm. Air T for 3 July (day 185) to 20 September (day 264) ranged from 4.8 to 34.6 °C with a daily mean of 18.7 °C. Relative humidity averaged 76.3 % for the season. Average daily RH was ≥ 85 % on 13 days, with 24 days with more than 12 h of RH ≥ 85 % . The average daily L was 16 h, ranging from 2 h 40 min to 24 h over the 78 days recorded and, there were 65 days with L ≥ 12 h and 39 days with L ≥ 16 h (Figure 5.3).

Figure 5.2 1996 dollar spot epidemic at the Guelph Turfgrass Institute for day 184 to 264 (4 July to 21 September). Disease progress on the pathology and native greens presented as cumulative foci to date, and observations of *S. homoeocarpa* mycelia at the GTI (A). Percent new foci represents the new spots noted each day divided by the cumulative spots to date and multiplied by 100% on the pathology green (B) and the native green (C).

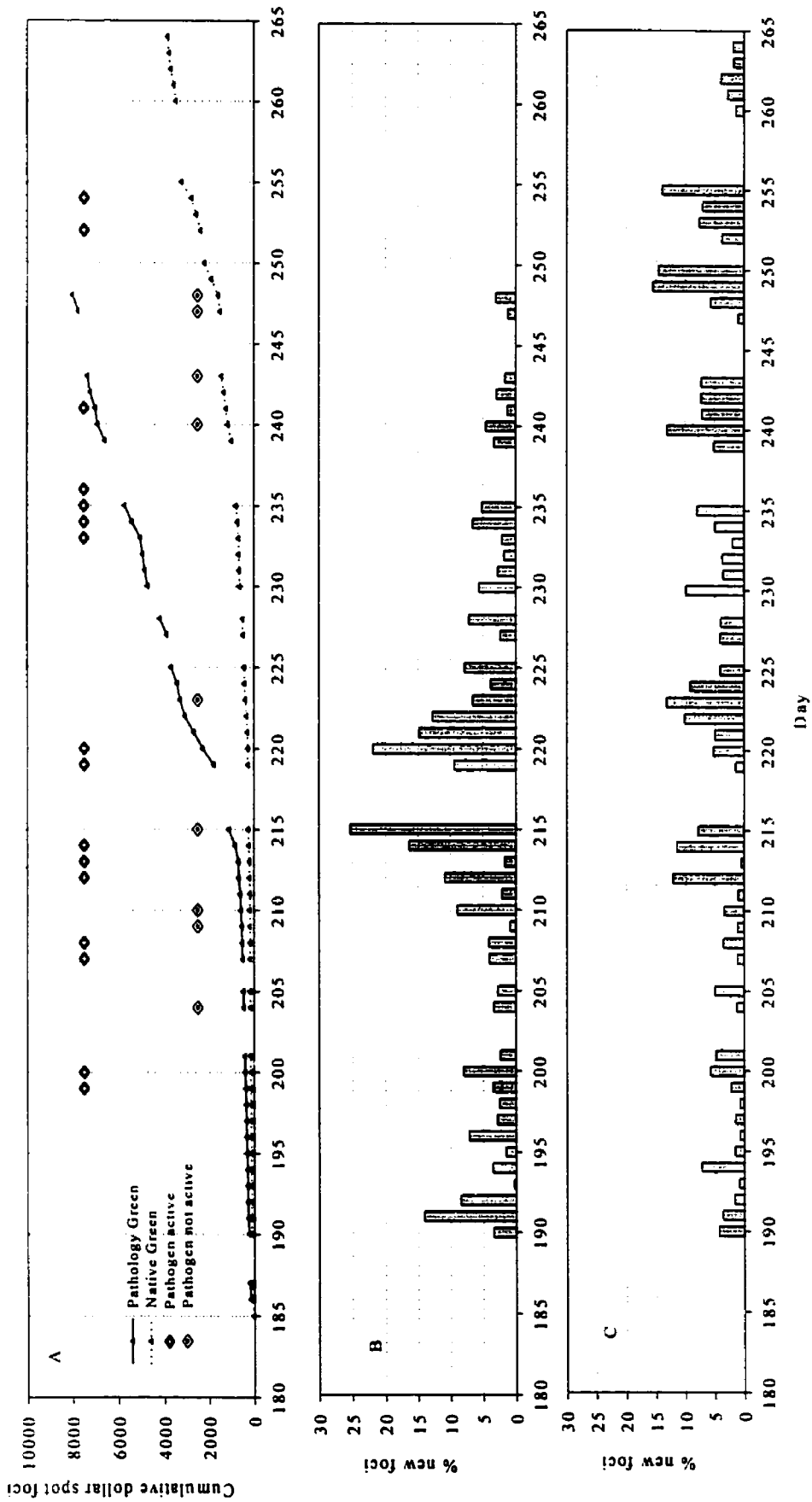
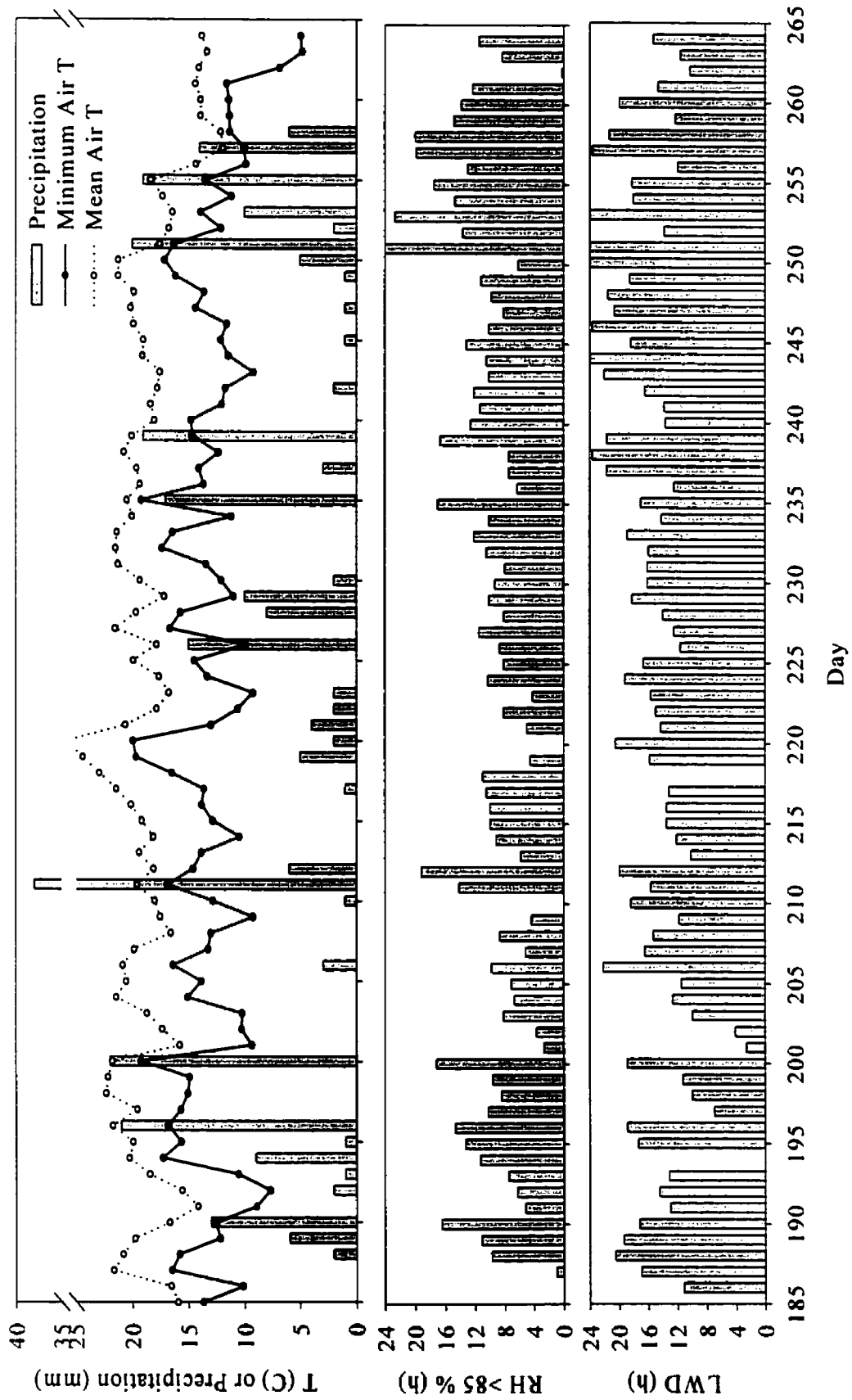


Figure 5.3 Precipitation (rainfall and irrigation), mean and minimum air temperature during day 185 to 264 (4 July to 21 September), hours of relative humidity > 85% for day 187 to 264, and hours of leaf wetness duration for day 186 to 264 at the Guelph Turfgrass Institute during 1996.



1997. Dollar spots were first noticed on the plot area on 17 June 1997 (day 168). Air T ranged from 0.5 - 32.6 °C with an mean of 17.5 °C for the period between 24 May (day 144) and 3 October (day 276). Plots were irrigated on 35 days with 0.3 - 4.3 mm of water, totaling 61.8 mm. Rain of 0.1 - 24.3 mm occurred on 40 days with total rainfall of 248 mm. Mean RH for the season was 71.3 % and 15 of 132 days had average daily RH \geq 85 % and 46 days had \geq 12 h of RH \geq 85 %. The mean daily L was 15 h (range of 2 h 40 min - 24 h), 103 days had L \geq 12 h and 49 days had L \geq 16 h of the 126 days monitored for L (Figure 5.4). The number of foci on the native green was 319 by 22 August (day 234) when this epidemic was no longer monitored because the turfgrass was physiologically stressed due to poor management. Cumulative foci on the pathology green increased to 2,187 by October 3 (day 276). Disease pressure during 1997 on the pathology green was only 27.4 % of the 1996 epidemic (Figure 5.5).

1998. The dollar spot epidemic began on 22 May 1998 (day 142) at the GTI. Temperature was warm with little precipitation during April and May. Greens at the GTI received only 76 mm of rainfall from 4 April (day 94) to 20 June (day 171) but were not irrigated until the later part of May, causing the pathology green to die. As a result, the dollar spot epidemic was delayed on test plots. Temperature above the native green ranged from -7.3 to 35.1 °C with an mean air T of 13.1 °C between 4 April and 20 June. Temperature above the pathology green ranged from -4.2 to 44.4 °C with a mean air T of 12.8 °C for the same period. Relative humidity on the site averaged 66.2 % and 11 of 77 days had daily RH \geq 85 %. Plots were relocated to Victoria West Golf Course and Cutten Club on 20 June (day 171). At Victoria West Golf Course, the air T ranged from -2.9 to 38.9 °C with mean of 19.8 °C for 21 June (day 172) to 8 October (day 281) (Figure 5.6). Relative

Figure 5.4 Precipitation (rainfall and irrigation), mean air temperature, minimum air temperature during day 170 to 273 (19 June to 30 September), hours of relative humidity >85 % for day 170 to 271, and hours of leaf wetness duration for day 170 to 269 at the Guelph Turfgrass Institute during 1997.

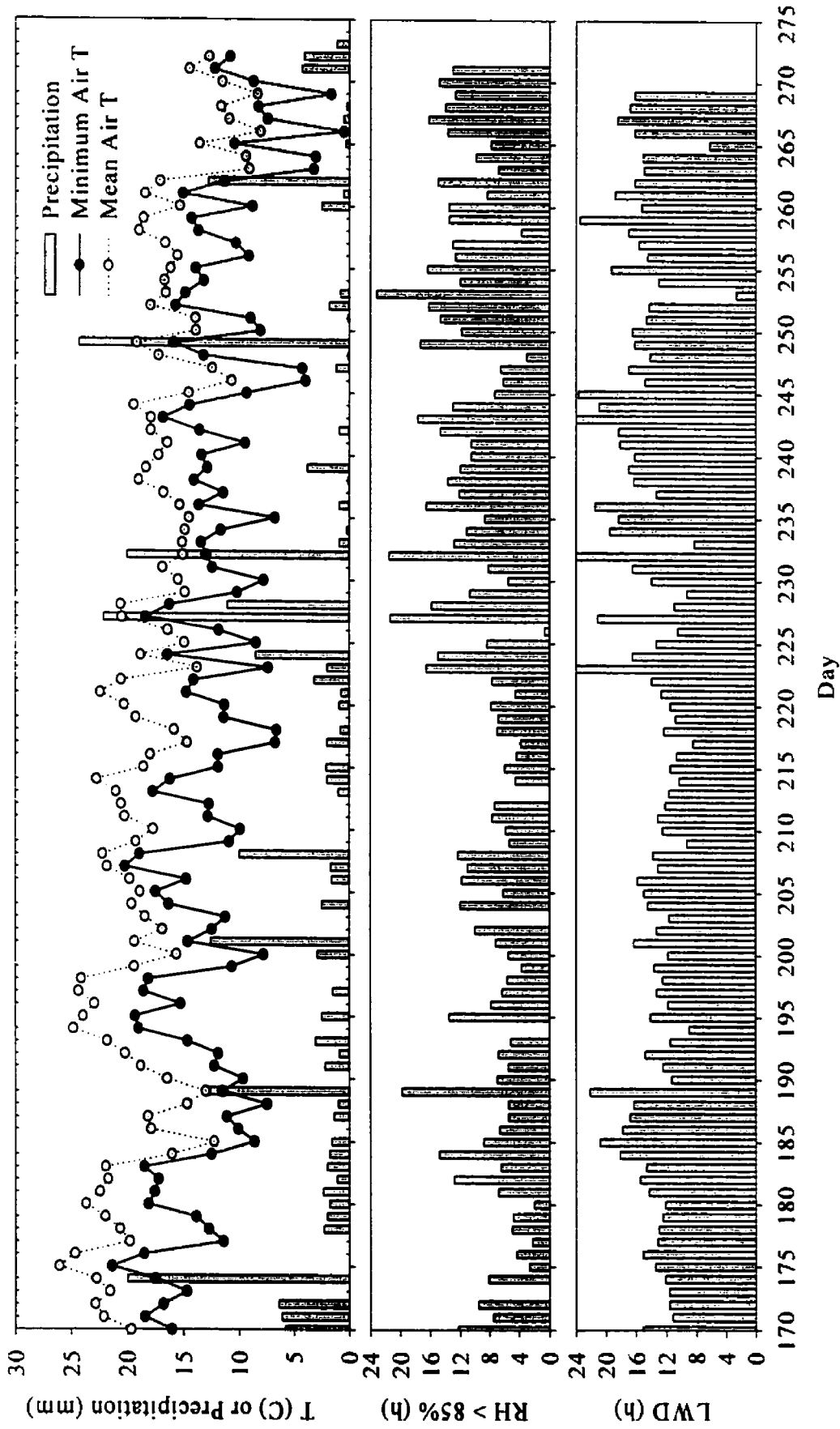


Figure 5.5 1997 dollar spot epidemic at the Guelph Turfgrass Institute from day 170 to 276 (19 June to 3 October). Disease progress on the pathology and native greens presented as cumulative foci to date, and observations of *S. homoeocarpa* mycelia at the GTI (A). Percent new foci represents the new spots noted each day divided by the cumulative spots to date and multiplied by 100% on the pathology green (B) and the native green (C).

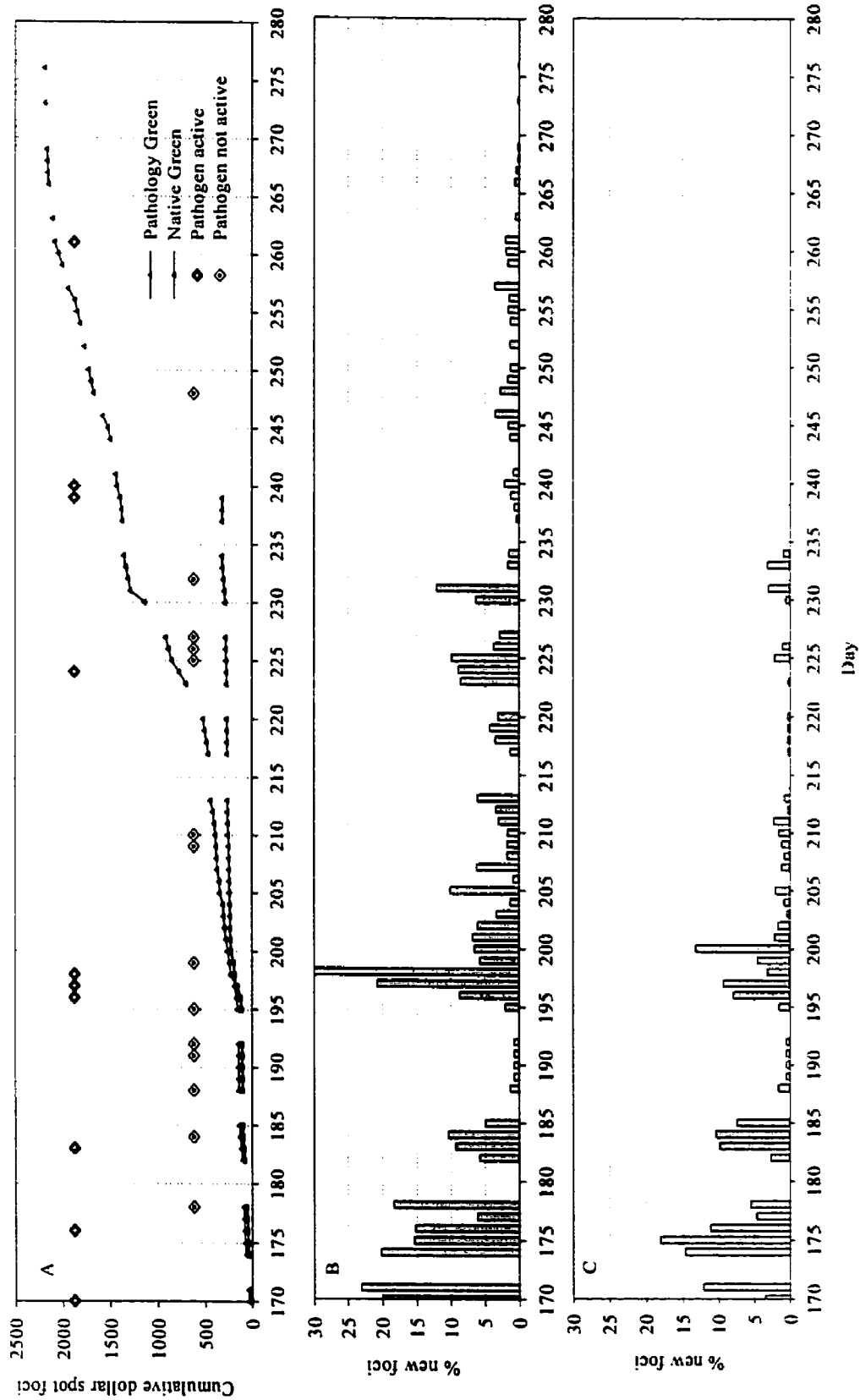
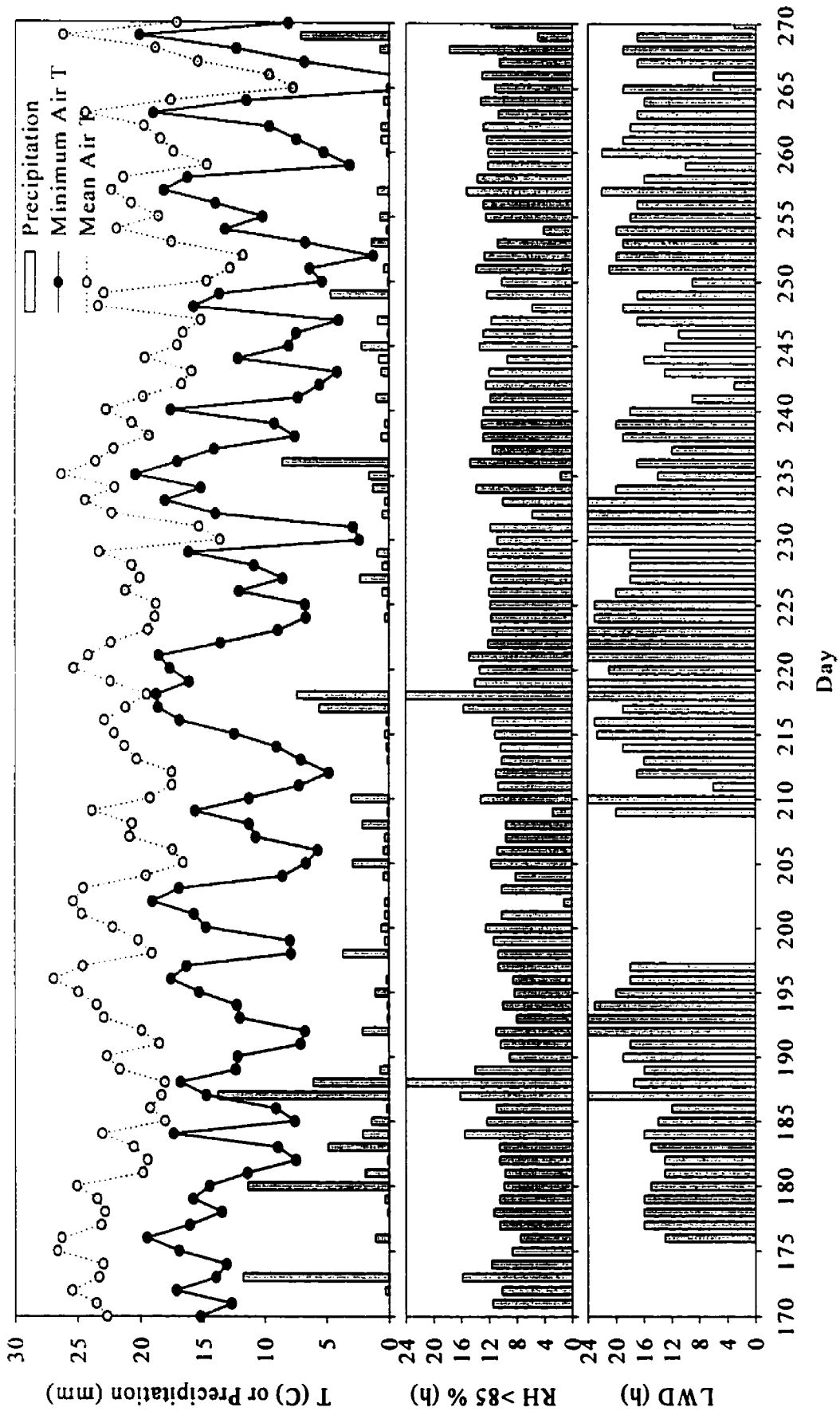


Figure 5.6 Precipitation (rainfall and irrigation), mean air temperature, minimum temperature for day 171 to 270 (20 June to 27 September) and hours of relative humidity > 85%, and hours of leaf wetness duration during day 171 to 270 at the Victoria West Golf Course during 1998.



humidity averaged 73.8 % and seven days had mean RH \geq 85 % with 15 days having \geq 12 h of RH \geq 85 %. The mean air T at the Cutten Club for the same period ranged from 0.3 - 37.3 °C with mean of 20.6 °C (Figure 5.7). Relative humidity averaged 71.0 % and nine days had a mean RH \geq 85 %. Rainfall, not including irrigation, was below seasonal with only 185.3 mm of rain from 4 April (day 94) to 1 October (day 274) (Table 5.1). Sixty-seven and 65 days had L \geq 12 h for the Cutten Club and Victoria West Golf Course, respectively, and 22 and 26 days had L \geq 16 h of the 102 days measured, respectively. The epidemic started quickly at the Cutten Club on 21 June 1998 (day 172); however, the Victoria West Golf Course epidemic did not eventuate until after 22 August (day 234) despite efforts to promote dollar spot, such as decreased mowing frequency, higher mowing height, and no dew removal (Figure 5.8).

Comparison of air T and precipitation for the period of 3 July (day 184) to 20 September (day 263) are summarized in Table 5.1. The mean air T differed from 1996 to 1998 by +3.3 °C and was reflected in RH that decreased by 4.2 %. Temperature was generally higher and precipitation less when compared to the 30-year norm (Table 5.2). The most notable year was 1998 when precipitation was 78.7 % less than the 30-year norm. The deficit in rainfall was compounded by the previous year receiving 31.6 % below-normal precipitation and, therefore, a large portion of the water was delivered via irrigation rather than natural rainfall. Leaf wetness duration appeared to be less during 1998, with 63.7 % of the nights with L \geq 12 h compared to 81% during the preceding two years.

5.4.2 Validation of the Mills & Rothwell and Hall Models for Predicting Dollar Spot

In 1996, criteria for the M&R model were met everyday from day 189 to 257 and

Figure 5.7 Precipitation (rainfall and irrigation), mean air temperature, minimum temperature for day 170 to 270 (19 June to 27 September) and hours of relative humidity > 85%, and hours of leaf wetness duration during day 171 to 270 at the Cutten Club in 1998.

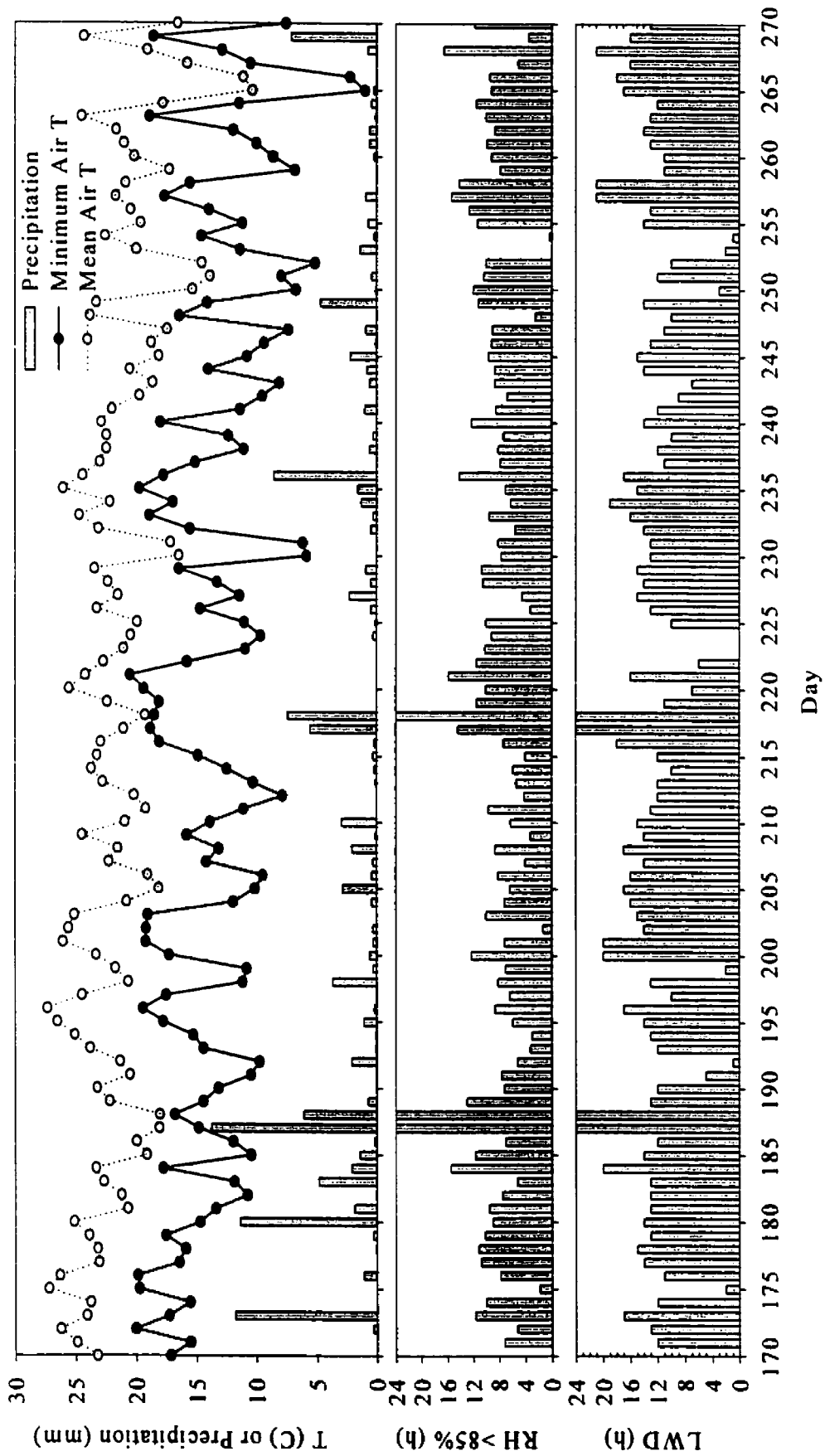


Table 5.1 Summary of air temperature (T), relative humidity (RH), rain and irrigation at the Guelph Turfgrass Institute during July 3 to September 20 for 1996, 1997, and 1998

Year	Mean T (°C)	Mean RH (%)	Mean RH≥85 % (days)	Rain (mm)	Rain + Irrigation (mm)
1996	17.4	74.3	13	252	296
1997	19.2	72.9	13	214.7	252.7
1998	20.7	70.1	13	143.2	326.3

Figure 5.8 1998 dollar spot epidemic at the Victoria West Golf Course and the Cutten Club from day 169 to day 273 (18 June to 30 September). Disease progress at both sites are presented as cumulative foci to date, and observations of *S. homoeocarpa* mycelia at either site. Percent new foci represents the new spots noted each day divided by the cumulative spots to date and multiplied by 100 %.

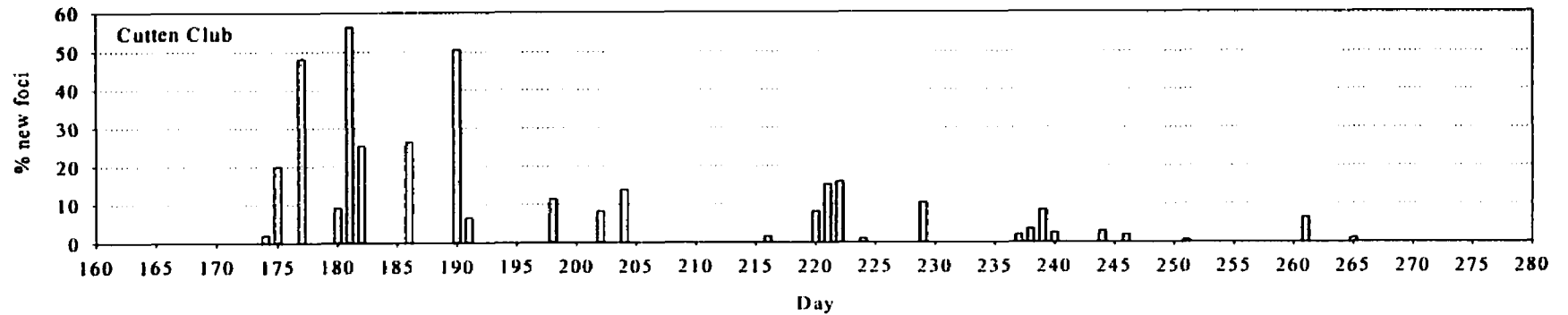
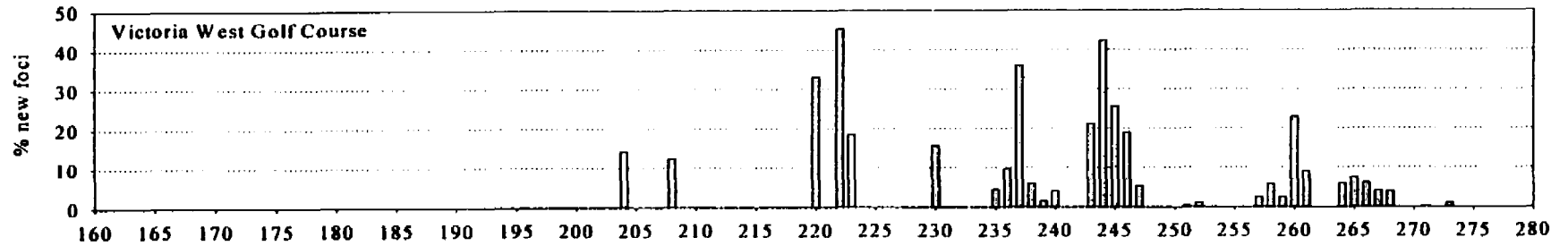
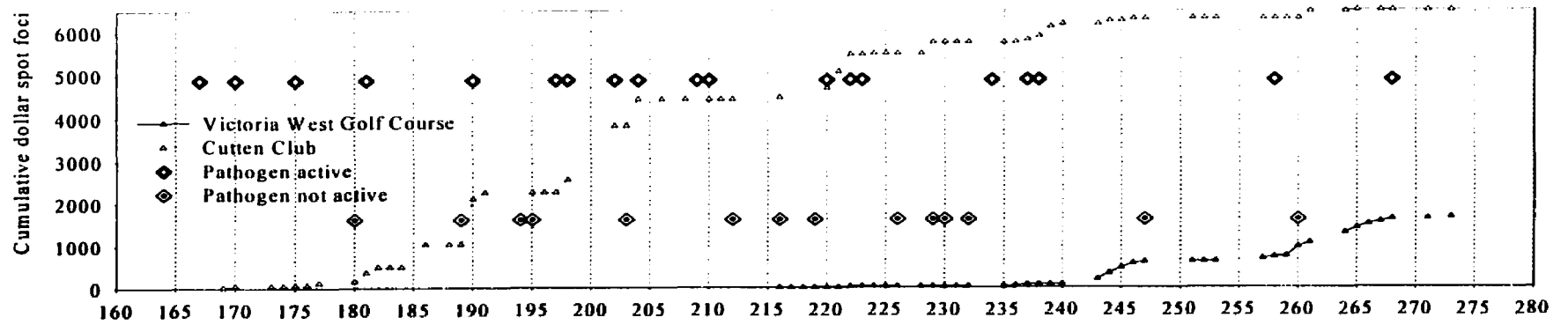


Table 5.2 Mean air temperature (T) and precipitation at the Guelph Turfgrass Institute during 1996 - 1998 compared to the 30-year norm (30-yr) in Guelph, ON

Year	Mean air T (°C)					Precipitation (mm)				
	May	June	July	Aug	Sept	May	June	July	Aug	Sept
1996			19.0	19.8	16.6			109.0	69.0	74.0
1997		19.2	19.5	17.5	14.5		79.8	46.7	72.4	42.7
1998	14.5	18.7	21.1	21.0	17.6	0	82.7	30.7	24.5	13.0
30-yr*	12.5	17.0	19.9	18.7	14.3	75.8	79.5	90.4	93.3	89.6

*Environment Canada (1999)

criteria for the Hall model were never met. Neither model demonstrated ability during 1996 to predict the need for fungicide applications (Figure 5.9). In 1997, the M&R model predicted disease activity on days 156 - 229, 237 - 246, and 257 - 265 (Figure 5.10). These periods were too vast to be of value for timing prediction-based fungicide applications. The Hall model predicted only one disease incident when the epidemic first started on day 171 - 173 and did not predict subsequent disease increases. In 1998, the criteria for the M&R model were met on days 110 - 131, 133 - 157, and 161 - 282 (Figure 5.11). The disease epidemic had not begun when the first period was predicted by the M&R model. The first dollar spots of the season appeared during the second predicted infection period. However, the M&R criteria were met for a period of 3 weeks in duration, a time frame more suited to a control program administered at regular intervals rather than a program applied in accordance with a prediction model. The prediction for day 161 - 282 spanned the entire duration of the 1998 epidemic. The Hall model predicted disease on day 139 - 140 and 149 - 150. Both predictions were the result of >5 mm of irrigation, not rainfall. The model would not have predicted any infection periods during 1998 if rainfall was the variable used. The first prediction coincided with the epidemic start on day 142. The second prediction was not verified since it was simultaneous with the drought and death of the GTI greens. The Hall model did not predict any other incidences, even though severity increased for the remainder of the season.

5.4.3 Predicting the Start of the Dollar Spot Epidemic

During 1996 to 1999, dollar spots were first noticed in May or June. There appeared to be no correlation with the start of the epidemic and calendar date. Informal observations

Figure 5.9 Relationship between selected environmental variables in 1996 and criteria of the Mills & Rothwell model of maximum RH $\geq 90\%$ and maximum air T $\geq 25\text{ }^{\circ}\text{C}$ for any of 4 days of a 7 day period (top graph). Criteria of the Hall model of 2 days of rainfall (water inputs $>5\text{ mm}$ indicated rainfall instead of irrigation) and mean daily T $\geq 22\text{ }^{\circ}\text{C}$ (bottom graph). The Hall model also predicted disease following 3 days of rain; however, there were never 3 consecutive days of rain.

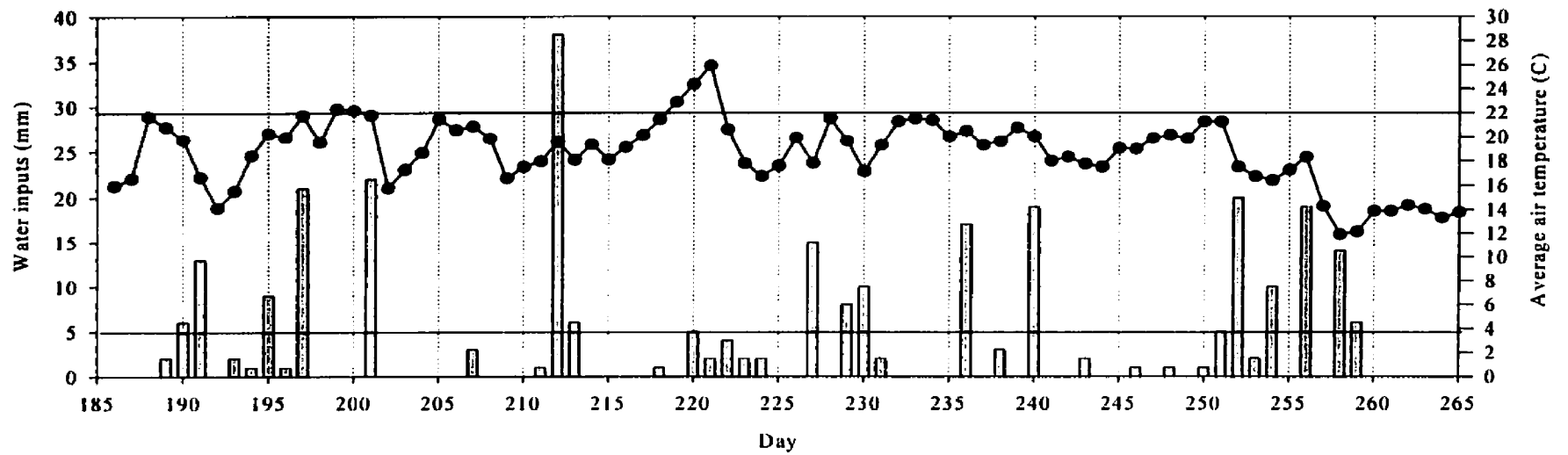
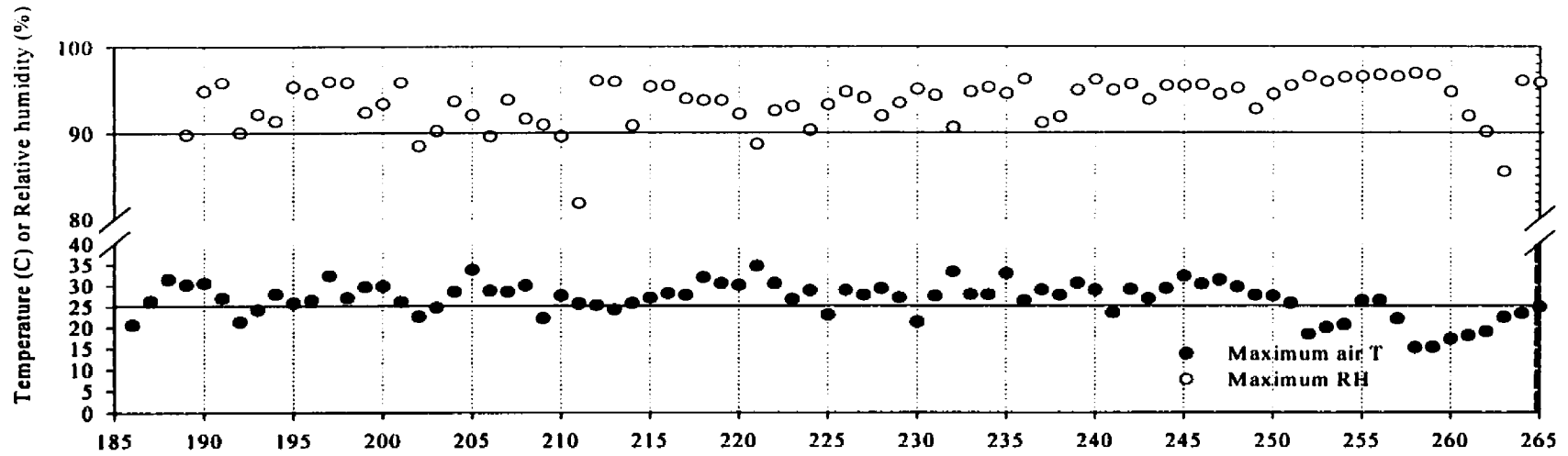


Figure 5.10 1997 weather conditions. Criteria of the Mills & Rothwell model of maximum relative humidity $\geq 90\%$ and maximum air T $\geq 25\text{ }^{\circ}\text{C}$ for any of 4 days of a 7 day period (top). Criteria of the Hall model of 2 days of rainfall (water inputs $>5\text{ mm}$ indicated rainfall instead of irrigation) and mean daily T $\geq 22\text{ }^{\circ}\text{C}$ (bottom). The Hall model also predicted disease following 3 days of rain; however, there were never 3 consecutive days of rain.

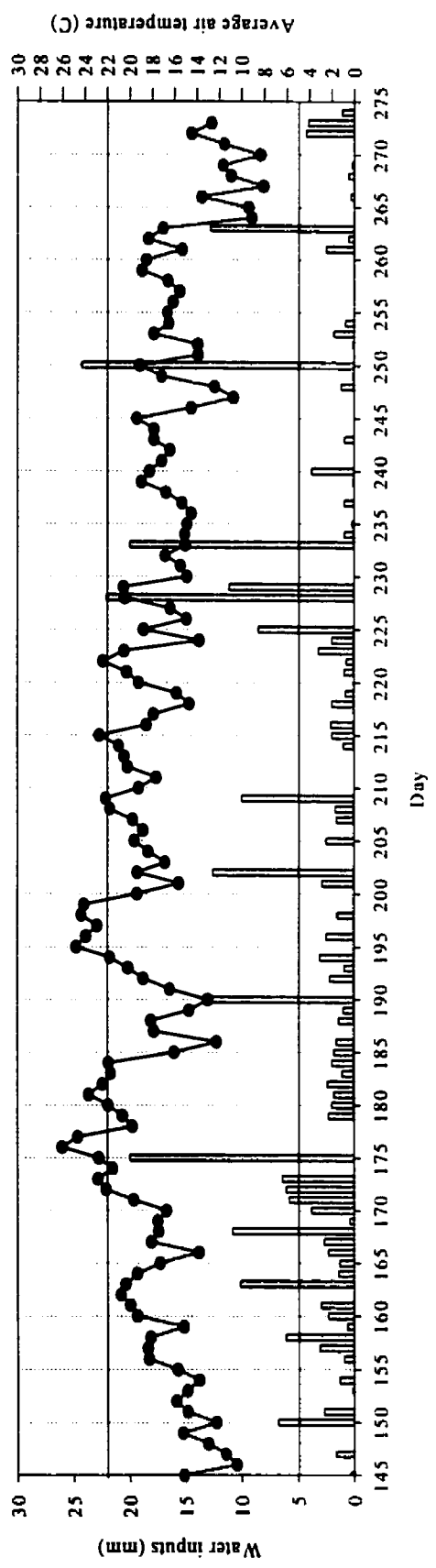
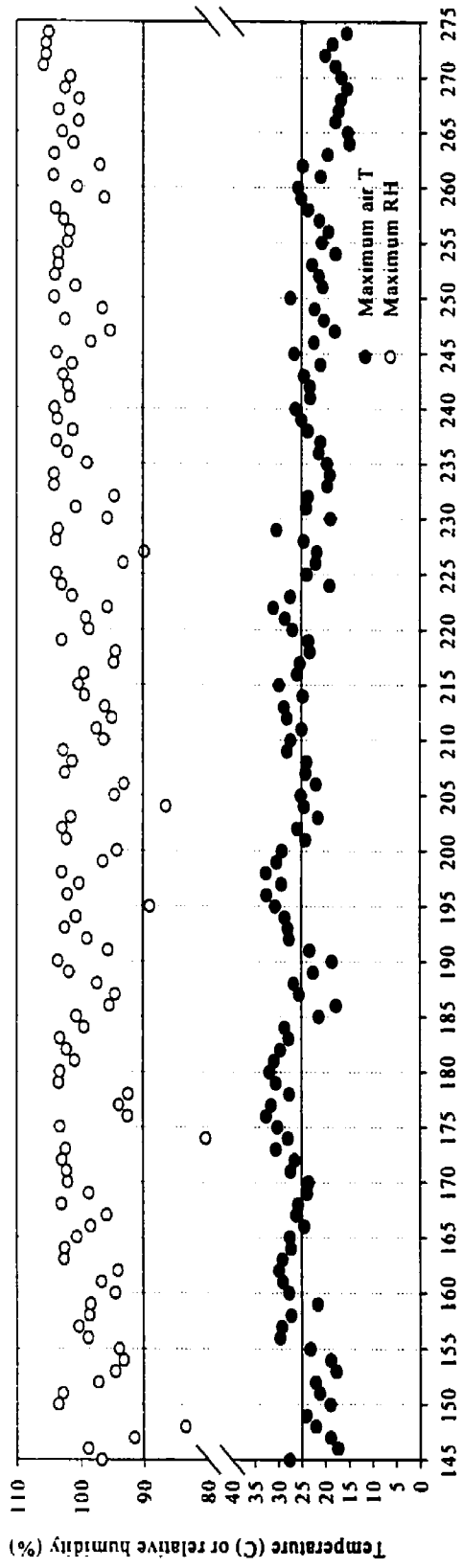
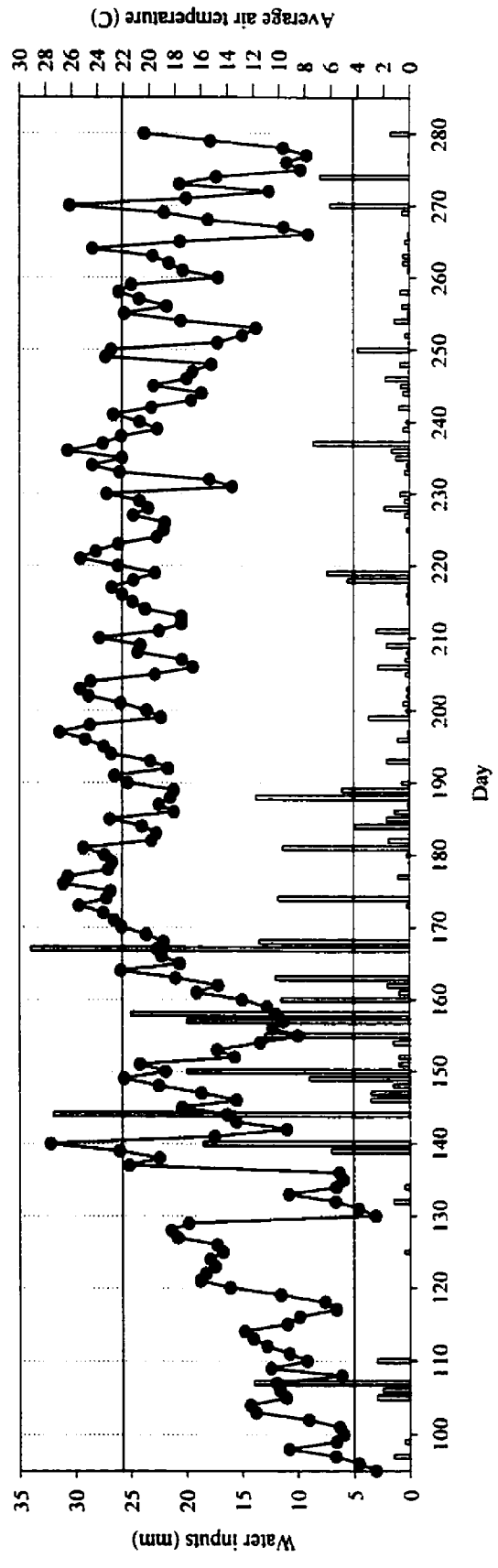
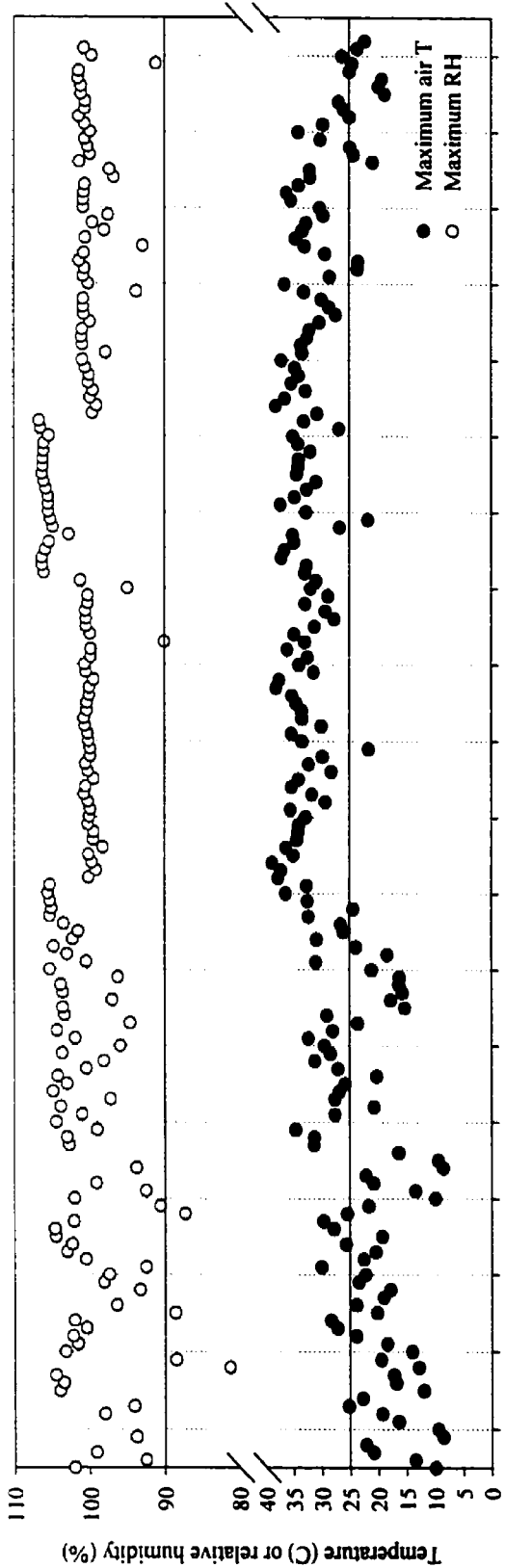


Figure 5.11 1998 weather conditions. Criteria of the Mills & Rothwell model of maximum relative humidity $\geq 90\%$ and maximum air T $\geq 25\text{ }^{\circ}\text{C}$ for any of 4 days of a 7 day period (top). Criteria of the Hall model of 2 days of rainfall (water inputs $>5\text{ mm}$ indicated rainfall instead of irrigation) and mean daily T $\geq 22\text{ }^{\circ}\text{C}$ (bottom). The Hall model also predicted disease following 3 days of rain; however, there were never 3 consecutive days of rain.



linked dollar spot appearance with late-bloom of lilac and irises. Temperature is known to be a variable that influences the development of plants and disease; thus, daily mean T was explored as a variable that could possibly be used to predict development of the first dollar spots of the season. All T data for predicting the start of the epidemic were recorded by the Environment Canada weather station at the GTI. This data source was used for consistency because the instrumentation for the green was not installed during May of 1996 and 1999. Air T was recorded using a standard thermistor placed in a Stevenson instrument shelter at 1.5 m above the ground. Daily mean air T was used to predict the onset of dollar spot epidemics at the GTI. A threshold T for disease development was targeted based on the work of Endo and Malca (1963) who found that dollar spot symptoms developed when T exceeded 15.5 °C. Results from the growth room experiment (Chapter 3) supported this threshold T because *S. homoeocarpa* caused significant turfgrass damage when T was ≥ 14 °C with 16 h L. Beginning on 1 May, days with daily mean air T ranging from 10 to 21 °C were totaled until the day before spots appeared on the greens at the GTI (Table 5.3). The totaled days with T above 14, 15, 16, and 17 °C had the smallest range in data over the 3 years. There were 13 - 14 days of daily mean air T ≥ 14 °C that accumulated before the start of the epidemic and 6 - 7 favorable days with T ≥ 17 °C. At mean daily air T of ≥ 16 °C, 9 - 10 favorable days after 1 May had occurred before the start of the epidemic.

The above criteria for predicting the start of dollar spot epidemics was tested in 1999 at the GTI. Dollar spots first became visible on the pathology green at the GTI on the rainy day of 1 June 1999 (day 152), and spots were obvious on 2 June (day 153) after a thunderstorm at 16:00h. There were 10 days after May 1 with mean air T ≥ 16 °C, which was the same as calculated during 1996 - 1998.

Table 5.3 Cumulative days with temperature of 10 - 21 °C until the start of the dollar spot epidemics for 1996 - 1999

Mean Daily T °C	Cumulative days until the start of the dollar spot epidemic			
	1996	1997	1998	1999
10	29	24	20	25
12	22	20	18	21
14	14	13	13	16
15	11	10	11	14
15.5	9	10	10	12
16	9	10	10	10
17	7	6	7	8
18	6	5	7	5
19	6	3	4	3
20	3	1	3	1
21	1	0	0	0

5.4.4 Predicting Dollar Spot During the Epidemic

The calibration data set derived from both greens during 1996 and 1997 was regressed against percent disease to date (y) to create Model 1: $\log(y + 1) = 0.78243 + 0.13143 \times \text{Rain Event} - 0.00942 \times \text{Daily Mean RH} + 0.03387 \times \text{Daily Mean Air T}$ during the L. This model was highly significant ($P < 0.0001$, $n=204$) but the R^2 was only 0.1525, indicating the model did not explain a large portion of variability in the data. Through plotting the deviations (plot not shown) the graph illustrated that the model was over predicting days when disease was closest to zero, and under predicting the days with great disease increase ($>20\%$). Therefore, the next step was to limit y to $\leq 20\%$ by eliminating days with disease increases $>20\%$, or by setting these days at 20% . Neither strategy improved the R^2 of the model. The dependent variable was then limited to those points that fell between 2 and 15% disease, but this also did not improve the fit of the model. During 1996, there was a very high incidence of disease resulting in >7000 spots on the pathology green by the end of the monitoring period and, thus, the disease increased each day and was not sporadic in occurrence. My hypothesis was that a model cannot decipher differences in weather variables when the disease appeared over a vast range of conditions. The 1996 data from the pathology green were removed, and Model 2 resulted in: $\log(y + 1) = -0.28551 - 0.03644 \times \text{Minimum Air T} + 0.05193 \times \text{Daily Mean Air T during L} + 0.02754 \times \text{Daily Mean Air T}$. The model was significant at $P < 0.0001$ and $n=164$ and R^2 was 0.1638, which was likely elevated by the collinearity of the three weather parameters used in Model 2. All 1996 data were then removed from the calibration data set and showed that 1996 data did not contribute to the model significance or R^2 ($P = 0.0456$, $R^2 = 0.0456$). The analysis of the 1997 data ($n = 120$) yielded Model 3: $\log(y + 1) = -0.31868 + 0.05038 \text{ Daily Mean Air T}$,

with $R^2 = 0.1805$ and $P < 0.0001$.

The models made with the calibration data sets were tested for accuracy with 1998 validation data set, using only the data gathered during the epidemic and not including days prior to the epidemic start. Predictive ability of the model was assessed by plotting deviations of predicted percent disease minus actual percent disease to date for the Victoria West Golf Club (Figure 5.12) and the Cutten Club (Figure 5.13). Model accuracy was similar among all three models across both greens, and all were poor at predicting disease. The deviations showed the model did not predict important increases in disease. The validation data sets were minimized to only the portion of the season when disease was most active on each individual green. However, this did not improve the prediction of disease.

Nevertheless, all models were taken to further evaluation to tentatively set a decision-threshold through ROC curve plots and to assess the accuracy of the models to predict above and below the decision-threshold (Figures 5.14 - 5.15). All curves followed very closely to the "line of no discrimination", thus a decision-threshold was not obvious. The ROC curve for Model 3 at the Cutten Club extended from the line of no discrimination, and the inflection point was estimated to be 0.5 % increase in disease to date, which was tested for accuracy as a decision-threshold. The decision-threshold of 0.5 % was 80.8 % accurate at predicting disease when it actually increased, but had a 19.2 % error because the model failed to predict disease even though it had actually increased. On the other hand, the model predicted a disease increase 55.8 % of the time when disease failed to do so. In other words, a superintendent may miss 19 % of necessary fungicide sprays, and over-spray by 56 % if he or she was to follow the predictions of Model 3. An arbitrary decision-threshold was selected by using the tolerance of one spot per m^2 . If a dollar spot of 3 cm in diameter is

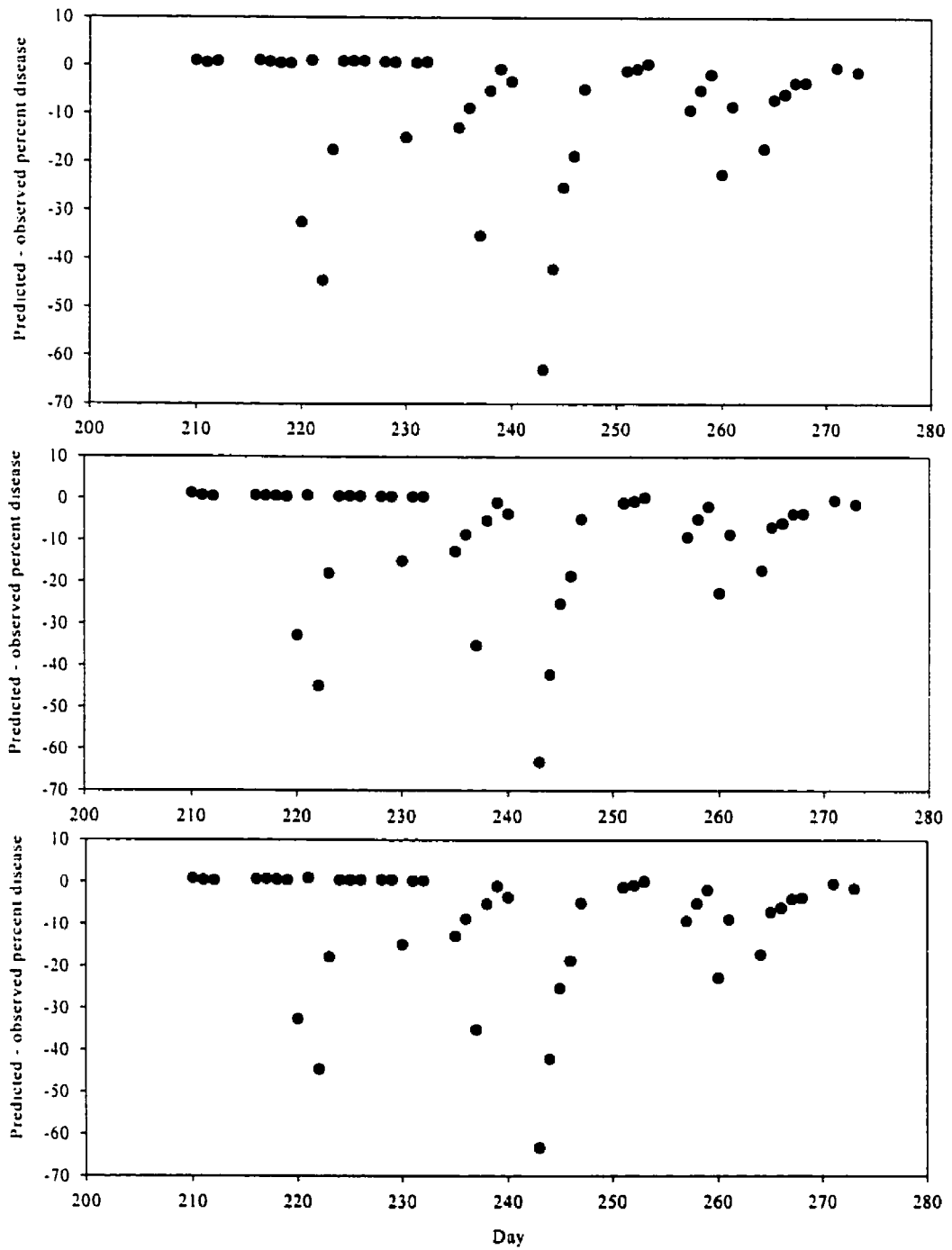


Figure 5.12 Deviations between predicted - observed percent disease at Victoria West Golf Course during 1998 for Model 1 (top), Model 2 (middle) and Model 3 (bottom) for the days following the start of the dollar spot appearance.

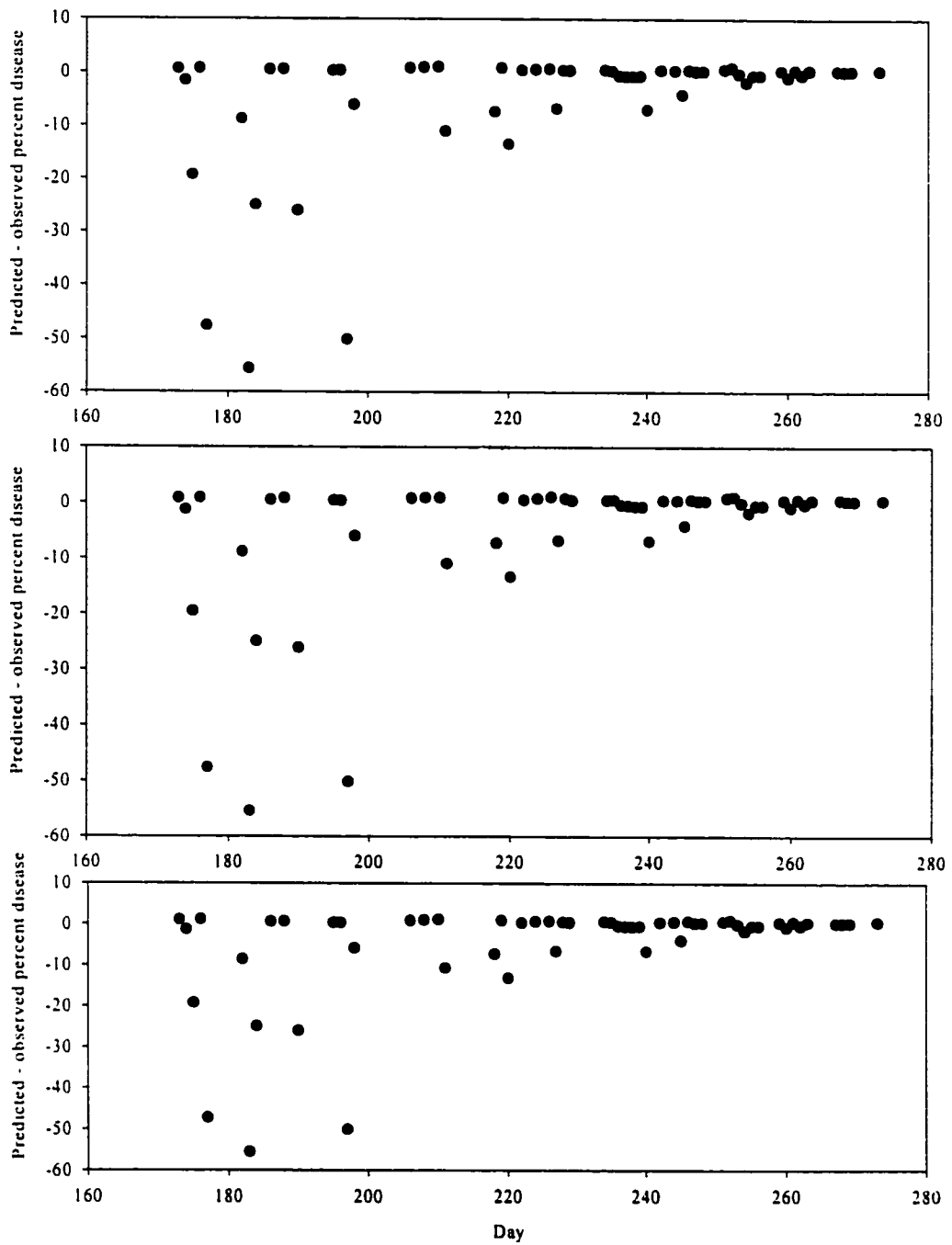


Figure 5.13 Deviations between predicted - observed percent disease at the Cutten Club during the active period of the epidemic in 1998 for Model 1 (top), Model 2 (middle) and Model 3 (bottom) for the days following the start of the dollar spot appearance.

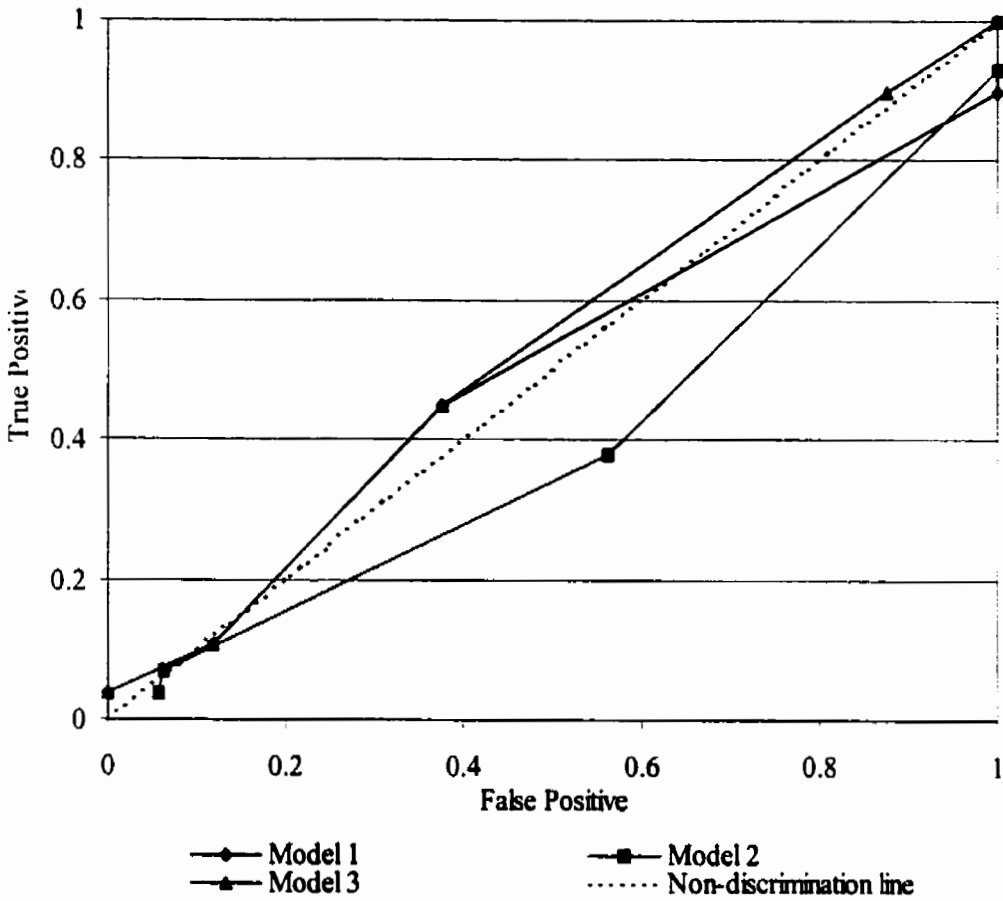


Figure 5.14 Receiver operator characteristic (ROC) curves for Models 1 - 3 tested against the percent disease from the Victoria West Golf Course during 1998.

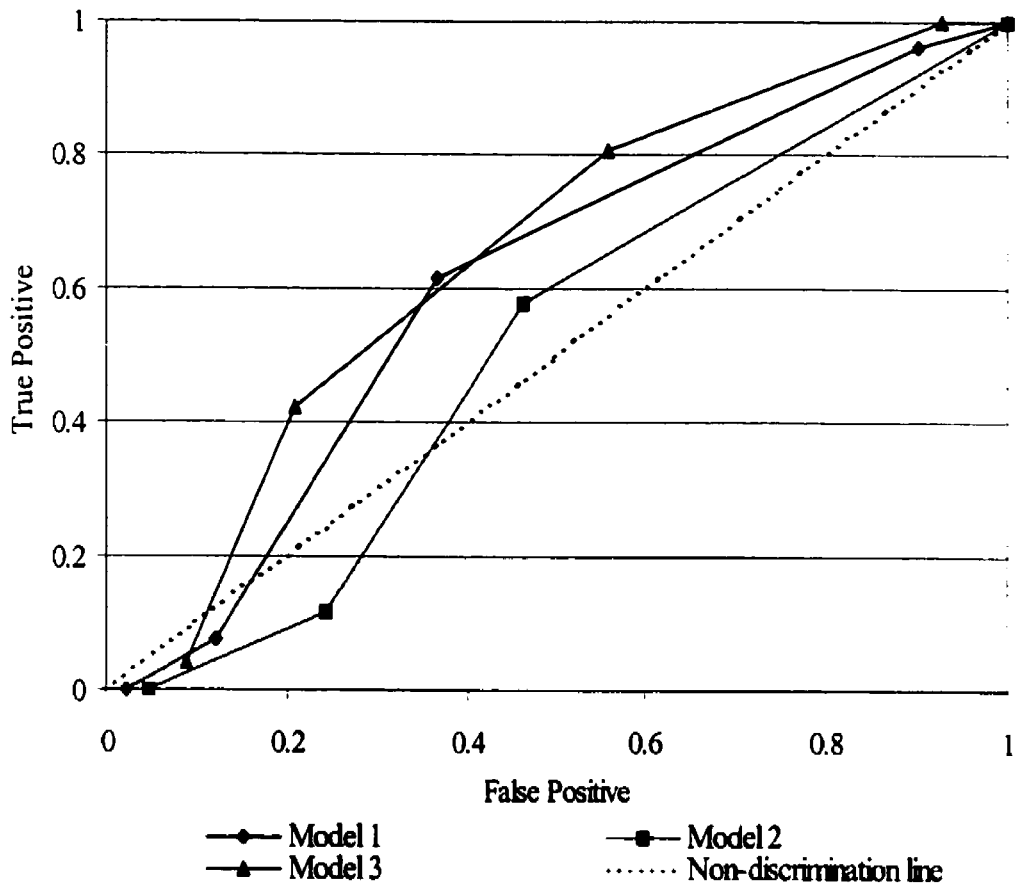


Figure 5.15 Receiver operator characteristic curves for Models 1 - 3 tested against the percent disease at the Cutten Club during 1998.

assumed, with an area of 7 cm², then one spot per m² would be 0.07% disease. Based on this threshold, the accuracy of the models was zero and the recommendation was to treat the disease under all weather conditions.

5.5 DISCUSSION

In this study, several approaches to designing disease prediction models for dollar spot of creeping bentgrass were developed and assessed. Predicting the start of epidemics through cumulative air T was particularly promising and may integrate well with existing industry practices. At present, the decision to start fungicide applications for dollar spot is made through scouting of the golf course or is based on a calendar date. Predicting the start of dollar spot appearance on golf courses using this proposed model of accumulation of 9 - 10 days, after May 1, with mean air T ≥ 16 °C will be easily calculated from local air T data. The information gathered from this study conflicts with that of Sears et al. (1996) who stated that dollar spot developed after June. The proposed model is necessary because most people cannot diagnose dollar spot at the single lesion stage, but instead have to wait until the fully developed dollar spots are visible, or until mycelia is observed. Many days can pass from infection to this stage of symptom development, and subsequently, the inoculum can colonize a greater amount of the turfgrass sward. When the disease is controlled before the epidemic starts, additional inoculum is not immediately produced and disease may be postponed until the next infection period and not reach the magnitude of an unanticipated, uncontrolled epidemic. In addition, fungicide applications are not administered when there is no threat of disease. Limiting fungicide applications will reduce selection for fungicide-

resistant pathogens, allow managers to not exceed early in the season the annual limit of applications, save money on fungicide purchase and application costs, and lower golfer and applicator exposure to toxic substances.

Step-wise multiple regression has the potential to be a powerful tool to select the weather variables contributing to dollar spot increases. Unfortunately, the models that resulted from analysis did not exhibit predictive ability. This could be a factor of the disease increasing throughout the summer under a variety of weather conditions. Even though disease developed on most days during the summer, a large increase in new dollar spots were observed on some days, while on other days only a few new spots were counted. The model was not able to differentiate between days with large increases from days with small, or no, disease based on weather conditions. According to the literature, dollar spot development occurs over a large T range (Couch 1995, Endo 1963, Sears et al. 1996), and the present study supports that observation. There may be other variables that influence disease more than the weather, such as N fertility, condition of the green, soil characteristics underlying the turfgrass, UV radiation, etc.; these alternative variables warrant investigation. An assumed prediction threshold of one spot per m², used in conjunction with the models from the regression analysis, recommended a disease control treatment under all circumstances. This showed that either the disease is not sporadic and weather conditions are always favorable, or the models were totally inaccurate. When the ROC curves were used to assign a decision-threshold, models at both greens yielded curves that were close to the line of no discrimination, and thus a reasonable decision-threshold was not set. Model 3 on the Cutten Club showed a point of inflection close to 0.5%, which was tested as a prediction threshold. Use of the 0.5 % threshold resulted in an over-prediction of disease; hence the resulting

disease control would be a function of over-prediction rather than the ability to correctly predict dollar spot increases and treat appropriately.

Only 185.3 mm of rainfall was recorded between 4 April and 1 October 1998 and the dry conditions were compounded by the previous year's receiving 31.6 % below normal precipitation. Therefore, a large portion of the water was delivered via irrigation rather than natural rainfall. Leaf wetness duration appeared to be less during 1998, with 63.7 % of the nights with $L \geq 12$ h compared to 81% in the previous two years. More rainfall and greater L in 1996 may have caused the 1996 epidemic to have 365 % more disease than in 1997, and 124 % more disease than 1998. The reduction of disease in 1997 at the GTI may have been caused by less rainfall and also the poor management of the greens. While N deficiencies usually lead to a greater number of dollar spots on the turfgrass, the turfgrass was so nutrient stressed there was no host tissue for the pathogen to infect and the disease progress curve was affected.

Dollar spot epidemics started at the GTI between May and June in 1996 - 1997. Dollar spot development occurred at the GTI on 22 May 1998; however, the epidemic did not start on the nursery green at Victoria West Golf Course until 22 August 1998. The pathogen was assumed to be present because it was active on the nursery green during the previous year. Every practice to encourage dollar spot symptoms was utilized including fertilizer reduction because N applications decrease dollar spot severity, and by less frequent mowing to increase dew duration, moisture retention in the turfgrass canopy and to leave diseased tissue intact (Cook et al. 1964, Endo 1966, Freeman 1969, Markland et al. 1969, Watkins & Wit 1995). *S. homoeocarpa* appeared to be less active on newly established golf green, as seen with the late start of the epidemic on the nursery green, while other areas of

the golf course exhibited dollar spot activity. Less dollar spot pressure on new greens may be explained by Vargas' (1994) belief that *S. homoeocarpa* develops saprotrophically until some point in time it switches to pathogenic growth. Perhaps the population was not established to the point it could cause a dollar spot epidemic on the young bentgrass green. The factors that may influence this change in behavior are not understood.

The Hall model predicted the start of a dollar spot epidemic in 1998 only, and the M&R model failed to predict the start of any dollar spot epidemic. Neither model accurately predicted disease increases during 1996, 1997 or 1998. The assessment of the predictive accuracy of the M&R and Hall models was consistent with that of Burpee and Goultz (1986). The Hall model did not predict disease because there were rarely two consecutive days of rain and there were never three consecutive days of rain. The Hall model based predictions around rainfall and did not include irrigation or dew as influential sources of water input on golf greens. Rain was not the only influential weather variable because disease continued to develop in the absence of rainfall. Perhaps irrigation provided enough water for foliage or soil to sustain fungal growth. The M&R model grossly over-predicted disease because RH was >90 % most days due to irrigation temporarily raising RH and usually RH briefly exceeded 90 % at night. The same applies for maximum air T which commonly exceeded 25 °C for a short time each day in the summer when dollar spot was prevalent. Criteria of the M&R model were usually met because the M&R model had a seven-day window that often bracketed many infection periods. Perhaps the M&R model would predict the daily flux in disease activity if it stipulated T and RH that occurred for a fixed period of time on a daily basis.

Dollar spot is active when daily T is 15 - 27 °C, with the greatest severity at 21 - 27

°C (Couch 1995, Endo 1963, Sears et al. 1996). Mycelia grew on turfgrass when T was 10 - 25 °C in the growth room experiments (Chapter 3) and, Endo (1963) noted mycelial growth on PDA between 4.5 and 32 °C. Bennett (1937), Endo (1963), and Fenstermacher (1980) theorized that there are many biotypes of *S. homoeocarpa* that collectively occupy a large T niche on turfgrass. Further evidence of the diversity of *S. homoeocarpa* was gathered on 25 September 1998 at Victoria West Golf Course when mycelia were noted after night-time T of <10 °C and frost two days prior. There was no proof of cold season dollar spot, regardless of L, when conditions were tested against the 1996 - 1998 disease progress curves. Perhaps the strain was locally isolated or, there was a weather condition or other stimulus that activated the mycelia during the cool T. Characterization of the cardinal T of different isolates would answer the question of whether these are biotypes that each have a different optimum T or if this species is capable of growth at temperatures that span over 28 degrees. The genetic diversity of *S. homoeocarpa* is currently being investigated at a molecular level by Dr. G. Harman at Cornell University. Genetic comparison of the “cool temperature” isolate with other isolates of *S. homoeocarpa* from Victoria West Golf Course, other turfgrass in Guelph, and isolates collected internationally, may reveal biotypes identifiable at the genetic level that differ in optimal growth T.

The weather conditions during dollar spot development were remarkably similar to the criteria of other turfgrass disease forecasting systems. A fungicide application to control brown patch was recommended when RH \geq 95 % for \geq 8 h and mean RH \geq 75 %, L \geq 6 h or precipitation \geq 12 mm, and minimum air and soil T was \geq 16 °C during a noon-to-noon interval (Fidanza et al. 1996). Rhizoctonia blight was controlled when a fungicide was applied when minimum and mean air and soil T was 15, 20, 18, and 21 °C, respectively,

during the 24 h proceeding the tenth consecutive hour of RH \geq 95 %. The weather criteria used to predict brown patch and rhizoctonia blight are regularly achieved on a typical green in southern Ontario. The fact that these diseases appear to be occupying the same T and moisture niche on turfgrass is interesting, especially when dollar spot is the most prevalent of the diseases. A study that includes simultaneous monitoring of prevalent turf diseases may reveal weaknesses in the above mentioned models, or categorize the climatic niche that each of these diseases is occupying.

Leaf wetness was necessary for dollar spot development, as shown in the growth room when mycelia did not grow in the absence of L and in the field when disease did not result if L was $<$ 6 h. Williams et al. (1996) also demonstrated that L was important in dollar spot severity. However, using L for predicting dollar spot was problematic because L was often \geq 12 h during the normal season, and growth room data show that the pathogen was active when L exceeded 12 h and T was $>$ 10 °C . The conditions identified in the growth room study were met on many nights during the epidemics, thus discrediting the need for a forecasting system. Disease forecasting systems are usually of value if designed for diseases that are sporadic and for which a control measure exists. Dollar spot increased by \geq 10 %, or mycelia were active, on 65.1, 67.6, and 53.7 % of the days during the 1996, 1997 and 1998 epidemics, respectively, posing a threat throughout the summer. Treatments available to manage this disease are eradicants, meaning they can cure an infection; superintendents can allow the disease to infect before treating and then mow the diseased tissue away. Superintendents do not need to rely on a disease forecasting system for dollar spot because dollar spot is developing during most summer days and curative fungicides are available. However, this study provided a method to predict the start of the epidemic, thus allowing for

application of fungicides just as the epidemic is beginning, resulting in benefit for the turfgrass industry.

6. GENERAL DISCUSSION

6.1 DISCUSSION

Studies reported in this thesis contribute to an improved understanding of the etiology and epidemiology of dollar spot, an important disease of turfgrass. The comprehensive literature review in Chapter 1 consolidated the well-studied aspects of the *S. homoeocarpa*-turfgrass pathosystem, such as host range, symptom development, and management through fungicide or cultural control. However, Chapter 1 revealed that the taxonomy was incorrect according to Whetzel's (1945) definition of *Sclerotinia*. Also, environmental factors that influenced disease were not well defined and epidemic progress was not characterized and documented. Disease forecasting systems for dollar spot were designed on at least three occasions (Hall 1984, Mills & Rothwell 1982, NEOGEN), but none could accurately predict disease. Therefore, this thesis examined the environmental factors that contributed to the start and progress of dollar spot. Controlled environment experiments in Chapter 3 were conducted with one isolate of *S. homoeocarpa* (Sh48B) and employed a statistical design that required only five leaf wetness durations and three different temperatures to model a surface response curve for dollar spot focus diameter. Agrometeorological instrumentation for measurement of leaf wetness duration (LWD) had not previously been adequately tested for suitability for estimating dew duration on creeping bentgrass greens. Therefore, available leaf wetness sensors (LWSs) and prototypes were assessed for accuracy and precision of estimating dew onset and dissipation (Chapter 2). The data gathered from the most accurate and precise LWS, Campbell Scientific Model 237, was used in the analysis. Natural epidemics of dollar spot were studied from May to September for three seasons to monitor disease progress and determine the

effect of weather conditions on *S. homoeocarpa* activity. Temperature (T) was shown to influence the first appearance of symptoms on greens at the Guelph Turfgrass Institute. After initial symptoms were noticed, dollar spot increased on most days during the summer; therefore, weather variables were not significantly correlated with increases in disease. Six disease progress curves resulted from the daily counting of spots on greens. Comparison of disease progress curves (Chapter 4) to traditional temporal models used in plant disease epidemiology showed that dollar spot progressed in accordance with the logistic or exponential models and exhibited disease progress curves similar to those of “compound interest” or polycyclic diseases. This research resulted in a thesis that contributes to an improved understanding of the epidemiology of dollar spot in turfgrass.

The literature review in Chapter 1 was the first consolidation of the host range of *S. homoeocarpa*. This pathogen is reported to infect more than 40 dicotyledon and monocotyledon species, most of which are grasses cultivated for use on golf courses, sports fields, parks and homelawns. The history of the taxonomy of *S. homoeocarpa* is completely documented in Chapter 1, and thus explains the course of events that resulted in the name, *S. homoeocarpa*, no longer being correct. In 1945, Whetzel described the family Sclerotiniaceae, restricting the genus to include fungi in which the apothecium arose from a tuberoïd sclerotium that was formed free on aerial mycelium. Based on these criteria, *S. homoeocarpa* was excluded from this family (Kohn 1979a, Whetzel 1945) because true sclerotia have not been found on turfgrass affected by dollar spot (Baldwin & Newell 1992). Powell and Vargas (1999) are in the process of proposing that the causal agent of dollar spot be placed in the genus *Rutstroemia*.

The literature review also revealed that environmental conditions that influenced dollar spot were not well understood. A T range of 15 - 27 °C was reported to be conducive to dollar spot activity; however, the minimum, maximum and mean T for dollar spot development on turfgrass

were not established. There were no studies to correlate LWD to dollar spot severity; however, Williams et al. (1996, 1998) did provide detailed evidence that LWD was critical in dollar spot epidemics. Existing disease forecasting systems for dollar spot did not accurately predict disease. Therefore, the purpose of the research reported in this thesis was to develop a better understanding of the environmental conditions that influence dollar spot development and to expand on the etiology and epidemiology of this disease.

Before environmental conditions could be investigated, a method to measure LWD on turfgrass had to be determined. An electronic LWS was deemed the most inexpensive and practical means of collecting leaf wetness data, especially since the datalogger was being used to capture weather data. When this research was started, there were two LWSs used to measure LWD on turfgrass, but only the Campbell Scientific LWS could be used to monitor LWD on the small blades of creeping bentgrass. The existing Campbell Scientific LWS (painted or unpainted) and several prototypes (described in Chapter 2) were constructed and tested. Prototypes were designed to better monitor the unique turfgrass surface that accumulated dew from above (condensation) and below (distillation and guttation) the leaf tissue.

Ideally, sensor output would be zero when completely dry, and 100 when completely wet. However, this scale was offset in some cases because certain sensors were difficult to calibrate, a common problem when using LWSs. Therefore, for each dew observation, a response threshold for each sensor was calculated by adding the average output during a three-hour dry period (usually between 15:00 h and 18:00 h) with 2.5 % of the maximum output during the wet period. When the sensor output was above or below this threshold the sensor was deemed wet or dry, respectively. Establishment of this method for calculating the response threshold for turfgrass LWSs was an intensive calibration process, but now the method can be used as a standard practice when using

electronic LWS to monitor turfgrass LWD.

A system to evaluate and rank the accuracy and precision of the LWSs also was established for this thesis. Sensors with least squares means (LSM) closest to zero were considered the most accurate. The sensor with the most consistent response had the smallest standard deviation (SD), thus was considered the most precise instrument. The absolute LSM and SD for each sensor were summed ($|LSM| + SD$), then the value was used to assign an overall ranking of the sensors. The sensor with the smallest $|LSM| + SD$ was deemed most appropriate for field use. As a result of using this evaluation technique, the Campbell Scientific sensor on creeping bentgrass (S1) was most accurate for estimating dew onset with LSM of 11.7 minutes (min) and green sensor on creeping bentgrass (S6) was most precise with SD of 44.7 min. Campbell Scientific sensor in Kentucky bluegrass (S2) responded within 65.7 min of actual dew onset. For dew dissipation, S2 had the smallest LSM and SD of -0.9 min and 39.7 min, respectively, thus S2 responded within 40.6 min of actual dew dissipation. S2 estimated, on average, within of 1.7 h of the actual entire dew duration. Therefore, the Model 237 sensor, when placed within a Kentucky bluegrass canopy maintained at fairway height, was the best sensor for monitoring LWD on creeping bentgrass greens. Sensors 1, 2, 6 and 9 warrant further investigation in an experiment with at least three replications of each LWS. The next step will be to use the sensor in conjunction with weather variables to better correlate LWS output with the actual dew condition. The unmodified Campbell Scientific LWS has the accuracy required for use in other turfgrass disease models (Danneberger et al. 1984, Fidanza et al. 1996). These prediction models are robust and will accommodate LWD information with a resolution of one hour. In the case of this thesis, a more accurate LWS would not be a contributing factor to design of a usable dollar spot prediction model.

A statistical design not commonly employed by plant pathologists was used to model a

surface response curve for the relationship of LWD and T to pathogen growth on creeping bentgrass. This design allowed for testing at only five LWDs and three Ts. Using only three Ts was advantageous because the availability and function of controlled environment cabinets was a limiting factor in this kind of experiment. The controlled environment experiment identified the conditions conducive to the growth of *S. homoeocarpa* isolate Sh48B. The estimated minimum T for disease development ranged from 10 to 12 °C with 22 and 12 h L, respectively. The optimum T was 21 to 24 °C, depending on L. The linear response of disease to increases in L resulted in estimated focus diameter increasing as L was increased, at all Ts tested. This model is the first to quantify the role of L in dollar spot severity. Conditions identified were consistent with the literature that stated the fungus is active between 15 and 27 °C and that disease is most prevalent when T is between 21 and 27 °C. However, the experiment did not identify the maximum T this isolate could tolerate because the pots of grass were not viable after exposure to T >25 °C. Subsequent testing of different isolates to establish the cardinal T should be pursued, and perhaps differences between the isolates will help explain why four attempts (Hall, Mills & Rothwell, NEOGEN, Walsh) to develop a disease prediction model were not successful. The statistical model used to design the surface response curve violated one assumption of the model when the control treatment of 0 h LWD had no variance. The 0 h LWD interval had to be removed during the analysis. In future use of this statistical design, the minimum LWD tested should allow for enough growth of the pathogen to show variance (e.g., LWD = 2 h for *S. homoeocarpa* on bentgrass).

Dollar spot symptom development observed during this research was similar to that described by Couch (1995), Monteith & Dahl (1932), Sears et al. (1996), Smiley et al. (1992) and Vargas (1994). Symptoms of dollar spot began when mycelia in contact with green foliage caused a water-soaked appearance. The tissue was greyish-green without a reddish-brown lesion border. Grass

exhibiting these symptoms was marked with golf tees in the morning and, by the next day, water-soaked lesions had desiccated to form typical bleached tissue of dollar spot. The lesions were anywhere on the foliage of the Kentucky bluegrass, whereas the lesion primarily developed on the leaf tip of creeping bentgrass. During the early morning, or following a rain event, mycelia grew from diseased tissue to healthy tissue. Mycelia tended to resemble spider webs on the turfgrass surface, and vice versa. Tests were conducted to confirm the two structures were indeed different. Sampling of the mycelia growing from the diseased tissue yielded *S. homoeocarpa* in culture, whereas the spiderweb never resulted in cultures of *S. homoeocarpa* and always gave rise to isolates of sporulating fungi (*Fusarium* spp., *Bipolaris* spp. and *Drechslera eruthrospila*). Apothecia were never noticed during the course of this study, which was not surprising since few documented cases of fertile apothecia exist (Jackson 1973, Baldwin & Newell 1992). This thesis adds to the evidence that spores do not contribute to dollar spot epidemics. However, apothecia were regularly observed growing on the surface of three-month-old liquid cultures of isolate Sh48B, as they were observed by other researchers (Fenstermacher 1970, Jackson 1973, Baldwin & Newell 1992). However, dissection of many of these apothecia did not reveal ascospores or asci in the hymeneal layer of the apothecium surface. Attempts to produce fertile apothecia by changing the medium, providing solid substrate, adjusting the light, or the T were unsuccessful. Ascospores were not produced even when multiple isolates of *S. homoeocarpa* were combined on solid or in liquid culture. While molecular procedures may determine the taxonomic status of this fungus without the use of ascospores, the production of fertile apothecia from this species would be a mycological milestone.

Epidemics of dollar spot have not been fully characterized. In this thesis, disease progress curves were analyzed using EPIMODEL (Nutter & Parker 1997) to fit five temporal population growth models used in the analysis of plant disease epidemics (i.e., monomolecular, exponential,

logistic, Gompertz and linear). These temporal models stemmed from the simple and compound interest models first proposed by Vanderplank (1963). The disease progress curves for every year, except for 1997 on the native green, were logistic or exponential and resembled the curves associated with compound interest epidemics. This, in conjunction with the fact that apothecia are rarely seen in nature, supports the evidence that dollar spot is disseminated and infects new areas throughout the summer because small fragments of mycelia are moved across the turfgrass. Early control of dollar spot is necessary to effectively control this pathogen that reproduces exponentially until the green is completely diseased. Furthermore, minimization of the overwintering inoculum is important in this perennial crop since the fungus appears to overwinter in the host and reappear in the same location the following year.

Before this thesis was written, dollar spot in Ontario was thought to appear in June and continued to exhibit disease symptoms until October (Sears et al., 1996). Results presented in Chapter 5 showed that dollar spot begins before June. The first sightings of dollar spot coincided with the blooming of irises and peonies and when lilac were in full- to late-bloom. Dollar spot developed in Ontario when 10 days with average air $T \geq 16$ °C had accumulated after May 1. Individual lesions were very small and were not yet visible to most people at this stage of the epidemic. This proposed prediction method must be tested through comparison of treatments according to T-based predictions with treatments that are applied when symptoms are apparent. Currently, curative fungicides are used for dollar spot management. This allows for control of the disease after the fungus has infected and caused symptoms. Using fungicides curatively is not conducive to responsible resistance and environment management. Curative applications require higher rates of fungicide and are sprayed after the fungal population has increased. Fungicide resistance can be managed by using preventative fungicides, tank-mixes of fungicides with different

modes of action and, applications timed according to disease prediction models. Prediction of when the epidemic will start allows for the fungicide to be applied only when conditions are conducive to disease and therefore, fungicide applications are not wasted or used curatively.

Ideally, a prediction model would have been created to forecast disease increases during the epidemic so a fungicide could be applied only when required. Fewer fungicide applications would reduce the quantity of pesticide used, reduce costs, and would exert less selection pressure on resistant populations of *S. homoeocarpa*, thus prolonging the effectiveness of the fungicide. However, a prediction model could not be generated from the weather data gathered and step-wise multiple regression analysis. Dollar spot symptoms and mycelial growth occurred over a large T range and at temperatures that were common during the summer months in Ontario. Mycelia were noticed on greens after nights with air T far below the lowest T (15 °C) that was previously thought to cause disease and below the minimum T identified during the controlled environment experiments. Even after frost, the fungus survived and a major colonization of the green was observed 2 days later on 25 September 1998 at Victoria West Golf Course. In the past, spots observed in September and October were believed to have arisen during summer and lingered because diseased tissue was not removed by mowing due to the decreased rate of grass growth in the fall, thus giving the illusion that the dollar spot epidemic was continuing. However, this thesis proves the pathogen continues to grow, produce mycelia and cause new spots until at least the end of September. At the end of September in 1998, the minimum T dropped to <5 °C and the mean air T was <15 °C. The large T range for pathogen activity was likely the reason significant T requirements for dollar spot increase were not identified during the analysis of the three years of data. Further research should determine when this disease finally ceases growth in the fall and research should be focused on the “cool-season” dollar spot reported by members of the industry that

appears to exist in populations of *S. homoeocarpa* populations in southern Ontario.

Another possible reason why weather conditions did not correlate with disease is that LWD required for *S. homoeocarpa* growth and infection was not limited on the golf course green. Greens were irrigated on a regular basis and the average LWD measured on the greens for 1996 - 1998 was 13.9 h. Based on the controlled environment studies, this was sufficient LWD for the pathogen to grow across the turfgrass and infect the healthy foliage. Therefore, the LWD on the green was likely adequate for some pathogen growth on the majority of evenings during the season. This, in combination with the large T range for pathogen activity, may have caused new spots to develop on most days of the epidemic. Receiver Operating Characteristic (ROC) curve analysis was used to establish a decision-threshold for fungicide application. No action threshold could be established since the disease continued to increase regardless of environmental conditions that were monitored.

A second strategy to build a model was to explore weather conditions that resulted in periods when no infection was observed. This result could not be modeled mainly because there appeared to be no minimum T for disease development in the data set collected during this research. In addition, there were few days that had short LWDs. Limited field evidence revealed a trend that disease did not develop when LWD was less than six hours; but this must be proven with a larger sample size. Dew removal experiments by Williams et al. (1996, 1998) showed that prematurely ending the dew period helped manage dollar spot. Perhaps interrupting the dew period before it exceeds six hours may limit the severity of dollar spot

This thesis has contributed to the understanding of dollar spot epidemiology. Further research is required to comprehend how this pathogen can flourish in the warm, humid conditions of June and July and can continue to grow aerial mycelia and cause dollar spot symptoms in the cool temperatures of September and October. Controlled environment experiments with isolates of *S.*

homoeocarpa, in addition to the isolate collected on 25 September 1998 at Victoria West Golf Course, will determine if biotypes exist that differ significantly in response to cool temperatures. Four attempts were made to develop a forecasting system for dollar spot, but none were successful. Rather than predict disease, perhaps research should focus on when to apply fungicide to have the greatest impact on the disease progress. The modeling of the disease progress curves in this thesis has illustrated a typical epidemic and can be used to demonstrate what will happen when a fungicide is applied during a specific point in the epidemic. A method to predict the start of symptom appearance was proposed in this thesis, and now must be tested under practical conditions. Four of the LWSs should be rigorously tested and used in conjunction with other weather parameters to estimate dew duration on creeping bentgrass. While this thesis has made a contribution to the overall understanding of dollar spot, there are many more aspects of this disease left to investigate.

6.2 CONCLUSIONS

- The Model 237 sensor, when placed within a Kentucky bluegrass canopy maintained at fairway height, was the best sensor tested for monitoring LWD on creeping bentgrass greens. This sensor estimated, on average, within of 1.7 h of the actual entire dew duration.
- Sensors 1, 2, 6 and 9 (as described in Chapter 2) warrant further investigation in an experiment with at least three replications of each sensor and in conjunction with other environmental parameters.
- Controlled environment experiments were conducted as a modified central composite rotatable response surface design. This design utilized three temperatures and five leaf wetness durations to develop a surface response curve of disease severity (y), expressed as focus diameter, as it relates to L measured in hours and T measured in °C.
- Controlled environment experiments estimated minimum T for disease development ranged from 10 to 12 °C with 22 and 12 h L , respectively. The optimum T was 21 to 24 °C, depending on L . The linear response of disease to increases in L resulted in estimated focus diameter increasing as L was increased, at all temperatures tested. This model is the first to quantify the role of L in dollar spot severity.
- The most effective artificial inoculum for dollar spot was *S. homoeocarpa* mycelia grown for ten days on autoclaved grass clippings.

- Symptoms of dollar spot began when mycelia, in contact with green foliage, caused a water-soaked appearance. The tissue was greyish-green without a reddish-brown lesion border. Water-soaked lesions desiccated to form typical bleached tissue of dollar spot.
- Dollar spots reappeared in the same location as they were previously observed. This three-year study supported the hypothesis of Britton (1969), Couch (1995), Smiley et al. (1992) and Fenstermacher (1980) that stroma and/or mycelia of *S. homoeocarpa* survive locally in turfgrass crowns or foliage and are the initial inoculum for the disease.
- During 1997 and 1998, epidemics began when irises and peonies were in bloom and when lilacs were in full- to late-bloom.
- Predicting the start of dollar spot appearance on golf courses can be easily calculated from local air T data by recording the accumulation of 9 - 10 days, after May 1, with mean air T ≥ 16 °C.
- Disease progress curves were best fit to exponential or logistic models, both of which describe compound interest diseases. Each piece of mycelium (in grass clippings or individually) is a potential colony forming unit of secondary inoculum that leads to new infections.
- The Mills & Rothwell and Hall models proposed during the 1980s were tested against disease progress curves produced during 1996 - 1998, but these models were not predictive

of dollar spot increases.

- Receiver Operating Characteristic (ROC) curve analysis was used to establish a decision-threshold for fungicide application. No action threshold was established since disease continued to increase regardless of environmental conditions that were monitored.
- Step-wise multiple regression did not detect correlations between weather variables and dollar spot increases during epidemics. This study supports the literature that there are biotypes of this fungus capable of pathogenicity over a broad T range.
- Apothecia were never noticed during the course of the field study.
- Apothecia without asci or ascospores were regularly observed growing on the surface of >3 month-old cultures of isolate Sh48B in potato-dextrose liquid medium. Preliminary studies on nutrition, light, solidity of substrate and T did not produce new methods to encourage ascospore generation.

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