Bermudagrass Resistance to Spring Dead Spot Caused by *Ophiosphaerella herpotricha*

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ABSTRACT

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Field and greenhouse studies were conducted to evaluate the resistance of seed- and vegetatively propagated bermudagrass entries (*Cynodon* spp.) to spring dead spot caused by *Ophiosphaerella herpotricha*. In Kansas greenhouse studies, *O. herpotricha* caused root discoloration and root weight reductions in all entries tested. However, in Kansas field plots, root weight reductions were not different among entries and were not correlated with disease severity ratings. In an inoculated field study in Oklahoma, diseased areas ranged from 47 cm² for the entry Jackpot to 262 cm² for Poco Verde in 1995, and from 121 to 1,810 cm² for the entries Guymon and Common in 1996. African bermudagrass (*Cynodon transvaalensis*) exhibited the greatest number of live shoots per diseased area in both years, due in part to its greater shoot density, but also indicating greater potential to recover from the disease. African bermudagrass, Guymon, Sundevil, Midlawn, Midfield, Ft. Reno, Mirage, and several experimental seed-propagated entries were most resistant to spring dead spot, having the lowest diseased area and greatest number of live shoots within diseased areas. In Oklahoma, severity of spring dead spot among bermudagrass entries was correlated with freeze injury that occurred during the first winter after planting.

Additional keywords: *Cynodon dactylon*, *Ophiosphaerella korrae*

Spring dead spot (SDS) is a common and often serious disease of bermudagrass (*Cynodon dactylon* and *C. dactylon* × *C. transvaalensis*) in North America and Australia (13,14,17). Symptoms appear in early spring as dormant bermudagrass resumes growth and occur as circular, dead areas ranging from a few centimeters up to several meters in diameter. In severe cases, dead areas coalesce to encompass a much larger area of turf, which may be mistaken for winter kill or winter desiccation. Bermudagrass regrowth into dead areas is often slow and usually follows weed invasion. SDS is primarily a disease of mature stands of bermudagrass (usually 3 or more years old) that are intensively managed at low mowing heights and high nitrogen fertility. High incidences of SDS have been noted on turf that received heavy applica-

tions of nitrogen in late summer and fall (11).

SDS is caused by at least three different ectotrophic, root-rotting fungi. *Ophiosphaerella korrae* (= *Leptosphaeria korrae*) and *L. narmari* incite the disease in Australia (14,17); whereas *O. herpotricha* and *O. korrae* have been reported to cause symptoms in North America (3,5,16). The geographic distribution of these SDS pathogens in North America is still not clear, although isolations from SDS patches in Kansas, Oklahoma, and Texas suggest that *O. herpotricha* is the primary cause of SDS in the southern Great Plains region (16). *O. korrae* has only recently been isolated (1995) from symptomatic bermudagrass in Oklahoma (N. A. Tisserat, unpublished data).

The severity of SDS can be reduced, although not necessarily eliminated, by cultural practices (11). Avoidance of heavy, late-season nitrogen fertilization, insurance of adequate drainage, and reduction of soil compaction and thatch help suppress symptoms. Ammonium-based nitrogen and potassium fertility programs also have been shown to reduce disease severity (4,12). Several fungicides are labeled for control of SDS; however, disease control often has been inconsistent. Also, fungicides must be applied in fall and winter,

and sometimes early spring, before symptoms are present (10,13).

A promising approach to management of SDS disease may be the use of resistant bermudagrass cultivars. Furthermore, because SDS severity is often enhanced by cold temperatures, cold-hardy bermudagrass cultivars may be better able to resist the disease (11,16). At Oklahoma State University, a breeding project is ongoing to develop seed- and vegetatively propagated bermudagrass cultivars with superior winter hardiness, turf quality characteristics, and disease resistance. The purpose of this study was to evaluate commercial and experimental bermudagrass entries for resistance to SDS in the greenhouse and the field.

MATERIALS AND METHODS

Greenhouse experiments. In Kansas, bermudagrass entries (Table 1) were inoculated in the greenhouse to determine their susceptibility to *O. herpotricha*. Stolons, 2 to 5 cm long, were collected from bermudagrass in field plots in Wichita. Stolons were surface-sterilized for 3 min in 0.5% NaOCl, rinsed in distilled water, and placed in pots (20 cm diameter) containing a steamed soil:perlite:sand mixture (1:1:1 vol/vol/vol). Sample stolon pieces were placed on acidified potato dextrose agar (APDA), but no ectotrophic fungi were recovered. After establishment, five stolon pieces (each approximately 2 cm long), each containing at least one shoot, were transferred to a 6 × 25 cm Cone-tainer and allowed to grow for 4 months. Five pots of each bermudagrass entry were then inoculated by inserting 3 g of oats infested with *O. herpotricha* (KS28) per pot into the soil surrounding roots and stolons. Oat inoculum was prepared as previously described (16). Soil in five additional pots of each entry was amended with 3 g of sterile oats as controls. Pots were placed in the greenhouse in a randomized complete block factorial (bermudagrass entry × inoculum) arrangement with five replicates. Plants were fertilized at the start of the experiment with 4 g of Osmocote 14-14-14 (Sierra Chemical Co., Milpitas, CA) and watered daily as needed. Air temperatures during the experiment ranged from 15 to 30°C.

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Plants were maintained in the greenhouse for 120 days, then washed with water to remove soil from roots. Roots were rated for the percentage of root surface area discolored by *O. herpotricha* as an indication of the amount of root rot present. Roots were then oven-dried for 96 h at 50°C and weighed. Since bermudagrass roots in all pots amended with sterile oats exhibited <5% discoloration, root discoloration percentages were compared only among entries inoculated with *O. herpotricha*. Root weight reductions resulting from inoculations were determined by subtracting the root weight of inoculated from noninoculated plants in each block, dividing by the root weight of the noninoculated plants, and multiplying by 100. Data were subjected to analysis of variance, and means were separated using Fisher's (protected) least significant difference (LSD) test ($\alpha = 0.05$) (15). The experiment was conducted twice.

Field experiments. Sixteen entries of bermudagrass (Table 1) were established

from stolons or seed at the Kansas State University Horticultural Research Center, Wichita, in a Canadian Aldeck sandy loam in May 1986. Selections were planted in 3×3 m plots in a randomized complete block design with three replicates. Many of the entries did not survive more than 1 or 2 years because of winter kill. In a second study, 26 bermudagrass entries (Table 1) were established at the Oklahoma State University Turfgrass Research Center, Stillwater, in August 1993. Twenty-three of the bermudagrass entries were established from seed at a rate of 49 kg of pure live seed per ha, and three bermudagrass entries were established vegetatively from 8-cm-diameter plugs established on 30-cm centers on a Kirkland silt loam in 1.5×3.0 m plots arranged as previously described. Upon plot establishment at both sites, the turf was maintained at a 1.3-cm mowing height, with mowing performed three times per week using a reel mower. The turf received 49 kg N/ha/month from May through September throughout the experi-

ments. Soil acidity was 6.8, and P and K were maintained at adequate levels for turf growth.

Bermudagrass entries in Wichita were inoculated with a mixture of four Kansas isolates (KS2, KS3, KS11, and KS23) of *O. herpotricha* in September 1987; whereas a single isolate from Stillwater (KS188) was used to inoculate plots in Oklahoma in September 1994. In Wichita, two soil-turf plugs were removed to a depth of 10 cm from each plot using a standard 10-cm-diameter (84-cm² area) golf cup cutter. Ten grams of nondried infested oats was placed into one of the holes; the remaining hole was amended with 10 grams of sterile oats. The plugs were then replaced, and the turf was irrigated to prevent desiccation. Inoculations in Stillwater were identical except that six soil-turf plugs were removed from each plot; three were inoculated with *O. herpotricha*-infested oats, and three were treated with sterile oats. Plugged areas within each plot were mapped in order to monitor disease development during subsequent years.

Disease evaluations in Wichita were made in May 1989 and 1991. Disease ratings were not taken in 1990 because of extensive winter damage to many entries. Because plots had a high level of natural infection in addition to symptoms at the inoculation site, each bermudagrass plot was rated visually for the percent area damaged by spring dead spot.

Winter kill ratings were made in April 1994 in Stillwater. A 0.5×0.5 m grid containing 48 contact points was randomly tossed twice onto each plot. Percent winter kill was determined by dividing the number of contact points intersecting live green shoots by 48. In addition, bermudagrass green up was determined visually on a 1 to 9 scale, where 1 = dead or dormant turf and 9 = completely green, healthy turf. SDS evaluations were made during bermudagrass green up in April 1995 and 1996 and when disease symptoms were most prominent. The spots completely recovered in May with the onset of rapid bermudagrass growth. The diameter of each necrotic spot was measured in two directions, and the averaged value was used to calculate each area of disease. Values reported for diseased area represent the average of the three inoculated areas taken per replication for a total of nine area measurements taken per entry.

Winter kill, green up, and disease area data were subjected to analysis of variance, and means were separated using Fisher's protected LSD test ($\alpha = 0.05$) (15). The numbers of live shoots contained within the SDS areas were counted at the same time as the area measurements were made. Data were subjected to analysis of covariance (15). The number of shoots within a 46-cm² plug was determined for each bermudagrass treatment during July 1996 and was used as the covariate in the analysis.

Table 1. Bermudagrass entries tested for reaction to *Ophiosphaerella herpotricha*

Entry	Species	Origin/source	Location tested
Commercial seeded			
Common	<i>Cynodon dactylon</i>	Arizona	KS, OK
Poco Verde	<i>C. dactylon</i>	Desert Sun Marketing Co.	OK
NuMex Sahara	<i>C. dactylon</i>	Farmers Marketing Corp.	OK
Primavera	<i>C. dactylon</i>	Farmers Marketing Corp.	OK
NMS-4	<i>C. dactylon</i>	Farmers Marketing Corp.	KS
Cheyenne	<i>C. dactylon</i>	Jacklin Seed Co.	OK
Jackpot	<i>C. dactylon</i>	Jacklin Seed Co.	OK
Sundevil	<i>C. dactylon</i>	Jacklin Seed Co.	OK
Sonesta	<i>C. dactylon</i>	Scotts Co.	KS, OK
NM-72	<i>C. dactylon</i>	New Mexico State University	KS
Guymon	<i>C. dactylon</i>	Johnston Seed Co.	OK
Mirage	<i>C. dactylon</i>	International Seeds, Inc.	OK
Pyramid	<i>C. dactylon</i>	International Seeds, Inc.	OK
Tropica	<i>C. dactylon</i>	Turf Merchants	OK
C ²	<i>C. dactylon</i>	Finelawn Research	OK
Experimental seeded			
OKS (8 numbered entries)	<i>C. dactylon</i>	Oklahoma State University	OK
Ft. Reno	<i>C. dactylon</i>	Oklahoma State University	OK
African bermudagrass	<i>C. transvaalensis</i>	Oklahoma State University	OK
Experimental vegetative			
A-29	<i>C. dactylon</i> × <i>C. transvaalensis</i>	Kansas State University	KS
Commercial vegetative			
Midlawn	<i>C. dactylon</i> × <i>C. transvaalensis</i>	Kansas State University Research Foundation	KS, OK
Midfield	<i>C. dactylon</i> × <i>C. transvaalensis</i>	Kansas State University Research Foundation	KS, OK
Midiron	<i>C. dactylon</i> × <i>C. transvaalensis</i>	Kansas State University Research Foundation	KS
MS-Choice	<i>C. dactylon</i> × <i>C. transvaalensis</i>	Mississippi State University	KS
Tifgreen	<i>C. dactylon</i> × <i>C. transvaalensis</i>	USDA/ARS, Tifton, GA	KS
Tifway (II)	<i>C. dactylon</i> × <i>C. transvaalensis</i>	USDA/ARS, Tifton, GA	KS
Texturf 10	<i>C. dactylon</i>	Texas A&M University	KS
Vamont	<i>C. dactylon</i>	Virginia Polytechnic & State University	KS
Tufcote	<i>C. dactylon</i>	National Plant Materials Center, Beltsville, MD	KS
Tifton 10	<i>C. dactylon</i>	USDA/ARS, Tifton, GA	OK
Quickstand Common	<i>C. dactylon</i>	University of Kentucky	KS

This was done in order to account for differences in live shoot counts that were due to differences in turf density inherent within each entry and not attributable to disease recuperative capacity.

RESULTS

In Kansas, *O. herpotricha* caused significant root discoloration to all bermudagrass entries tested in the greenhouse. Although differences in root discoloration were detected among inoculated bermudagrass entries, these ratings were not correlated ($r = 0.03$) with root weight reductions (Table 2). Therefore, root discoloration ratings may more accurately reflect a combination of both ectotrophic fungal growth and root or stolon rot. Root discoloration and weight reductions in the greenhouse also were not consistent with disease severity ratings in field plots in 1989 or 1991 (Table 2). For example, the entries Midiron and Tifgreen had similar disease severity ratings in the greenhouse but were significantly different in susceptibility to SDS in the field. A second greenhouse study (data not shown) also showed extensive root discoloration and weight reduction in all bermudagrass entries 4 months after inoculation with *O. herpotricha*, but these ratings were not consistent with field observations. Therefore, the greenhouse inoculation techniques we used were not useful in predicting the relative resistance of bermudagrass entries to SDS.

In Oklahoma, African bermudagrass, Guymon, Midlawn, and several Oklahoma State seed-propagated entries were least affected by winter temperatures following

establishment (Table 3). In general, bermudagrass cultivars, especially seed-propagated cultivars, are most susceptible to freeze injury during the first winter after establishment. All entries recovered from freeze injury resulting during the winter of 1993-94. No apparent injury occurred during the winters of 1994-95 or 1995-96.

SDS symptoms in Oklahoma were observed 7 months (April 1995) after inoculation (Table 4). All of the bermudagrass entries showed some symptoms after inoculation, but no symptoms were observed in areas inoculated with sterile oats. Disease severity was greater in 1996 than in 1995; however, relative disease resistance among the entries was consistent for both years. African bermudagrass, Guymon, Sundevil, Midlawn, Midfield, Ft. Reno, Mirage, and several Oklahoma State seed-propagated entries exhibited the greatest resistance to SDS in terms of area of diseased turf. Conversely, dead spots in the susceptible entry Common expanded to greater than 20 times the original area of inoculation within 2 years. Disease ratings were consistent with those bermudagrass entries common to both the Kansas and Oklahoma field studies (Tables 2 and 4).

Inherent shoot density of African bermudagrass ranged from 2.5 to 5 times greater than the other bermudagrass entries (data not shown). Consequently, the greatest number of live shoots was counted within the diseased areas of African bermudagrass even though the data were adjusted for shoot density (Table 5). Furthermore, disease severity in 1995

correlated with winter kill severity and green up in 1994 (Table 6).

DISCUSSION

The lack of correlation between greenhouse and field SDS ratings noted in this study and by Crahay et al. (3) following inoculations with *O. korrae* suggests that factors other than direct colonization and rotting of roots influence disease severity. Extensive root rot of bermudagrass after prolonged exposure to the fungus at optimal temperatures for disease in the greenhouse probably does not accurately reflect disease development in the field. Nus and Shashikumar (12) reported that root colonization by *O. herpotricha* and *O. korrae* reduced freezing resistance of bermudagrass. We did not attempt to evaluate the susceptibility of bermudagrass genotypes to *O. korrae* because this fungus was only recently identified in Oklahoma, and it did not occur in plots. Therefore, disease severity ratings we report here may vary in other regions of the United States or Australia where *O. korrae* or *L. narmari* are the primary pathogens.

Table 2. Severity of spring dead spot in the greenhouse and in the field in bermudagrass entries following inoculation with *Ophiophora herpotricha* in Kansas

Entry	Greenhouse inoculations		Field inoculations	
	% root weight reduction ^x	Root discoloration ^y	(% plot area damaged) ^z	
			1989	1991
Midiron	17.2	50	0	4.0
Midlawn	38.5	52	0.8	6.2
Midfield	32.0	50	0.6	7.8
A-29	8.9	60	1.1	12.9
Quickstand Common	18.8	64	0.4	20.6
Texturf 10	6.8	60	2.2	24.7
Vamont	13.2	64	2.2	24.7
Tifgreen	24.8	48	4.0	36.1
Tufcote	31.4	56	0.7	Winter kill
MS-Choice	-2.0	56	1.8	Winter kill
Tifway II	26.8	44	2.5	Winter kill
Tifway	9.8	44	3.6	Winter kill
NM-72	28.2	48	Winter kill	Winter kill
Sonesta	20.6	64	Winter kill	Winter kill
NMS-4	10.0	64	Winter kill	Winter kill
Common	54.6	64	Winter kill	Winter kill
LSD (0.05)	NS	15	2.1	14.7

^x Root weight reductions determined by subtracting the root weights of inoculated from noninoculated plants within the same block, dividing by the root weight of the noninoculated plants, and multiplying by 100. Negative values in the table represent a net increase in root weight following inoculation.

^y Mean percent root discoloration 120 days after inoculation. Roots on all bermudagrass entries amended with sterile oats exhibited <5% root discoloration and were not included in the analysis.

^z Percent plot area damaged determined visually in May 1989 and 1990. Those entries that could not be rated for spring dead spot damage because of severe winter injury are indicated.

Table 3. Green up and winter kill evaluations for bermudagrass entries in April 1994 following planting in August 1993 in Oklahoma

Entry	Green up ^w	Winter kill (%)
Common	1	98
Poco Verde	1	98
Jackpot	2	92
Sonesta	2	98
Tifton 10 ^x	5	... ^y
Primavera	1	98
OKS ^z 91-1	2	94
NuMex Sahara	2	98
Pyramid	2	95
Cheyenne	2	94
Tropica	1	95
OKS 91-2	2	84
C ²	1	98
OKS 91-6	3	79
Mirage	3	60
Ft. Reno	5	39
Midfield ^x	5	... ^y
OKS 91-13	5	46
Midlawn ^x	6	... ^y
OKS 91-4	6	28
Sundevil	5	34
OKS 91-3	5	49
OKS 89-3	6	23
OKS 91-12	6	13
African bermudagrass	6	14
Guymon	6	15
LSD (0.05)	1	20

^w On a scale of 1 to 9, with 1 = dead or dormant turf and 9 = completely green turf.

^x Midfield, Midlawn, and Tifton 10 were vegetatively propagated. The remaining entries were seed-propagated.

^y Winter kill evaluations were not taken for bermudagrasses that were vegetatively propagated.

^z OKS = Oklahoma State University experimental seed-propagated entries.

Table 4. Severity of spring dead spot for bermudagrass entries in 1995 and 1996 following inoculation with *Ophiosphaerella herpotricha* in 1994 in Oklahoma

Entry	Average diseased area ^x (cm ²)	
	4/18/95	4/18/96
Common	230	1,810
Poco Verde	262	1,749
Jackpot	219	1,712
Sonesta	197	1,658
Tifton 10 ^y	180	1,541
Primavera	179	1,433
OKS ^z 91-1	194	1,432
NuMex Sahara	165	1,344
Pyramid	195	1,288
Cheyenne	185	1,091
Tropica	137	1,034
OKS 91-2	246	971
C ²	156	755
OKS 91-6	176	653
Mirage	99	601
Ft. Reno	64	552
Midfield ^y	63	473
OKS 91-13	107	396
Midlawn ^y	102	360
OKS 91-4	155	353
Sundevil	47	316
OKS 91-3	95	303
OKS 89-3	126	239
OKS 91-12	105	218
African bermudagrass	103	140
Guymon	80	121
LSD (0.05)	87	538

^x Area of inoculation (area of cup cutter) = 84 cm².

^y Midfield, Midlawn, and Tifton 10 were vegetatively propagated. The remaining entries were seed-propagated.

^z OKS = Oklahoma State University experimental seed-propagated entries.

The influence of cold temperatures on disease development and severity is consistent with observations that SDS only develops after winter dormancy and cultivars with increased winter hardiness are less susceptible to the disease (1,2,11,16). Gatschet et al. (6,7) found higher levels of a chitinase protein in crown tissue of the cold hardy Midiron bermudagrass cultivar compared with the less cold hardy Tifgreen following cold acclimation. Chitinases are thought to function in disease resistance by attacking cell walls of invading fungal pathogens and signaling plant defense response (8). Furthermore, several chitinases possess antifreeze activity, which is thought to protect sensitive tissue from damage at near-freezing temperatures (9). Chitinase involvement in both disease and cold resistance suggests that this protein may partially explain the correlation between bermudagrass cold hardiness and resistance to SDS in the field.

Overall, the bermudagrass entries found to have the smallest area of SDS also had the greatest number of live shoots within each area. Therefore, these data indicate that disease area measurements together

Table 5. Least squares means^x representing the number of live shoots per diseased area for bermudagrass entries in 1995 and 1996 following inoculation with *Ophiosphaerella herpotricha* in 1994 in Oklahoma

Entry	No. of live shoots/ diseased area	
	4/18/95	4/18/96
African bermudagrass	5.9 a	8.4 a
Midlawn ^y	2.6 b	3.0 b
OKS 89-3 ^z	1.0 cd	2.5 bc
OKS 91-13	1.1 cd	2.4 bc
Mirage	1.9 bc	2.3 bc
OKS 91-12	1.6 bc	2.3 bc
Midfield ^y	1.4 c	2.2 bc
Ft. Reno	0.9 cd	2.2 bc
Sundevil	0.7 cd	1.8 c
OKS 91-4	0.3 cd	1.8 c
Guymon	0.7 cd	1.7 cd
OKS 91-3	1.1 cd	1.6 cd
OKS 91-6	0.1 d	0.8 d
OKS 91-2	0.3 cd	0.7 d
Pyramid	0.3 cd	0.7 d
Cheyenne	0.3 cd	0.7 d
Tifton 10 ^y	0.4 cd	0.7 d
Tropica	-0.1 d	0.6 d
Jackpot	0.0 d	0.5 d
NuMex Sahara	0.1 d	0.5 d
Primavera	0.2 d	0.4 d
Common	-0.1 d	0.3 d
OKS 91-1	0.3 cd	0.3 d
Poco Verde	-0.2 d	0.2 d
Sonesta	0.2 d	0.2 d
C ²	0.6 cd	0.1 d

^x Least squares means within a column followed by different letters are significantly different (alpha = 0.05). Covariate = shoot density measured in July 1996.

^y Midfield, Midlawn, and Tifton 10 were vegetatively propagated. The remaining entries were seed-propagated.

^z OKS = Oklahoma State University experimental seed-propagated entries.

with live shoot counts within spring dead spots are useful indicators of the relative degree of resistance among bermudagrass cultivars. We are continuing to evaluate bermudagrass entries for resistance to SDS causal agents and to determine the molecular mechanism(s) responsible for SDS resistance and cold hardiness in bermudagrass. In summary, our results indicate that use of resistant bermudagrass cultivars may be an effective management strategy to reduce severity of SDS caused by *O. herpotricha*.

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LITERATURE CITED

- Anderson, J. A., Kenna, M. P., and Taliaferro, C. M. 1988. Cold hardiness of 'Midiron' and 'Tifgreen' bermudagrass. HortScience 23:748-750.
- Anderson, J. A., Taliaferro, C. M., and Martin, D. L. 1993. Evaluating freeze

Table 6. Pearson correlation coefficients (*r*) and probability (*P*) values for the effects of bermudagrass winter kill and spring green up in 1994 on spring dead spot disease severity in 1995 in Oklahoma

1995	1994	
	Winter kill	Green up
Diseased area	<i>r</i> 0.56 <i>P</i> 0.0001	0.20 0.002
Live shoots/ diseased area	<i>r</i> -0.42 <i>P</i> 0.0003	-0.64 0.0001
Live shoots ^z / diseased area	<i>r</i> -0.26 <i>P</i> 0.032	-0.61 0.0001

^z Adjusted for shoot density measured in July 1996.

- resistance of bermudagrass in a controlled environment. HortScience 28:955.
- Crahay, J. N., Dernoeden, P. H., and O'Neill, N. R. 1988. Growth and pathogenicity of *Leptosphaeria korrae* in bermudagrass. Plant Dis. 72:945-949.
- Dernoeden, P. H., Crahay, J. N., and Davis, D. B. 1991. Spring dead spot and bermudagrass quality as influenced by nitrogen source and potassium. Crop Sci. 31:1674-1680.
- Endo, R. M., Ohr, H. D., and Krausman, E. M. 1985. *Leptosphaeria korrae*, a cause of the spring dead spot disease of bermudagrass in California. Plant Dis. 69:235-237.
- Gatschet, M. J., Taliaferro, C. M., Anderson, J. A., Porter, D. R., and Anderson, M. P. 1994. Cold acclimation and alteration in protein synthesis in bermudagrass crowns. J. Am. Soc. Hortic. Sci. 119:477-480.
- Gatschet, M. J., Taliaferro, C. M., Porter, D. R., Anderson, M. P., Anderson, J. A., and Jackson, K. W. 1996. A cold-regulated protein from bermudagrass crowns is a chitinase. Crop Sci. 36:712-718.
- Graham, L. S., and Sticklen, M. B. 1994. Plant chitinases. Can. J. Bot. 72:1057-1083.
- Hon, W.-C., Griffith, M., Mlynarz, A., Kowk, Y. C., and Yang, D. S. C. 1995. Antifreeze proteins of winter rye are similar to pathogenesis-related proteins. Plant Physiol. 109:879-889.
- Lucas, L. T. 1980. Control of spring dead spot of bermudagrass with fungicides in North Carolina. Plant Dis. 64:868-870.
- Lucas, L. T. 1980. Spring deadspot of bermudagrass. Pages 183-187 in: Advances in Turfgrass Pathology. B. G. Joyner and P. O. Larsen, eds. Harcourt Brace Jovanovich, Duluth, MN.
- Nus, J. L., and Shashikumar, K. 1993. Fungi associated with spring dead spot reduces freezing resistance in bermudagrass. HortScience 28:306-307.
- Smiley, R. W., Dernoeden, P. H., and Clarke, B. B. 1992. Compendium of Turfgrass Diseases. 2nd ed. American Phytopathological Society, St. Paul, MN.
- Smith, A. M. 1971. Spring dead spot of couchgrass in New South Wales. J. Sports Turf Res. Inst. 47:54-59.
- Steel, R. G. D., and Torrie, J. H. 1980. Principles and Procedures of Statistics—A Biometrical Approach. McGraw-Hill, New York.
- Tisserat, N. A., Pair, J. C., and Nus, A. 1989. *Ophiosphaerella herpotricha*, a cause of spring dead spot of bermudagrass in Kansas. Plant Dis. 73:933-937.
- Walker, J., and Smith, A. M. 1972. *Leptosphaeria narmari* and *L. korrae* sp. nov., two long-spored pathogens of grasses in Australia. Trans. Br. Mycol. Soc. 58:459-466.