Heritability of Dollar Spot Resistance in Creeping Bentgrass

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ABSTRACT

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The dollar spot disease incited by *Sclerotinia homoeocarpa* is an important disease of creeping bentgrass (*Agrostis stolonifera*). Genetic resistance is an important control strategy and could reduce fungicide use. Despite recent research, the genetic mechanism of dollar spot resistance in turfgrasses is still not fully understood. The objectives of this study were to (i) determine narrow-sense heritability and predicted gain from selection for dollar spot resistance in creeping bentgrass and (ii) evaluate inheritance characteristics of dollar spot disease resistance. Inheritance characteristics such as the detection of major genes, heterosis, maternal effects, and combining ability were determined by evaluating the disease

severity of progeny from crosses between resistant and susceptible bentgrass clones. Parental clones and progenies from crosses were established in a field trial in a randomized complete block design and inoculated with one isolate of *S. homoeocarpa* applied at a rate of 0.25 g m⁻² of prepared inoculum. Differences in progeny means between crosses were observed over both years. Progeny from resistant × resistant crosses had significantly less disease severity than resistant × susceptible and susceptible × susceptible crosses. High narrow-sense heritability estimates (0.79 [2002], 0.79 [2003]) and large mean squares for general combining ability support the idea that additive gene action plays a significant role in disease resistance and support previous research that dollar spot resistance is most likely quantitatively inherited.

Dollar spot disease incited by *Sclerotinia homoeocarpa* F.T. Bennet is one of the main disease problems on close-cut creeping bentgrass (*Agrostis stolonifera* L.). More than 70% of fungicides used on golf courses are used to control dollar spot, brown patch (*Rhizoctonia solani* Kuhn.), and anthracnose (*Colletotrichum graminicola* (Ces.) G.W. Wils.) diseases (Meritz Marketing Research Inc., St. Louis, MO).

The utilization of cultivars with genetic resistance to dollar spot disease would be a promising control strategy compared with costly fungicide applications. Genetic resistance to dollar spot disease has been identified in creeping bentgrass clones (3,4). Additionally, some cultivars of creeping bentgrass have been identified that exhibit apparent partial resistance to dollar spot (15,16,19). However, no cultivars currently in production are considered completely resistant to *S. homoeocarpa*. One of the reasons for this may be that the severity of dollar spot disease is also partly dependent on the environment. Vincelli et al. (19) and Bonos et al. (5) documented that dollar spot disease varied over years due to environmental conditions. Bonos et al. (5) also found that disease resistance among most creeping bentgrass clones was unstable across environments.

Previous research on the inheritance of resistance reported high broad-sense heritability estimates (0.90) based on variance components of 265 replicated creeping bentgrass clones inoculated with a mixture of five isolates of the fungus (4). The crosses evaluated in the previous study were inoculated with a mixture of three different isolates. Authors indicated that dollar spot resistance may be quantitatively inherited with a minimum of two to five genes involved in resistance. However, since it is possible that different races of the fungus may be interacting with different resistance loci, it is unknown whether the results observed were due to major resistance genes specific to each isolate or due to a larger number of genes indicating quantitative disease resistance

(4). Therefore, this current study was designed to evaluate the inheritance of dollar spot resistance based on inoculation with a single isolate of the pathogen. Additionally, the previous study reported results on the disease response of a limited number of crosses (i.e., no resistant × resistant crosses) and a limited number of progeny from crosses (i.e., only 21 to 35 from susceptible × susceptible crosses) and only reported results from 1 year. This study was conducted to provide additional information from a larger number of crosses, more progeny from each cross and results from 2 years of evaluation.

Furthermore, the previous study only reported results for broad-sense heritability. Broad-sense heritability estimates include all genetic effects (additive, dominance, and epistatic), while narrow-sense heritability estimates include only additive variance (17). Breeding for resistance to quantitative traits involves shifting the population mean toward resistance (17). In order to do this, breeding programs rely heavily on additive genetic variation for successful population improvement toward more resistant phenotypes. Since dollar spot resistance in creeping bentgrass is proposed to be quantitatively inherited, narrow-sense heritability would be more useful to plant breeders than broad-sense heritability. Narrow-sense heritability estimates for dollar spot resistance have not yet been reported for any turfgrass species. Therefore, this study was conducted to determine the narrow-sense heritability of dollar spot resistance in creeping bentgrass.

The objectives of this study were twofold: (i) determine narrow-sense heritability estimates and predicted gain from selection of dollar spot resistance in creeping bentgrass and (ii) evaluate inheritance characteristics of dollar spot disease resistance (major gene calculations, heterosis, maternal effects, and combining ability) of pseudo F₂ progeny from crosses between fixed resistant and susceptible clones.

MATERIALS AND METHODS

Crosses and field trial establishment. Three resistant (Penn-cross-2 [Penn-2], L93-10, and 7335-2) and two susceptible (Crenshaw-5 [Cren-5] and 7418-3) (Table 1) creeping bentgrass

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clones were selected from a previous study described by Bonos et al. (4). Controlled crosses between these clones were made in 2001. Clones were used as both male and female parents to develop full-sib pseudo F₂ (F₁) progeny (9). Progeny from these plants were F₁'s but were considered pseudo F₂ because these plants had not been test-crossed or inbred prior to their selection and are most likely heterozygous at many loci due to the cross-pollinated nature (1) of creeping bentgrass. The parents Penn-2, L93-10, and Cren-5 were selected from cvs. Penncross, L93, and Crenshaw, respectively; the parent 7418-3 was collected from Piping Rock Golf Course in Long Island, NY, and the parent 7335-2 was collected from Harkers Hollow Golf Course in Harmony, PA. All possible combinations of crosses were attempted (susceptible × susceptible, resistant × susceptible, and resistant × resistant), yet not all were successful. A full description of the crosses is presented in Table 2.

Plants of each vernalized clone were induced to flower with 18 to 20 h of supplemental overhead lighting in the greenhouse. Just before anthesis, individual plants of each clone were matched together and isolated from all other crosses with plastic walls. One individual plant of each clone was also isolated to measure

the degree of selfing in each of the clones. Pollen movement was enhanced with manual tapping of the inflorescences.

Seed was harvested from both plants in the cross, dried, and then treated with 0.2% KNO₃ to induce germination. Individual seedlings were put in seedling flats described above and transplanted to the field on 10 October 2001, along with at least eight vegetative replicates of the parental clones used in the crosses. Between 12 and 22 progeny were considered subsamples of one replicate of each cross depending on the number of progeny generated from each cross. Progeny were separated equally for each replicate within each cross. The parents and progeny plants were arranged in a randomized complete block design with four replicates. Plants were maintained as mowed spaced-plants at a height of approximately 1.88 cm with a Toro 216 Reel Mower (Toro Corporation, Bloomington, MN). Progeny were inoculated with one isolate of *S. homoeocarpa* (H2—isolated from Crenshaw creeping bentgrass, described by Bonos et al. [4]), at a rate of 0.25 g m⁻² of prepared inoculum, on 24 June 2002 to facilitate uniform infection across the field. The field was inoculated once during the 2-year evaluation period.

Dollar spot symptoms appeared 2 weeks after inoculation and plants were rated several times during the growing season of each year (June through September) in 2002 (six rating dates) and 2003 (two rating dates) using a 1-to-9 rating scale: 9 represented 0 to 5% diseased turf, 8 represented approximately 10% diseased turf, 7 represented approximately 15 to 25% diseased turf, 6 represented approximately 30 to 40% diseased turf, 5 represented 40 to 50% diseased turf, 4 represented approximately 60 to 70% diseased turf, 3 represented approximately 75 to 85% diseased turf, 2 represented approximately 90% diseased turf, and 1 represented 95 to 100% diseased turf. Due to the nonlinearity of the 1-to-9 rating scale, these data were transformed to percent disease (6). All data analysis was conducted on percent disease using the average of rating dates for each year.

TABLE 1. Dollar spot disease severity of five parental clones used in controlled crosses generated in 2001^a

Parent	Dollar spot disease severity (%)
L93-10 (R)	6
7335-2 (R)	19
Penn-2 (R)	24
Cren-5 (S)	60
7418-3 (S)	62
LSD _{0.05}	9.1

^a Parental creeping bentgrass clones were established in a mowed spaced-plant trial and evaluated for 2 years (2002 and 2003) after inoculation with *Sclerotinia homoeocarpa* in 2002. R, resistant; S, susceptible.

TABLE 2. Description of controlled crosses between dollar spot resistant and susceptible creeping bentgrass clones including heterosis calculations^a

Cross no.	Female × male ^b	Number of progeny	Year	Mid-parent mean disease severity	SD	Pseudo F ₂ progeny mean disease severity		Hetosis <i>t</i> test ^c		<i>P</i> value ^d
						SD			df	
1.	7418-3 × Cren-5 (S×S)	46	2002	51	18.1	43	5.7	−1.5	12	ns
			2003	75	20.6	82	3.3	1.3	12	ns
2.	Cren-5 × 7418-3 (S×S)	43	2002	51	18.1	46	8.2	−0.7	10	ns
			2003	75	20.6	80	10.4	0.7	9	ns
3.	7418-3 × 7335-2 (S×R)	40	2002	39	9.4	42	11.1	0.5	4	ns
			2003	44	19.4	73	7.9	4.7	13	**
4.	7335-4 × 7418-3 (R×S)	29	2002	39	9.4	42	3.9	1.1	13	ns
			2003	44	19.4	78	10.7	4.7	9	**
5.	Cren-5 × 7335-2 (S×R)	45	2002	33	18.1	45	6.2	2.1	12	*
			2003	49	19.6	73	7.6	3.7	12	*
6.	7335-2 × Cren-5 (R×S)	34	2002	33	18.1	55	3.3	4.2	12	**
			2003	49	19.6	84	8.4	5.2	11	**
7.	Cren-5 × L93-10 (S×R)	47	2002	26	17.1	22	1.8	−0.9	11	ns
			2003	42	15.1	45	8.7	0.3	8	ns
8.	L93-10 × Cren-5 (R×S)	45	2002	26	17.1	27	7.2	0.2	11	ns
			2003	42	15.1	49	4.9	1.4	13	ns
9.	Penn-2 × L93-10 (R×R)	71	2002	14	9.2	13	2.7	−0.6	17	ns
			2003	16	13.1	28	10.0	−2.0	6	ns
10.	L93-10 × Penn-2 (R×R)	94	2002	14	9.2	10	2.6	−1.5	17	ns
			2003	16	13.1	28	14.1	2.1	6.5	ns
11.	L93-10 × 7335-2 (R×R)	70	2002	14	7.1	20	1.5	3.3	18	**
			2003	11	13.4	46	22.0	2.9	4	*
12.	7335-2 × L93-10 (R×R)	55	2002	14	7.1	26	4.3	4.4	8	**
			2003	11	13.4	48	10.4	6.0	6	**
	LSD _{0.05}		2002			7.9				
			2003			14.2				

^a Parents and progeny were established in mowed spaced-plant trial in the fall of 2001 at North Brunswick, NJ, inoculated with *Sclerotinia homoeocarpa* in June 2002 and evaluated for dollar spot disease in 2002 and 2003.

^b S = dollar spot susceptible, R = dollar spot resistant, selected as R or S from clonally replicated field trials evaluated in 1998 and 1999 (4).

^c Heterosis effects were tested by comparing pseudo F₂ population means with mid-parent means using a two sample *t* test.

^d * and ** denote significance at *P* < 0.05 and 0.01, respectively; ns is not significant.

Statistical analysis. Broad-sense heritability estimates were determined from restricted maximum likelihood (REML) variance and covariance components using the random model of Proc MIXED (SAS Institute, Cary, NC) of replicated parental clones (Table 3). Narrow-sense heritability was determined from mid-parent versus progeny regression analysis where the slope of the regression line (b) is equal to h^2 (8). Mid-parent and progeny means utilized in the analysis are presented in Table 2. Analysis was conducted separately for each year (Figs. 1 and 2). The standard error of the estimate and F tests of the regression equation were analyzed using Proc Reg (SAS Institute).

Gain from selection was calculated using the equation $G_s = i\sigma_p h^2$ (8,17), where G_s is the genetic gain from selection, i is constant based on the selection intensity (top 5% = 2.06), σ_p is the standard deviation of the phenotypic variance, and h^2 is the heritability.

The detection of major genes was determined by the equation proposed by Fain (7) and described by Lynch and Walsh (14): $\text{Var}(o_i) = a + b_1 \bar{o}_i + b_2 \bar{o}_i^2$, where $\text{Var}(o_i)$ is the phenotypic variance within the i th sibship and $\bar{o}_i$ is the mid-parental value for this sibship. A significant value of b_2 is taken as an indication of a major gene (14).

Heterosis, the comparison between progeny and mid-parent means, and maternal effects, the comparison of progeny of reciprocal crosses, were evaluated for significance with the two-sample t test (12).

General combining abilities (GCA) and specific combining abilities (SCA) were calculated for a subset of the crosses for

which a complete diallel set of crosses was available (L93-10, 7335-5, and Cren-5) using the DIALLEL-SAS (20) program. The analysis used model I method 1 (10), which included parents, pseudo F_2 progeny, and their reciprocals.

RESULTS

Dollar spot reaction of parents and progeny. Significant differences were observed between resistant and susceptible parent plants used for the crosses (Tables 1 and 3). L93-10 exhibited the least dollar spot disease of the parental clones. Individual “selfed” crosses of the parents yielded no progeny, indicating that seed production by accidental self-pollination was believed to be infrequent, and indicated a high level of self-incompatibility among these creeping bentgrass clones. Most grasses, including creeping bentgrass, have a high level of self-incompatibility (2,18).

Dollar spot disease was significantly more severe in 2003 than in 2002 based on the analysis of variance (ANOVA) from progeny means of controlled crosses (Table 4); therefore, data on progeny performance are reported for both years (Table 2). Differences in disease severity between years was thought to be due mainly to environmental effects as data from other trials conducted during the same time period exhibited similar results (data not shown). Significant differences between progeny means from crosses were also observed (Table 2 and 4). Progeny from resistant $\times$ resistant crosses exhibited less disease severity compared with resistant $\times$ susceptible and susceptible $\times$ susceptible crosses (Table 2). There were also significant differences between resistant $\times$ susceptible crosses depending on which parent clone was used in the cross (Table 2). For example, progeny means of L93-10 $\times$ Cren-5 (cross 8) and its reciprocal (cross 7) had significantly less disease severity than 7335-2 $\times$ Cren-5 (cross 6) and its reciprocal (cross 5) for both 2002 and 2003. Most population distributions of the

TABLE 3. Analysis of variance of dollar spot disease severity of five parental creeping bentgrass clones established in a mowed spaced-plant trial and evaluated for 2 years (2002 and 2003) after inoculation with *Sclerotinia homoeocarpa* in 2002

Sources of variation	df	Mean square	$P > F^a$	Variance component ^b
Year	1	1,406.61	0.007	...
Rep (year)	14	475.02	0.127	...
Parent clone	4	8,236.81	0.0009	609.37
Parent clone $\times$ year	4	579.69	0.006	37.32
Error = parent clone $\times$ year $\times$ rep	47	142.19	...	17.78
$H = 0.92^c$	...	...	...	...

^a F tests were determined by Satterthwaite approximation.

^b Variance components were determined using restricted maximum likelihood (REML) of Proc Mixed in SAS (SAS Institute, Cary, NC).

^c Heritability = σ^2_C/σ^2_P , 95% confidence interval for eight-plant mean heritability = 0.99 to 0.83; computed according to Knapp and Bridges (13).

Fig. 1. Mid-parent-offspring regression of pseudo F_2 population means regressed on the mid-parent value from 12 crosses between five creeping bentgrass parents evaluated for dollar spot resistance in 2002.

Fig. 2. Mid-parent-offspring regression of pseudo F_2 population means regressed on the mid-parent value from 12 crosses between five creeping bentgrass parents evaluated for dollar spot resistance in 2003.

TABLE 4. Analysis of variance of dollar spot disease severity of 12 crosses established in a mowed spaced-plant trial and evaluated for 2 years (2002 and 2003) after inoculation with *Sclerotinia homoeocarpa* in 2002

Sources of variation	df	Mean square	$P > F^a$
Year	1	17,307.51	<0.0001
Rep (year)	6	132.95	0.14
Cross	11	2,503.85	<0.0001
Cross $\times$ year	11	108.76	0.076
Error = cross $\times$ year $\times$ rep	66	64.05	...

^a F tests were determined by Satterthwaite approximation.

pseudo F_2 progeny of the crosses showed continuous variation (data not shown) similar to previous studies (4). There were no discrete reaction classes and transgressive segregation was observed in most crosses.

Broad- and narrow-sense heritability and gain from selection. Broad-sense heritability estimated from REML variance components from the Proc MIXED analysis of five parental clones with eight replications evaluated over 2 years and inoculated with one isolate was 0.92 (95% confidence interval = 0.99 to 0.83) (Table 3). This is consistent with the broad-sense heritability estimate (0.90) reported by Bonos et al. (4), who evaluated dollar spot resistance of 265 creeping bentgrass clones in two different environments over 2 years with six replications in each environment and inoculated with five isolates.

Narrow-sense heritability estimates based on mid-parent progeny regression analysis were also quite high (0.79 ± 0.18 , 2002 [Fig. 1]; 0.79 ± 0.18 , 2003 [Fig. 2]) and consistent across years (Figs. 1 and 2).

A 28% gain in 2002 and a 44% gain in 2003, assuming the top 5% of the population is selected, can be expected using phenotypic selection. This is slightly lower than the gain from selection reported by Bonos et al. (4) (68%-11-plant mean basis); however, this is expected since broad-sense heritability estimates were used in their calculation (4).

Detection of major genes. The detection of major genes using the equation proposed by Fain (7) and described by Lynch and Walsh (14) resulted in a P value of 0.46 for the quadratic b_2 term, indicating that a major gene was not segregating for the crosses evaluated in this study. These results are similar to that reported by Bonos et al. (4).

Heterosis and maternal effects. Progeny means of most of the controlled crosses were not consistently significantly different from the mid-parent mean (Table 2) based on the two-sample t test statistic (12). This is consistent with heterosis results reported by Bonos et al. (4). In contrast, progeny means of Cren-5 $\times$ 7335-2 (cross 5) and its reciprocal (cross 6) and L93-10 $\times$ 7335-2 (cross 11) and its reciprocal (cross 12) were consistently significantly different from their mid-parent values in both 2002 and 2003 indicating significant heterosis results for these crosses (Table 2). For both sets of crosses, mid-parent means were lower (exhibited less disease severity) than progeny means. Only these two crosses showed significant heterosis effects over both years, and both contained 7335-2 as a parent.

Maternal effects, determined by comparing progeny means from reciprocal crosses (Table 2), were not significant for the resistant $\times$ susceptible crosses or the susceptible $\times$ susceptible crosses for either year (data not shown). Maternal effects were significant for the resistant $\times$ resistant crosses (L93-10 $\times$ 7335-2 [crosses 11 and 12] and Penn-2 $\times$ L93-10 [crosses 9 and 10]) in 2002 but not in 2003. In contrast, Bonos et al. (4) reported significant maternal effects for susceptible $\times$ susceptible crosses only. Their study did not include resistant $\times$ resistant crosses and only reported results for 1 year. These results indicate that maternal effects did not play a significant role in dollar spot resistance and provide further evidence that disease severity is partly controlled by the environment.

Combining ability. GCA and SCA were calculated for a subset of crosses for which a complete diallel set of crosses was available. These included L93-10, Cren-5, and 7335-2 crossed in all possible combinations. GCA and SCA effects were both significant (Table 5) although mean squares for GCA were higher, indicating that GCA was more important in predicting the performance of progenies. The individual parental GCA estimates revealed both positive and negative values. L93-10 had the most negative GCA (-14.4) (since analysis was conducted on percent disease), indicating that this parent contributed the most toward disease resistance, Cren-5 had the most positive GCA (14.8), and 7335-2 had a close to neutral GCA estimate (-0.4).

DISCUSSION

The broad-sense heritability estimate reported in this study was consistent with the estimate reported by Bonos et al. (4) even though different creeping bentgrass clones were evaluated in different environments, and different numbers of isolates and replications were used. It should be noted however, that the estimate reported is specific for this population and the particular environments sampled. These data indicate the strong genetic component associated with dollar spot resistance in creeping bentgrass. Additionally, the high narrow-sense heritability estimate (0.79) reported in this study indicated that most of the genetic variation can be attributed to additive gene effects. The larger gain from selection predicted in 2003 (44%) versus 2002 (28%) indicates that selection during periods of higher disease pressure (2003) may be more effective in improving resistance than selection during periods of moderate disease pressure (2002). The high heritability estimates and the 28 to 44% gain from selection expected also indicate that selection should result in improved dollar spot resistance in subsequent generations. This is supported by the fact that progeny from resistant $\times$ resistant crosses had significantly less disease severity than resistant $\times$ susceptible and susceptible $\times$ susceptible crosses. This was also proven by a separate study that found experimental selections of creeping bentgrass selected for improved dollar spot resistance exhibited greater disease resistance than standard cultivars and selections not selected for dollar spot resistance (3).

The observation of continuous distribution of disease severity phenotypes among progeny reported here and by Bonos et al. (4) is taken as support for a large number of genes of roughly equal effect. This is an assumption, however, because the presence of major genes can be obscured if the environmental variation is large or if the major genes are at a low frequency (14). The significant year effect in the ANOVAs (Tables 3 and 4) indicated the presence of a strong environmental effect; however, the lack of significance in the quadratic term of the Fain calculation for major genes supports the idea that a major gene was not segregating for the crosses evaluated in this study and that environmental factors and/or other polygenes could contribute to the differences between crosses. Bonos et al. (4) also did not detect a major gene. The previous study conducted by Bonos et al. (4) evaluated the inheritance of resistance to several isolates, while the current study evaluated resistance to one isolate. Since similar results were observed in both studies, it indicates that the response observed was most likely not due to the interaction of major resistance genes to specific isolates and further supports the idea that dollar spot resistance is most likely inherited quantitatively.

However, the fact that high narrow-sense heritability estimates were observed could be an indication that only a few genes with large effects may control dollar spot resistance. Bonos et al. (4) reported the minimum the number of genes to be approximately two to five depending on the cross.

TABLE 5. Mean squares for dollar spot disease severity in a creeping bentgrass diallel with three parents (L93-10, 7335-2, and Cren-5) arranged in a randomized complete block design with four replications evaluated over 2 years

Sources of variation	df	Mean square ^a
Year	1	6,766.72***
Rep (year)	6	260.20*
Cross	8	3,520.91***
General combining abilities (GCA)	2	10,213.78***
Specific combining abilities (SCA)	3	2,379.18***
Cross $\times$ year	8	334.91**
GCA $\times$ year	2	587.53**
SCA $\times$ year	3	496.68**
Error	48	105.10

^a *, **, and *** indicate significant F tests at $P < 0.05$, 0.01, and 0.001, respectively.

A significant difference between mid-parent means and progeny means indicates the presence of heterosis and provides evidence for the presence of dominant or nonadditive genetic effects. Lack of consistently significant *t* tests between mid-parent and progeny means for most of the controlled crosses further support the strong environmental effect but also indicate that heterosis was not present for those crosses. These results suggest that additive and not dominance or epistasis may be the main gene effects involved in dollar spot disease resistance. *t* tests were significant, however, for two crosses (Cren-5 $\times$ 7335-2 and L93-10 $\times$ 7335-2) and their reciprocals for both years. In both crosses, progeny means were significantly higher (more disease susceptible) than mid-parent means indicating that heterosis for susceptibility could be involved in these particular crosses. Both of these crosses contained the parent 7335-2. Additionally, progeny from 7335-2 were significantly more susceptible to dollar spot in both years compared with progeny from L93 when both were crossed to susceptible Cren-5. These data indicate that 7335-2 may have some dominant or epistatic loci that affect dollar spot disease resistance, albeit toward susceptibility.

Results of combining ability analysis indicated that both SCA and GCA were significant; however, GCA had larger mean squares than SCA. This indicated that GCA, which is a measure of additive gene action, plays an important role in dollar spot disease resistance. These results along with the lack of detection of major genes and high narrow-sense heritability estimates further support the idea that additive gene action plays an important role in dollar spot disease resistance. Similar results were also reported for gray leaf spot (caused by the fungus *Pyricularia grisea*) resistance in perennial ryegrass (*Lolium perenne*) (11).

However, SCA estimates from the diallel analysis of these three parents were also significant. SCA analysis is used to indicate nonadditive gene action including dominant and epistatic gene effects. These data suggest that dominant gene effects could also play a role in dollar spot disease resistance especially in the 7335-2 parent plant. These data also support the heterosis results observed. This hypothesis is further supported by the fact that 7335-2 did not have a high negative GCA estimate even though it was considered a more resistant plant. On the other hand, L93-10 had the highest negative GCA estimate of the three parents in the diallel analysis indicating that L93-10 had a strong contribution toward reducing disease severity and improving resistance when it was used as a parent in crosses. These results indicate that there were differences in dollar spot severity and that different types of gene action may be involved in dollar spot resistance depending on the parent contribution. To our knowledge, this is the first report documenting the importance of parent selection in improving dollar spot resistance in creeping bentgrass.

It should be noted that the combining ability analysis reported here was conducted with only three parents. A more useful combining ability study should include a larger number of parents with different disease responses.

In summary, the results reported here, including the high narrow-sense heritability estimate (0.79) and lack of detection of a major gene, are consistent with previous research and support the idea that additive gene action is important in dollar spot disease resistance and that this trait is most likely quantitatively

inherited in creeping bentgrass. Heterosis and combining ability results indicate, however, that dominant gene effects may be important for resistance/susceptibility in particular creeping bentgrass plants. Our data support selecting plants with high general combining ability (i.e., L93-10) for improving dollar spot resistance in subsequent generations. This is the first report documenting the importance of parent selection in improving dollar spot resistance in creeping bentgrass.

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