Gray Leaf Spot Disease of Maize: Rating Methodology and Inbred Line Evaluation

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ABSTRACT

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Development of gray leaf spot (GLS) of maize (*Zea mays*), caused by *Cercospora zeae-maydis*, was evaluated in seven inbreds in 1990 and 19 inbreds in 1991 in field plots with naturally infested corn debris. In 1990, nine ratings over 35 days were made beginning 9 August. In 1991, 10 ratings over 43 days were made beginning 17 July. Individual plants were assessed by two methods: 1) a disease index (DI) score was assigned on a 1–5 scale (1 = no lesions, 5 = all leaves dead); and 2) disease severity (DS) was expressed as diseased leaf area divided by the total area multiplied by 100, assessed on the ear −1 leaf and all leaves above it. The mean of ratings over time and the area under the disease progress curve (AUDPC) rankings for seven inbreds were similar both years. Rankings for resistance among inbreds based on all 1991 individual ratings, as well as various combinations of ratings for both DI and DS methods, were highly correlated ($P \leq 0.001$). Such close associations between the two rating methods indicate that the DI method could replace the DS procedure, which was perceived as the more precise rating method. Of the seven inbreds tested both years, each rating procedure identified T222, NC290, and Pa875 as the most resistant; NC250 as resistant; H93 as intermediate; NC286 as susceptible; and B87 as the most susceptible to GLS. Furthermore, the use of fewer and more widely spaced rating times, especially toward the end of the disease epidemics, could distinguish inbred GLS response nearly as well as the use of many evenly spaced ratings.

Additional keywords: percentage leaf area affected

Gray leaf spot (GLS) is a foliar disease of corn (*Zea mays* L.) caused by *Cercospora zeae-maydis* Tehon & E.Y. Daniels. Since the early 1970s, the prevalence and severity of GLS have increased markedly in the eastern United States from South Carolina to New York and west to Tennessee (7,11,12,16). Significant outbreaks of GLS also have been reported in several Corn Belt states (15). The increase in prevalence of GLS has been linked with conservation tillage practices (3,7,9,16,18,22), continuous corn production, and extended periods of high relative humidity and dew periods (3,11). Yield losses are estimated to be between 10 and 25% annually in areas where GLS is endemic, but they can be much higher (4).

Many types of GLS assessments have been used to ascertain disease reactions, estimate yield losses (2,7,17,22), evaluate resistance inheritance (4,5,8,14,23,24), and assess the influence of environment and plant maturity on disease development (18). However, methods, timing, and frequency of measurements vary widely. General rating scales incorporate both disease incidence and severity, whereas severity-rating procedures measure the percentage of leaf area damaged or the total number of lesions per sampling unit (5,8,13,18). However, lesion counts are sometimes difficult to make when leaves are severely blighted because of lesion coalescence (13).

Four strategies for frequency and timing of GLS ratings have been used: 1) rating two or three times at widely spaced (up to 1 mo) intervals throughout the period of disease development (2,4,7,8,17,22); 2) a single rating near the time of plant physiological maturity (8,14,24); 3) rating several times beginning at mid- to late-disease development (5,13,23); and 4) rating throughout disease development at frequent (weekly) intervals (18). Thompson et al (23) noted that the best time to rate GLS for assessing resistance was just before the onset of leaf senescence. Elwinger et al (5) concluded that a single late-season evaluation should result in a ranking of lines similar to that based on the average amount of disease measured over a period of time.

Researchers making multiple assessments with rating indices have either averaged ratings over time to get a mean index (4,22,23) or analyzed their data for each separate observation time (2,4,22). Conversely, most studies employing severity indices have quantified disease response over the season as the area under the disease progress curve (AUDPC) (5,8,13).

Resistance to GLS is inherited quantitatively by genes that act primarily in an additive manner (2,4,5,8,14,23,24). DNA restriction fragment length polymorphism (RFLP) analysis provides a potentially powerful tool for identifying the quantitative trait loci responsible for controlling resistance to GLS. However, to determine the type and magnitude of each gene's contribution to the overall phenotype in segregating populations, methods for accurate and reproducible estimation of disease reaction are essential. The objectives of this study were the following: 1) to compare two methods of rating GLS, a disease index (DI) and a disease severity (DS) procedure; 2) to determine optimal timing and frequency for rating; and 3) to evaluate the development of GLS on inbreds ranging from highly resistant to highly susceptible.

MATERIALS AND METHODS

The 1990 experiment. Seven public corn inbreds (B87, H93, NC250, NC286, NC290, Pa875, and T222) were evaluated at Whitethorne Plantation (Hayter silt loam, pH 6.3), Montgomery County, Virginia, with a two-replicate randomized complete block design. Single-row plots 3.4 m long and spaced 0.76 m apart were seeded on 5 May with a cone-planter equipped for no-tillage seeding. A known susceptible check hybrid, Pioneer Brand 3320, was planted every seven rows to gauge disease uniformity within the plot area. The field had been planted to no-tillage corn during the two previous years and had a history of severe GLS. Rows were hand-thinned to 12 plants per row 2 wk after emergence. Plots were rated for GLS nine times over 35 days beginning on 9 August, when disease symptoms were first observed. Individual ratings for all plants within each plot were made by the same individual by two methods: 1) a DI estimate on a scale of 1–5 in increments of 0.25, where 1 = no symptoms; 2 = moderate lesion development below the leaf subtending the ear; 3 = heavy lesion development on and below the leaf subtending the ear with a few lesions above it; 4 = severe lesion development on all but the uppermost leaves, which may have a few lesions; and 5 = all leaves

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dead; and 2) DS expressed as diseased leaf area divided by total leaf area multiplied by 100, assessed on the ear −1 leaf and all leaves above it. DS estimates were restricted to these leaves to evaluate only the portion of the canopy directly involved in photosynthate production for the developing ear (1), and to avoid rating the lowest leaves, which senesce early in the season when colonized by *C. zeae-maydis*.

The 1991 experiment. Nineteen inbreds including 16 public lines (A632, B73, B87, H93, H95, Mo17, NC250, NC286, NC290, Oh43, Pa91, Pa875, T222, Va22, Va59, and Wf9) and three Virginia experimentals (Exp1, Exp2, and Exp3) developed by H. L. Warren, Department of Plant Pathology, Physiology and Weed Science, Virginia Polytechnic Institute and State University, were evaluated. The additional lines were included to expand the range of disease reactions to GLS from highly resistant to highly susceptible. The experiment was conducted as in 1990 with the following modifications: 1) replications were increased to three; 2) seeds were planted on 16 May in the same field; 3) additional rows of the same GLS-susceptible hybrid (Pioneer Brand 3320) were planted adjacent to the sides of each range; and 4) ratings were made about every 5 days beginning 17 July, when disease symptoms were first observed, through 28 August, for a total of 10 ratings. Individual plant DI and single leaf DS estimates were made as in 1990. However, a correction factor was calculated for each leaf position by inbred to compensate for differences in leaf area among leaf positions within and among inbreds. A single, arbitrarily selected, mature plant of each inbred was harvested from nearby plots. These plants had been sprayed with the fungicide benomyl (Benlate, 50% WP, 280 g a.i./ha) at weekly intervals during the growing season beginning at anthesis. Leaves were removed from each plant and traced on paper. The "paper leaves" were weighed, and their areas were calculated based on weight per unit area of the paper. Percent contribution of

Table 1. Gray leaf spot disease index (DI) and disease severity (DS) overall means and area under the disease progress curves (AUDPC) for seven maize inbreds rated in 1990 and 1991

	Disease index				Disease severity			
	MDI[v]		ADI[w]		MDS[x]		ADS[y]	
Inbred	1990	1991	1990	1991	1990	1991	1990	1991
NC290	1.07 a[z]	1.28 a	1.76 a	12.01 a	0.00 a	0.00 a	0.00 a	0.00 a
Pa875	1.12 a	1.36 b	3.16 ab	15.60 b	0.00 a	0.00 a	0.00 a	0.00 a
T222	1.28 b	1.34 ab	7.13 b	14.89 ab	0.00 a	0.02 a	0.00 a	0.46 a
NC250	2.86 d	1.64 c	23.73 c	27.22 c	4.44 b	0.53 a	81.30 a	12.39 a
H93	2.29 c	2.46 d	39.77 d	64.47 d	7.00 d	14.39 b	169.67 b	389.14 b
NC286	3.02 e	2.88 e	62.56 e	81.92 e	21.88 c	29.96 c	600.89 c	790.55 c
B87	3.40 f	3.08 f	74.57 f	90.81 f	36.00 d	38.92 d	977.97 d	1,027.22 d

[v] Mean of all DI ratings (9 in 1990, 10 in 1991).
[w] AUDPC over all DI ratings.
[x] Mean of all DS ratings (9 in 1990, 6 in 1991).
[y] AUDPC over all DS ratings.
[z] Means within a column having the same letter do not differ significantly at the 0.05 level of probability according to the Tukey-Kramer test for unequal cell sizes.

Table 2. Gray leaf spot disease index (DI) and disease severity (DS) rating means and area under the disease progress curves (AUDPC) for 19 maize inbreds in 1991

	Disease index						Disease severity	
Inbred	MDI[r]	ADI[s]	$MDI_{5\text{-}10}$[t]	$ADI_{5\text{-}10}$[u]	MDI_3[v]	$ADI_{1\text{-}6}$[w]	$MDS_{5\text{-}10}$[x]	$ADS_{5\text{-}10}$[y]
NC290	1.28 a[z]	12.0 a	1.43 a	9.1 a	1.28 a	2.9 a	0.0 a	0.0 a
T222	1.34 a	14.9 a	1.48 a	10.0 a	1.32 ab	4.9 ab	0.0 a	0.0 a
Pa875	1.36 a	15.6 a	1.44 a	8.6 a	1.42 b	7.0 b	0.0 a	0.5 a
Va59	1.55 b	23.5 b	1.79 b	15.9 b	1.61 c	7.6 c	1.3 a	27.1 a
NC250	1.64 b	27.2 b	1.85 b	18.1 b	1.66 c	9.2 c	0.5 a	12.4 a
Mo17	2.03 c	45.0 c	2.55 c	26.5 c	2.31 d	18.5 d	9.5 b	260.0 b
H95	2.20 de	53.2 d	2.92 de	34.3 d	2.40 de	18.9 d	20.3 d	549.0 d
Exp 2	2.29 de	57.1 d	2.91 de	35.1 d	2.50 ef	22.0 e	14.2 c	385.1 c
Exp 1	2.33 ef	58.3 de	2.81 d	33.1 d	2.55 fg	25.0 f	14.0 c	372.8 c
Exp 3	2.44 fg	63.2 e	2.95 e	36.5 d	2.62 gh	26.8 fg	13.3 c	351.6 c
H93	2.47 g	64.5 e	2.93 de	36.3 d	2.65 hi	28.2 g	14.4 c	389.1 c
Va22	2.60 h	70.3 f	3.22 f	43.2 e	2.77 j	27.1 fg	22.9 d	617.9 d
Oh43	2.66 hi	73.6 fg	3.41 g	47.9 f	2.74 ij	25.7 f	30.6 g	818.5 gh
A632	2.70 hi	75.3 g	3.39 g	48.4 f	2.77 j	27.0 fg	26.2 e	708.4 e
B73	2.74 i	76.0 gh	3.33 fg	47.2 f	2.81 j	28.8 gh	26.9 ef	723.9 ef
Pa91	2.76 i	77.2 gh	3.44 g	48.9 f	2.83 j	28.4 gh	28.3 ef	767.2 efg
Wf9	2.77 ij	78.4 gh	3.38 g	47.9 f	2.84 j	30.5 h	31.9 g	865.1 h
NC286	2.88 j	81.7 h	3.58 h	52.9 g	2.84 j	28.8 gh	30.0 fg	790.6 fgh
B87	3.08 k	90.8 i	3.80 i	58.1 h	3.08 k	32.8 i	38.9 h	1,027.2 i

[r] Mean of all 10 DI ratings.
[s] AUDPC of all 10 DI ratings.
[t] Mean of the last six DI ratings.
[u] AUDPC over the last six DI ratings.
[v] Mean of DI ratings 1, 5, and 10.
[w] AUDPC over the first six DI ratings.
[x] Mean of the last six DS ratings.
[y] AUDPC over the last six DS ratings.
[z] Means within a column having the same letter do not differ significantly at the 0.05 level of probability according to the Tukey-Kramer test for unequal cell sizes.

each leaf position for each inbred was then determined and used to correct individual leaf DS values, which were summed to give plant DS estimates. The 1990 DS data were similarly corrected with 1991 leaf areas to make comparisons over years. While a few lesions were observed on the leaf below the ear for three inbreds at all rating periods in 1990, no lesions were seen this high in the canopy for any inbred earlier than 20 days after ratings began in 1991. For this reason, 1991 DS ratings are given only for the last six rating dates.

Analytical methods. Analysis of the 1990 experimental data was restricted to comparisons among lines that were common to both years, because inbreds used in the 1990 experiment did not include the full range of GLS disease expression. DI and DS rating means were averages over all nine rating dates. AUDPCs were calculated by trapezoidal integration (20). AUDPC values integrated over all rating dates were created for both DI and DS ratings and were designated ADI and ADS, respectively.

In 1991, individual plant DI scores for the 10 rating dates were averaged (designated as DI_1–DI_{10}) for each inbred. DS scores, which were available for only the last six rating dates (see above), were also averaged (DS_6–DS_{10}) for each inbred. A series of combined-date rating means (see footnotes for Tables 1–3) using DI and DS scores were generated to compare frequency and timing of disease rating. DI inbred means were averaged over all 10 rating dates (MDI), the last six rating dates ($MDI_{5\text{-}10}$), and three widely spaced rating dates ($MDI_3 = [DI_1 + DI_5 + DI_{10}]/3$); and DS inbred means were averaged over the last six rating dates ($MDS_{5\text{-}10}$). AUDPC values were calculated over all 10 DI rating dates (ADI), the first six rating dates ($ADI_{1\text{-}6}$), and the last six rating dates ($ADI_{5\text{-}10}$). An AUDPC parameter was determined for DS scores over the last six rating dates ($ADS_{5\text{-}10}$).

Analyses of variance of 1990, 1991, and the combined data were performed by the GLM, ANOVA, and repeated measures ANOVA procedures (19). The Tukey-Kramer procedure (10) for unequal sample sizes was employed for mean separation tests among inbreds. Spearman correlation coefficients (21) were calculated among 1991 ranked inbred means for individual and combined rating dates.

RESULTS

Highly significant ($P \leq 0.0001$) differences among inbreds' reactions to GLS expressed as means or AUDPCs calculated over all rating dates were observed in 1990, 1991, and the combined years, for both the DI and DS rating methods. A much smaller but highly significant year × inbred interaction was shown for the combined data.

Means and AUDPCs over all rating periods for both DI and DS procedures for the seven inbreds evaluated both years (Table 1) were ranked similarly by the Tukey-Kramer test in 1990 and 1991. The only change in ranking order between years was observed as small differences among means and AUDPCs of the two most resistant inbreds, Pa875 and T222.

Of the seven inbreds rated in both 1990 and 1991, five (B87, H93, NC250, NC286, and Pa875) representative of the range of observed responses to GLS with the DI and DS rating methods are shown in Fig. 1. The remaining two inbreds, T222 and NC290, were both highly resistant to GLS, with curves nearly identical to those for Pa875. Yearly curves show rating intervals following observation of initial symptoms. Individual year DI and DS disease progress curves were parallel or overlapped for each inbred except NC250, which showed a sharper rise in GLS levels late in the season in 1990 than in 1991 (Fig. 1). Susceptible inbreds B87 and NC286 developed GLS faster in 1990 than 1991 (Fig. 1), while rates of disease development on intermediate H93 and resistant Pa875 were similar in both years, as indicated by both DI and DS rating procedures (Fig. 1).

Overall, 1991 DI and DS values for

Table 3. Spearman rank correlations among 1991 inbred means for disease index (DI) and disease severity (DS) values

	MDI[p]	ADI[q]	$MDI_{5\text{-}10}$[r]	$ADI_{5\text{-}10}$[s]	MDI_3[t]	$ADI_{1\text{-}6}$[u]	$MDS_{5\text{-}10}$[v]	$ADS_{5\text{-}10}$[w]
MDI								
ADI	1.000[x]							
$MDI_{5\text{-}10}$	0.967	0.967						
$ADI_{5\text{-}10}$	0.972	0.972	0.990					
MDI_3	0.995	0.995	0.953	0.962				
$ADI_{1\text{-}6}$	0.970	0.970	0.899	0.911	0.976			
$MDS_{5\text{-}10}$	0.947	0.945	0.957	0.945	0.941	0.889		
$ADS_{5\text{-}10}$	0.952	0.952	0.949	0.933	0.943	0.901	0.992	
DI_1[y]	0.910	0.910	0.801	0.827	0.929	0.936	0.785	0.794
DI_2	0.896	0.896	0.784	0.796	0.899	0.938	0.751	0.771
DI_3	0.900	0.900	0.800	0.823	0.901	0.949	0.762	0.773
DI_4	0.932	0.932	0.853	0.866	0.929	0.951	0.811	0.830
DI_5	0.943	0.943	0.935	0.929	0.930	0.909	0.870	0.884
DI_6	0.914	0.914	0.967	0.946	0.902	0.856	0.889	0.871
DI_7	0.953	0.953	0.969	0.958	0.938	0.897	0.929	0.938
DI_8	0.956	0.956	0.960	0.972	0.949	0.888	0.964	0.960
DI_9	0.953	0.953	0.954	0.962	0.947	0.890	0.977	0.969
DI_{10}	0.906	0.906	0.927	0.919	0.899	0.828	0.981	0.973
DS_5[z]	0.942	0.942	0.960	0.947	0.921	0.880	0.911	0.901
DS_6	0.938	0.938	0.985	0.970	0.922	0.864	0.943	0.924
DS_7	0.974	0.974	0.960	0.957	0.965	0.944	0.942	0.951
DS_8	0.962	0.962	0.960	0.955	0.956	0.909	0.982	0.985
DS_9	0.941	0.941	0.946	0.932	0.932	0.887	0.981	0.988
DS_{10}	0.924	0.924	0.929	0.919	0.912	0.856	0.977	0.982

[p] Mean of all 10 DI ratings.
[q] AUDPC for all 10 DI ratings.
[r] Mean of last six DI ratings.
[s] AUDPC for last six DI ratings.
[t] Mean of DI ratings 1, 5, and 10.
[u] AUDPC for first six DI ratings.
[v] Mean of last six DS ratings.
[w] AUDPC for last six DS ratings.
[x] All correlations are significant at the $P \leq 0.0001$ level of probability.
[y] DI_1–DI_{10} = individual DI ratings for dates 1–10.
[z] DS_5–DS_{10} = individual DS ratings for dates 5–10.

various combinations of rating dates (Table 2) ranked GLS disease levels of the lines similarly. DI scores, MDI, ADI, MDI_{5-10}, and ADI_{5-10} (means and AUDPCs of DI over all 10 or the last six ratings) all ranked NC290, T222, and Pa875 somewhat more resistant than Va59 and NC250. MDI_3 and ADI_{1-6} distinguished an additional gradation of resistance between T222 and Pa875, while all five resistant lines were grouped together by the DS values, MDS_{5-10} and ADS_{5-10}. All combined-date rating means and AUDPCs except ADI_{1-6} distinguished Mo17 as moderately resistant; i.e., having significantly less resistance than NC250 or Va59 but more than other inbreds. All combined-date rating scores ranked B87 highly susceptible, and all but ADI_{1-6} (AUDPC over the first six DI ratings) ranked Oh43, A632, B73, Pa91, Wf9, and NC286 as susceptible, although different significant groupings of means were distinguished. Inbreds H95, Expl, Exp2, Exp3, H93, and Va22 fell into a definite intermediate category according to ADI_{5-10} (AUDPC over the last six DI ratings) and DS values, while several different significant groupings of means were found for the other DI values. Va22 was grouped with the susceptible class by DI parameters but with the intermediate class by DS scores. ADI_{1-6} did not clearly distinguish these intermediate and susceptible classes.

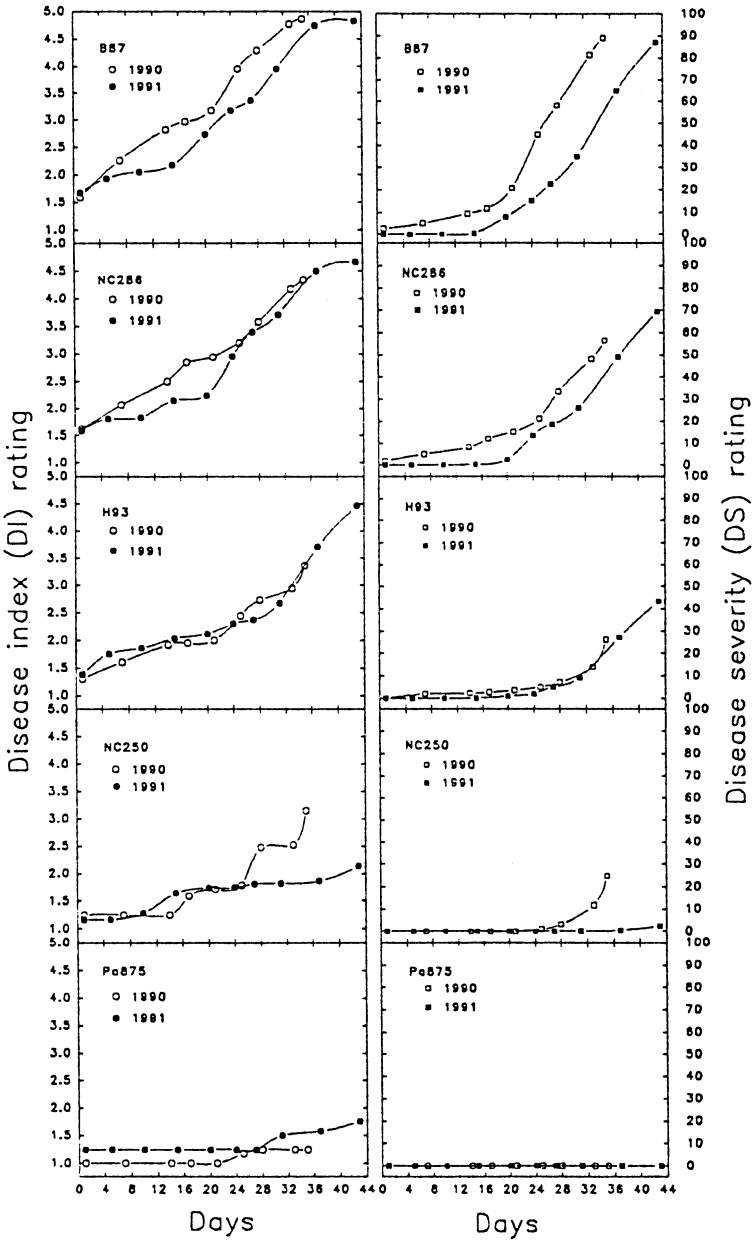

Fig. 1. Gray leaf spot development on five maize inbreds (B87, NC286, H93, NC250, and Pa875) representative of the observed reactions, by disease index (DI, left column) and disease severity (DS, right column) assessment. Individual plants were rated nine times over 35 days in 1990 and 10 times over 43 days in 1991, beginning when symptoms were first observed. DI estimates were made on a scale of 1–5 in increments of 0.25, where 1 = no symptoms and 5 = all leaves dead. DS is expressed as diseased leaf area divided by total area of the ear −1 leaf and all the leaves above it × 100.

Individual rating date and various combined-date disease scores for both DI and DS procedures were compared by mean separation tests (Table 2) and Spearman rank correlation analyses (Table 3) to evaluate their suitability for assessing inbred response to GLS. Highly significant ($P \leq 0.0001$) Spearman correlation coefficients were observed among all ranked 1991 DI and DS means and AUDPCs (Table 3). The mean (MDI) and AUDPC (ADI) over all DI rating dates ranked inbreds identically ($R = 1.000$). Correlation coefficients among other ranked DI and DS means and AUDPCs ranged from 0.895 (ADI_{1-6} vs. MDI_{5-10}) to 0.995 (MDI_3 vs. MDI and ADI) and reflect close similarities in ranking of and significance relationships among means of these parameters (Table 2). Disease-rating values encompassing the same time span, such as the DI mean (MDI_{5-10}) and AUDPC (ADI_{5-10}) over the last six ratings, separated means similarly (Table 2); and their rankings were nearly perfectly correlated (Table 3). Mean separations involving the last six ratings (MDI_{5-10} and ADI_{5-10}) more nearly resembled those of all 10 ratings (MDI and ADI) than did the mean separations of the first six ratings (ADI_{1-6}) (Table 3).

Among single-date DI ratings, DI_7, DI_8, and DI_9 gave the highest rank correlations with all combined-date rating values except ADI_{1-6} (Table 3). Among single-date DS ratings, DS_7 and DS_8 gave the highest rank correlations with MDI and ADI, while DS_9 gave the highest rank correlation with ADS_{5-10} and MDS_{5-10} (Table 3). Mean separations of inbred response to GLS for DI_7–DI_9 were similar to those for MDI and ADI, while those for DS_9 were similar to mean separations for MDS_{5-10} (*data not shown*).

DISCUSSION

We compared DI and DS assessment methods for evaluating germ plasm for resistance to GLS. DI is a whole-plant visual estimate of disease intensity, while DS is the mean percentage of individual leaf areas affected. Of the two methods, DS is more time-consuming, and restriction of the number of leaves used in estimates to those of middle and upper canopy results in truncation of early rating values. Moreover, this truncation may make it difficult to distinguish small differences in highly resistant germ plasm for which lesions never progress to or beyond leaves below the ear leaf.

The DI method has frequently been used for disease evaluation because of its ease, speed, and economy. However, it is perceived to be imprecise because of vagueness in describing each rating class. We attempted to reduce this imprecision and identify small differences in

disease reaction by dividing each rating category into increments of 0.25. The DI method is generally less time-consuming once the rater is familiar with the scale.

In our observation, the close association between DI and DS rating methods increases the validity of DI scores. When using RFLPs to establish associations with resistance genes, it is desirable to use a method that can evaluate disease development under heavy pressure and detect small differences in host response. This application may warrant the use of the DS rating method; however, some modification of the DI method, as in the present study, can provide similar results with greater ease and economy of time. Because of its ease, the DI is the method of choice for general resistance screening of germ plasm.

Several aspects of the frequency and timing of GLS ratings are apparent from this study. First, evenly spaced ratings made near the end of the disease epidemic give a more accurate ranking of inbred reaction than do evenly spaced ratings near the beginning of the epidemic. Second, the use of fewer, widely spaced rating times could distinguish inbred reaction nearly as well as many evenly spaced ratings. Third, a single, properly timed rating could distinguish resistance levels among inbreds nearly as well as the mean or AUDPC over many ratings made throughout the epidemic. The difficulty lies in determining the optimum time to make a single rating. Disease rating in the 1991 experiment was discontinued after DI ratings of 5 (all leaves dead) were first given to the most susceptible inbred, B87. Ranked means of D10 (all 10 rating periods), the last individual DI rating, were less highly correlated with those of MDI and ADI than DI_9 (0.91 vs. 0.95). The mean correlation of D10 with $ADS_{5\text{-}10}$ dropped even more sharply compared to that of DI_9 and $ADS_{5\text{-}10}$ (0.84 vs. 0.93). This indicated a leveling off of disease reaction at or near senescence, where two lines that were rated differently early in the disease cycle rated similarly near their deaths. Because under our conditions GLS progressively kills all but the most resistant lines, ratings taken too late in the season would be of little value in separating germ plasm response to GLS.

This study enables direct comparisons for a large number of inbreds exhibiting a range of reactions, in an environment highly favorable to GLS, by using multiple assessments of two rating methods. Results from genotypes previously evaluated by others are in general agreement with the present findings. Pa875 (4–6,8,24), T222 (6,10,24), NC250 (4,6,23), and Va59 (5,23) have all been reported as resistant to GLS. B73 (4,6,23,24), Pa91, and A632 (4) were rated susceptible. Ulrich et al (24) grouped NC250 and Va59 in an intermediate class based on 3 yr of data for six inbreds. In our evaluation of 19 inbreds, both Va59 and NC250 were resistant, although significantly less so than NC290, T222, and Pa875, according to all DI values (Table 2). Elwinger et al (5) found that, of six inbreds evaluated, Va59 ranked second to Pa875 in resistance. H93 was rated intermediate in reaction to GLS in two studies (5,8) in addition to this report (Tables 1 and 2). Donahue et al (4) categorized H93 as susceptible based on positive general combining ability effects without an inbred evaluation. Mo17 was on the resistant side of intermediate, and was classified as intermediate by Donahue et al (4) and Goodman and Bubeck (6), but as susceptible by Ulrich et al (24). The reaction of B87 to GLS has not been previously reported. The highly susceptible reaction of this inbred, coupled with the absence of potentially confounding Helminthosporium leaf spot caused by *Bipolaris zeicola* (G.L. Stout) Shoemaker, makes B87 a good susceptible standard control.

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