Evaluation and Heritability of Resistance to Sugarcane Red Rot

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ABSTRACT

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Heritability of resistance to red rot, caused by *Colletotrichum falcatum*, was studied with progeny from 40 crosses among 24 parental clones of sugarcane. Resistance was assessed by comparing disease development in detached, inoculated stalks. Disease severity was assessed as the number of nodes beyond which fungal infection symptoms were observed, the number of nodes rotted, the extent of internode rotting, and a rot index (RI) combining the number of nodes passed and internode rot severity. Significant differences in susceptibility were detected, although high levels of resistance were rare in the breeding and selection populations of the Louisiana sugarcane cultivar development program. Narrow-sense heritability estimates determined by mid–parent-offspring regression for the different disease traits ranged from 0.19 ± 0.04 to 0.31 ± 0.05. Potential genetic gain by selection for resistance, using a 10% selection intensity, ranged from 14 to 37% of the mean. The RI provided the highest heritability estimate and the most potential genetic gain from selection. We estimated low broad-sense heritabilities among years for the disease traits in the parent population. The range was from 0 to 0.42 ± 1.07 on an entry basis. The results indicated that the population level of red rot resistance can be increased by careful choice of parent clones and cross-based selection. Genotype by year interaction, however, greatly affects evaluation, hence single-year evaluations for red rot resistance are not reliable. The scarcity of resistance in the current breeding population indicates a need to identify new sources of resistance.

Red rot, caused by *Colletotrichum falcatum* Went, occurs in most regions where sugarcane, interspecific hybrids of *Saccharum*, is grown. The disease can affect stalks of growing plants and stalks or stalk sections used to propagate the crop (1,7,10,15). In Louisiana, red rot is primarily a disease of planted cane, and serious losses of buds and subsequent stand reductions are possible (1,2, 10). Whole stalks are planted during August and September. Bud germination and shoot growth is initiated during the fall, but unfavorable growth conditions during winter result in increased opportunities for red rot development. Severe losses due to red rot do not occur every year, but the disease also causes indirect losses to the sugarcane industry because high planting rates are used to insure against stand failure.

Infection of the leaf midrib and latent infections around nodes provide sources of inoculum for stalk infections (3,7). After the fungus invades the tissues of the stalk, the mycelium may spread from cell to cell. More rapid spread can occur through the vascular bundles. Infected internode tissues develop a rot with a characteristic red color that often contains interspersed areas with normal color known as "white spots" (1,10,15). The use of resistant cultivars is the most effective method for disease control (8, 15,16).

In recent years, sugarcane clones in the Louisiana cultivar selection program have not been screened for susceptibility to red rot in stalk inoculation tests. Plant breeders have assumed that highly susceptible genotypes will be eliminated during the selection process due to poor yields and that parental clones then possess some level of resistance. However, the actual frequency and levels of red rot resistance in clones in the breeding and selection populations are unknown.

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The results of previous small-scale studies (1,4,5,8) comparing the frequency of resistant progeny in crosses between resistant and susceptible parents were contradictory. Information about heritability of resistance to red rot and potential for genetic gain through selection will help determine the most appropriate breeding strategies to develop resistant cultivars. Therefore, the objectives of this study were (i) to evaluate the existence and variability of red rot resistance in the current breeding and selection populations; (ii) to estimate both narrow- and broad-sense heritabilities of resistance to red rot; and (iii) to predict the potential genetic advance (GA) from selection for red rot resistance. A portion of the results was presented previously (17).

MATERIALS AND METHODS

Studies were conducted during two 6-week periods in 1992 and 1993. The starting dates of the tests were 25 September and 13 November 1992 and 22 October and 15 November 1993. Ten unselected progeny from each of forty crosses and twenty-four randomly chosen but adapted sugarcane parental clones (Table 1) from the sugarcane breeding program of the Louisiana Agricultural Experiment Station were evaluated for red rot resistance. Five progeny from every cross were tested on one of the two dates within a year. Different progeny clones from a cross were used on different test dates. The same 10 progeny from each cross were tested in both years, but individual identity was not maintained. Mature stalks with at least 10 nodes and no stalk-borer injury were cut, and the leaf sheaths were stripped. The stalks were surface-disinfected for 30 min in a solution of approximately 0.26% NaOCl. Three stalks from every parent and its progeny were inoculated for disease evaluation in all experiments.

Inoculum preparation. Fresh leaves with small, red *C. falcatum* lesions on the midrib were collected from various sugarcane-growing areas in Louisiana. Small pieces of leaf tissue were cut from the margin of a lesion and soaked in 0.52% NaOCl solution

for 3 min. The surface-disinfected tissues were placed on Difco potato dextrose agar (PDA) or acid-PDA plates and incubated at room temperature for 48 h. A hyphal tip within a small piece of agar was transferred to a PDA plate to obtain a pure culture of *C. falcatum*. Several isolates from different sources were transferred to oatmeal agar medium (40 g of Quick Quaker oats, 20 g of Difco Bacto agar, 1 liter of water, and 100 ppm of streptomycin) and incubated at 27°C for 14 days. Inoculum was prepared by mixing six isolates. Distilled water (6 ml) was added to each plate, the surface of the agar colony was scraped, and the suspension was decanted through cheesecloth. The concentrations of conidia in the suspensions from the different isolates were determined with a compound microscope, using a hemacytometer. Conidial concentrations averaged 2.5×10^6 spores per ml in 1992 and 2.7×10^6 spores per ml in 1993.

Inoculation method. A 3.2-mm-diameter hole was drilled into, but not through, the center internode of each stalk, and 100 µl of inoculum was introduced (1,2). The inoculated stalks were wrapped in a perforated sheet of polyethylene with wet paper towels inside and incubated at 24 ± 3°C for 6 weeks.

Resistance assessment. After the incubation period, the inoculated stalks were split longitudinally, and different disease parameters were assessed, including (i) the number of nodes passed (NP) by internode rot symptoms in each direction from the inoculation point; (ii) the number of nodes rotted (NR); and (iii) internode rot severity (IRS) in the inoculated internode and in the four internodes on each side of the inoculation point. IRS was determined first by rating the portion of tissue exhibiting red discoloration in each internode on the following scale: 1 = 10% or less (average 5%); 2 = 11 to 25% (average 18%); 3 = 26 to 50% (average 38%); 4 = 51 to 75% (average 63%); 5 = 76 to 90% (average 83%); and 6 = >90% (average 96%). IRS was converted to average percent discolored area per internode by calculating the sum of individual internode discoloration percents and dividing by nine internodes. An aggregate disease trait, rot severity index (RI), that combined the traits describing the extent of internode rotting and disease spread was calculated as RI = NP × IRS. A resistant rating was assigned to a clone if NP was <2 and IRS was <18%; a susceptible rating was assigned if NP ranged from 2 to 3 and IRS ranged from 18 to 38%, and a highly susceptible rating was assigned if NP was >3 and IRS was >38%.

Narrow-sense heritability estimation. The narrow-sense heritability (h^2) of red rot resistance was estimated on a family-mean basis by mid–parent-offspring regression for each year and the 2 years combined. The h^2 was equated to the linear regression coefficient (11,13). Additive genetic variance ($\hat{\sigma}_A^2$) also was expressed as an additive genetic coefficient of variation (AGCV), where AGCV = $100\hat{\sigma}_A$/midparent mean. The phenotypic variance of the midparent population ($\hat{\sigma}_{mp}^2$) also was expressed as a midparent coefficient of variation (MPCV), where MPCV = $100\hat{\sigma}_{mp}$/midparent mean. GA from selection, expressed as a percentage of the midparent mean, was estimated as GA = $100ih_{mp}^2$/midparent mean with an assumed 10% selection intensity among a population of infinite size (i = 1.76) (6).

Broad-sense heritability for the parent population. The broad-sense heritability of red rot resistance for parental clones was estimated by intraclass correlations of variance components derived from two models. The full model included genotype, date, and year effects and all appropriate interactions. The reduced model did not include year or year interaction effects. Data were subjected to variance and covariance analysis, assuming all genetic components were random. Variance components were calculated by equating appropriate mean squares to their expectation and solving for the components (11,13).

Broad-sense heritability estimates with variance components from the reduced model were calculated in two ways: (i) on a single-stalk basis (H_{PR1}), with one experiment date (d = 1) and one stalk per plant (n = 1) tested, and (ii) on an entry-mean basis (H_{PR2}), with two experiment dates (d = 2) and three stalks per plant (n = 3) tested. Within-year heritability was estimated as $H_{PRi} = \hat{\sigma}_g^2/(\hat{\sigma}_g^2 +$

TABLE 1. Evaluation of red rot resistance in 24 sugarcane parental clones in 1992 and 1993

Parent	Internode rot severity (%)[a]			Nodes passed[b]			Nodes rotted[c]			Rot index (% number)[d]		
	1992	1993	Mean	1992	1993	Mean	1992	1993	Mean	1992	1993	Mean
CP62-258	17	9	13	1.6	1.2	1.4	1.2	0.7	0.9	31	11	20
CP65-357	35	22	29	4.0	2.8	3.7	4.0	2.7	3.3	140	64	99
CP70-321	32	26	29	3.6	3.0	3.3	3.2	2.8	3.0	115	81	97
CP72-370	22	14	18	3.2	2.3	2.7	3.2	2.3	2.7	70	32	49
CP74-383	22	21	21	2.8	3.5	3.2	2.6	3.5	3.1	67	72	70
CP75-1082	19	12	16	4.2	2.0	3.0	4.0	2.0	2.9	84	23	51
CP76-331	19	17	18	3.2	2.5	2.8	3.2	2.5	2.8	60	47	53
CP77-310	30	23	27	4.0	2.3	3.1	3.8	2.0	2.8	134	64	96
CP79-318	20	17	19	2.2	2.2	2.2	2.2	1.7	1.9	46	38	41
CP80-313	28	48	38	3.0	4.3	3.9	3.0	4.3	3.8	83	227	179
CP80-323	19	11	15	2.8	1.5	2.1	2.8	0.5	1.5	58	17	36
CP83-644	14	23	19	1.8	2.7	2.3	1.6	1.7	1.6	25	66	47
LCP81-10	27	13	19	3.2	2.0	2.5	3.2	1.7	2.4	91	23	56
LCP81-30	31	16	23	3.6	1.8	2.6	3.4	1.7	2.5	113	32	69
LCP82-89	26	19	21	2.8	2.0	2.4	2.6	2.0	2.3	70	37	52
LCP85-341	18	32	26	2.6	3.0	2.8	2.2	2.7	2.4	53	110	84
LCP85-345	26	25	25	2.8	2.5	2.6	2.6	2.5	2.5	80	65	72
LCP85-376	11	19	15	1.4	2.2	1.8	0.6	2.2	1.4	17	55	38
LCP85-384	31	22	27	3.6	2.3	2.9	2.8	2.0	2.4	117	59	85
LCP86-393	37	18	28	3.4	1.8	2.5	3.4	1.8	2.5	135	33	79
LCP86-408	33	18	26	3.0	2.3	2.6	3.0	2.3	2.6	104	41	70
LCP86-420	52	16	33	5.0	1.8	3.3	4.8	1.8	3.2	273	31	141
LCP87-17	43	18	30	4.0	2.2	3.0	3.0	2.2	2.5	176	39	101
LCP87-494	26	12	19	2.8	2.0	2.4	2.8	1.8	2.3	76	24	48
$LSD_{0.05}$	13	15	18	2.8	2.0	1.5	1.7	1.6	1.4	98	72	104

[a] The average percent discoloration in the inoculated internode and in four internodes on each side of the inoculation point.

[b] The number of nodes passed by internode rot symptoms in each direction from the inoculation point.

[c] The number of nodes in the assessed area with rot symptoms.

[d] Rot index = internode rot severity × nodes passed.

$\hat{\sigma}_{gd}^2/d + \hat{\sigma}_\varepsilon^2/nd)$, where $H_{PRi} = H_{PR1}$ and H_{PR2}. $\hat{\sigma}_g^2$, $\hat{\sigma}_{gd}^2$, and $\hat{\sigma}_\varepsilon^2$ refer to the genotype, the genotype by date, and the residual variance, respectively.

Broad-sense heritability estimates (H_{PFi}) with variance components from the full model analysis were calculated as: $H_{PFi} = \hat{\sigma}_g^2/(\hat{\sigma}_g^2 + \hat{\sigma}_{gd}^2/d + \hat{\sigma}_{gy}^2/y + \hat{\sigma}_{gdy}^2/dy + \hat{\sigma}_\varepsilon^2/dyn)$, where $H_{PFi} = H_{PF1}$ and H_{PF2}. $\hat{\sigma}_{gy}^2$ and $\hat{\sigma}_{gdy}^2$ refer to the genotype by year and by date by year interaction variances, respectively. The divisor, y, refers to the number of years. The heritabilities were calculated on an unreplicated single-stalk basis (H_{PF1}), where one year, one experiment date, and one stalk per plant ($y = d = n = 1$) were assumed, and on an entry-mean basis (H_{PF2}), where two years, two experiment dates per year, and three stalks per plant ($y = d = 2$, $n = 3$) were assumed. Heritability standard errors were calculated by Dickerson's approximation (9).

Genetic coefficients of variation (GCV) were calculated as GCV = $100\hat{\sigma}_g$/parental mean. GA_j, expressed as a percentage of the parental mean for the four estimated heritabilities ($H_j = H_{PR1}$, H_{PR2}, H_{PF1}, and H_{PF2}), was calculated as $GA_j = 100iH_j\hat{\sigma}_{mp}$/parental mean. The phenotypic standard deviation ($\hat{\sigma}_{mp}$) was equated to the square root of the denominator of the appropriate heritability (H_j). A 10% selection intensity ($i = 1.76$) was used.

Variance component estimation for family means. Two models were used for family mean analysis. The full model used included date, year, family effect, and their interactions and offspring within date, year, and family. The reduced model did not include year and year interaction effects. Analysis of variance and variance component estimation followed the same procedures as parent data analysis. GCV for family means and genotypes within families were calculated.

TABLE 2. Narrow-sense heritability (h^2) estimates and inheritance of resistance to red rot in sugarcane assessed by different disease traits in 1992 and 1993 and both years combined

	Disease trait[b]											
	Internode rot severity			Nodes passed			Nodes rotted			Rot index		
Parameter[a]	1992	1993	1992–1993	1992	1993	1992–1993	1992	1993	1992–1993	1992	1993	1992–1993
$\hat{\sigma}_A^2$	20.4[c]	24.3[c]	22.7[c]	0.32[d]	0.15[d]	0.22[d]	0.24[d]	0.16[d]	0.19[d]	9.7[e]	6.4[e]	7.7[e]
$\hat{\sigma}_{mp}^2$	126[c]	120[c]	122[c]	1.00[d]	0.48[d]	0.69[d]	0.98[d]	0.70[d]	0.81[d]	38.0[e]	17.2[e]	25.7[e]
Mean	29.9 ± 0.3[f]	21.8 ± 0.2[f]	25.1 ± 0.2[f]	3.17 ± 0.03[g]	2.41 ± 0.02[g]	2.72 ± 0.02[g]	2.94 ± 0.03[g]	2.22 ± 0.02[g]	2.51 ± 0.02[g]	98.6 ± 1.6[h]	55.1 ± 1.1[h]	72.8 ± 1.0[h]
h^2 (unitless)	0.16 ± 0.06	0.20 ± 0.05	0.19 ± 0.04	0.32 ± 0.07	0.30 ± 0.08	0.31 ± 0.05	0.25 ± 0.07	0.23 ± 0.06	0.22 ± 0.05	0.25 ± 0.06	0.37 ± 0.09	0.30 ± 0.05
AGCV (%)	15.1	22.6	19.0	17.8	15.8	17.1	16.8	18.0	17.5	31.5	46.0	38.2
MPCV (%)	37.5	50.2	44.1	31.2	28.8	30.4	33.8	37.5	35.9	62.5	75.3	69.6
GA (%)	10.7	17.8	14.4	17.8	15.3	16.9	14.7	15.2	15.1	28.0	49.5	36.9

[a] $\hat{\sigma}_A^2$ = additive genetic variance; $\hat{\sigma}_{mp}^2$ = midparent phenotypic variance; Mean = midparent mean; AGCV = additive genetic coefficient of variation; MPCV = midparent coefficient of variation; GA = genetic advance with a 10% selection intensity.

[b] Internode rot severity: the average percent discoloration in the inoculated internode and in four internodes on each side of the inoculation point. Nodes passed: the number of nodes passed by internode rot symptoms in each direction from the inoculation point. Nodes rotted: the number of nodes in the assessed area with rot symptoms. Rot index = internode rot severity × nodes passed. Combined years are the results based on analyses of combined data collected from 1992 and 1993.

[c] Percent squared.

[d] Number squared.

[e] Percent number squared × 10^2.

[f] Percent.

[g] Number.

[h] Percent number.

TABLE 3. Parental variance components, means, genetic coefficients of variation (GCV), broad-sense heritability estimates, and genetic advances from selection for red rot resistance in sugarcane based on disease traits in 1992 and 1993

	Disease trait[b]							
	Internode rot severity		Nodes passed		Nodes rotted		Rot index	
Parameter[a]	1992	1993	1992	1993	1992	1993	1992	1993
$\hat{\sigma}_g^2$	67.4** ± 702[c]	38.3* ± 548[c]	0.34 ± 0.75[d]	0.26* ± 0.48[d]	0.54** ± 0.79[d]	0.32** ± 0.56[d]	17.6* ± 44.5[e]	12.8** ± 29.3[e]
$\hat{\sigma}_{gd}^2$	12.3 ± 378[c]	37.8** ± 377[c]	0.50** ± 0.61[d]	0.15* ± 0.34[d]	0.37** ± 0.57[d]	0.36** ± 0.44[d]	14.2** ± 32.8[e]	6.8** ± 18.9[e]
$\hat{\sigma}_\varepsilon^2$	62.8 ± 29.4[c]	48.2 ± 19.7[c]	0.66 ± 0.04[d]	0.61 ± 0.03[d]	0.70 ± 0.03[d]	0.69 ± 0.02[d]	20.5 ± 1.9[e]	15.7 ± 1.2[e]
Parental mean	26.5 ± 1.1[f]	19.5 ± 1.0[f]	3.11 ± 0.11[g]	2.35 ± 0.08[g]	2.88 ± 0.12[g]	2.14 ± 0.10[g]	92.7 ± 6.7[h]	53.9 ± 4.9[h]
H_{PR1} (unitless)	0.47 ± 0.55	0.31 ± 0.49	0.23 ± 0.50	0.26 ± 0.47	0.33 ± 0.49	0.24 ± 0.41	0.34 ± 0.85	0.36 ± 0.81
H_{PR2} (unitless)	0.80 ± 0.94	0.59 ± 0.84	0.49 ± 1.07	0.60 ± 1.11	0.64 ± 0.97	0.53 ± 0.91	0.63 ± 1.58	0.68 ± 1.51
GCV (%)	31.0	31.7	18.8	21.7	25.4	26.6	45.3	66.4
GA_{PR1} (%)	37.5	31.0	15.7	19.3	25.8	22.7	46.3	70.4
GA_{PR2} (%)	48.8	42.8	23.0	29.5	35.8	33.9	63.1	96.4

[a] $\hat{\sigma}_g^2$, $\hat{\sigma}_{gd}^2$, and $\hat{\sigma}_\varepsilon^2$ refer to genotype, genotype by date, and error variances, respectively; H_{PR1} = broad-sense heritability on a single-stalk basis; H_{PR2} = broad-sense heritability on an entry-mean basis (two experiment dates and three stalks); GA = genetic advance from selection with a 10% selection intensity based on single ($PR1$) and entry mean ($PR2$) analyses.

[b] Internode rot severity: the average percent discoloration in the inoculated internode and in four internodes on each side of the inoculation point. Nodes passed: the number of nodes passed by internode rot symptoms in each direction from the inoculation point. Nodes rotted: the number of nodes in the assessed area with rot symptoms. Rot index = internode rot severity × nodes passed. Values are given ± standard error. * and ** = mean squares significant at $P = 0.05$ and 0.01, respectively.

[c] Percent squared.

[d] Number squared.

[e] Percent number squared × 10^2.

[f] Percent.

[g] Number.

[h] Percent number.

Genetic correlation. Broad-sense genetic correlation coefficients (r_g) among the disease traits were calculated for parent and family means as $r_g = \hat{\sigma}_{xy}/\hat{\sigma}_x\hat{\sigma}_y$, where $\hat{\sigma}_{xy}$ refers to the genetic covariance between traits x and y and $\hat{\sigma}_x$ and $\hat{\sigma}_y$ are the genetic standard deviations ($\hat{\sigma}_g$) for x and y.

RESULTS

Resistance evaluation in parents and families. Parental clones varied in their reaction to red rot (Table 1). The average IRS for nine internodes ranged from 11 to 52% in 1992 and from 9 to 48% in 1993. The number of nodes passed by red rot symptoms in both directions from the inoculation point varied from 1.4 to 5.0 nodes in 1992 and from 1.2 to 4.3 nodes in 1993. The number of NR differed from 0.6 to 4.8 in 1992 and from 0.5 to 4.3 in 1993. The RI ranged from 0.17 to 2.73 in 1992 and from 0.11 to 2.27 in 1993. The relative response of some clones was inconsistent across years. Only two clones, CP 62-258 and LCP 85-376, exhibited a resistant reaction to red rot in both years. Overall, red rot severity was lowest in clones CP 62-258, CP 80-323, and LCP 85-376 and highest in clones CP 65-357, CP 80-313, and LCP 86-420. Population means of the disease traits for parents and their families were similar, although the family mean values were usually slightly higher than the values of the parent population. The family mean results obtained in 1992 and 1993 were similar. However, the parent population exhibited more severe disease symptoms in 1992 than in 1993. The values for the disease traits suggest the disease reactions for most parents and progeny were in the susceptible to highly susceptible range.

Narrow-sense heritability of red rot resistance. Narrow-sense heritability estimates were determined by mid–parent-offspring regression from data collected during 1992 and 1993 and both years combined. The estimates for the combined year analysis ranged from 0.19 ± 0.04 to 0.31 ± 0.05 (Table 2). Heritability estimates were highest for resistance assessed by the number of nodes passed and the RI. However, the highest AGCV (38%) was obtained for the RI. The other disease traits had similar, low values of AGCV. The greatest seasonal variation in heritability estimates was exhibited by the RI.

Genetic gain in resistance. Assuming a 10% selection intensity, selection based on the RI resulted in the greatest expected genetic gain in resistance (37%) (Table 2). Selection for resistance based on the other disease traits resulted in lower, similar genetic gains.

Broad-sense heritability of resistance for parental clones. The GCV provides a measure of a trait's genetic variance relative to its mean. GCV varied among disease traits in different years (Table 3), from 19 to 66%. The RI gave the highest GCV in both years (45 and 66% in 1992 and 1993, respectively). GCV were stable for the same trait in different years. The genotypic and residual variances were of similar magnitude for IRS and the RI, whereas the error variance contributed more than genotypic variance to phenotypic variance for the number of NP and NR. Genotype by environment interaction commonly equaled the genotypic variance for all the traits. The broad-sense entry mean heritability estimates were moderate to high for all the disease traits in individual years, from 0.49 to 0.80. However, the estimates based on a single-stalk analysis were nearly half the estimates based on multiple-stalk entry means (ranging from 0.23 to 0.47).

With the combined-year model, at least one genotype by environment effect, genotype by year, genotype by date, or genotype by year by date interactions, significantly contributed to variance in all disease trait values (Table 4). Genotypic variance contributions to phenotypic variance were very small compared to the error variance and the cumulative genotype by environment variances. As a result, the GCV, broad-sense heritabilities, and predicted GA from selection calculated from variances of the disease traits were very low.

Variance component estimation for family means. The genetic variances among cross families ($\hat{\sigma}_f^2$) were much smaller than within family genetic variances ($\hat{\sigma}_{o(fd)}^2$ or $\hat{\sigma}_{o(fdy)}^2$) for all the disease traits (Tables 5 and 6). Genetic variance within families was stable across years (Table 5). Significant genotype by year and genotype by date interactions were detected in the progeny population (Table 6). Genotype by year interaction upwardly biased the within year estimates of genetic variance for all traits.

Correlation among test dates within and between years. Except for the number of NP in 1992 and the number of NR in 1993, all the disease traits were significantly correlated to a similar degree among dates within years in the parent population (Table 7). However, they were not correlated between years. Family means were not correlated among dates within years. Except for IRS, family means of all the other traits also were not correlated between years.

Genetic correlation of disease traits. The disease traits were highly correlated to each other (Table 8). Correlations among traits within the parent population were essentially identical to those among the family means.

DISCUSSION

Red rot resistance evaluation indicated that a range of susceptibility exists in the current Louisiana sugarcane breeding and selection populations. However, few clones showed consistent resistance to red rot. Genetic variances for resistance were generally low and genotype-environment variances were high. Therefore, the level of variation observed in the two populations was largely due to environmental effects on disease development.

TABLE 4. Parental variance components, means, genetic coefficients of variation (GCV), broad-sense heritability, and genetic advances from selection for red rot resistance in sugarcane assessed as different disease traits in 1992 and 1993 combined

	Disease trait[b]			
Parameter[a]	IRS	NP	NR	RI
$\hat{\sigma}_g^2$	1.2 ± 435[c]	0.05 ± 0.43[d]	0.18* ± 0.47[d]	–0.4 ± 25.4[e]
$\hat{\sigma}_{gd}^2$	29.4** ± 296[c]	0.04 ± 0.34[d]	–0.06 ± 0.36[d]	7.2* ± 19.1[e]
$\hat{\sigma}_{gy}^2$	49.5** ± 484[c]	0.24** ± 0.52[d]	0.15 ± 0.55[d]	14.5** ± 29.7[e]
$\hat{\sigma}_{gdy}^2$	–2.8 ± 188[c]	0.28** ± 0.40[d]	0.42** ± 0.48[d]	3.6 ± 16.8[e]
$\hat{\sigma}_\varepsilon^2$	54.4 ± 12[c]	0.63 ± 0.02[d]	0.70 ± 0.02[d]	17.7 ± 0.8[e]
Parental mean	22.6 ± 1.0[f]	2.69 ± 0.07[g]	2.47 ± 0.08[g]	71.4 ± 4.2[h]
H_{PF1} (unitless)	0.01 ± 0.37	0.04 ± 0.35	0.12 ± 0.32	0
H_{PF2} (unitless)	0.03 ± 1.11	0.15 ± 1.38	0.42 ± 1.07	0
GCV (%)	4.8	8.1	17.2	0
GA_{PF1} (%)	0.8	2.8	10.6	0
GA_{PF2} (%)	1.4	5.6	19.5	0

[a] $\hat{\sigma}_g^2$ = genotypic variance; $\hat{\sigma}_{gd}^2$ = genotype by date interaction variance; $\hat{\sigma}_{gy}^2$ = genotype by year interaction variance; $\hat{\sigma}_{gdy}^2$ = variance for genotype by date by year interaction; and $\hat{\sigma}_\varepsilon^2$ = error variances; H_{PF1} = broad-sense heritability on a single-stalk basis; H_{PF2} = broad-sense heritability on an entry-mean basis (two years, two experiment dates, and three stalks); GA = genetic advance from selection with a 10% selection intensity based on single (*PF*1) and entry mean (*PF*2) analyses.

[b] IRS = internode rot severity: the average percent discoloration in the inoculated internode and in four internodes on each side of the inoculation point. NP = nodes passed: the number of nodes passed by internode rot symptoms in each direction from the inoculation point. NR = nodes rotted: the number of nodes in the area with rot symptoms. RI = rot index: IRS × NP. Values are given ± standard error. * and ** = mean squares significant at $P = 0.05$ and = 0.01, respectively.

[c] Percent squared.

[d] Number squared.

[e] Percent number squared × 10^2.

[f] Percent.

[g] Number.

[h] Percent number.

Formerly, evaluation of resistance to red rot through stalk inoculations was a routine part of the sugarcane cultivar selection program (1,2). Clones were classified as resistant when disease symptoms did not progress beyond the inoculated internode. According to this standard, all the clones in the recent experiments would be classified as susceptible. The experimental conditions in this study differed from those in the previous work. In the current study, inoculated stalks were incubated for 6 weeks at 24 ± 3°C instead of for 3 weeks at 20 to 21°C as performed by Abbott et al. (2). The longer period of incubation extended the opportunity for disease development under favorable conditions, approximating more closely the situation of sugarcane germination in the field under Louisiana conditions. Preliminary experiment results comparing different lengths of incubation time after inoculation (from 3 to 9 weeks) indicated that disease continued to develop for 5 to 7 weeks, then disease progress slowed. Based on the results, we conclude that highly resistant clones are rare in the current breeding population.

Currently, there is no resistance evaluation in the cultivar development program. The pathogen is ubiquitous; therefore, the assumption has been that natural infection and selection will take place over the course of the 12-year selection program (12), and susceptible clones will be eliminated due to poor yield. In the inoculated stalk experiments, the mean values for the disease traits were always slightly higher for the progeny than the parents, suggesting some minor selection for resistance has occurred in the parent population. However, the current approach has resulted in a situation in which the parent population is predominantly composed of susceptible clones, and there is a high frequency of susceptibility in the selection population.

Resistance to red rot is apparently complex and is affected by morphological and physiological factors (15). Infection may occur through wounds or nodal tissues (1,15). Evaluation of resistance to red rot of standing cane is most often accomplished with nodal inoculation techniques (15). Evaluation of resistance to red rot as a disease of planted stalks traditionally has been done by the stalk inoculation method (1,2), which was modified for use in our study. Rind penetration and internode damage caused by the sugarcane borer, *Diatraea saccharalis*, provide an important avenue for red rot infection of planted stalks in Louisiana (1,10,14). The stalk inoculation technique used in this study mimics this infection process.

Estimates for narrow-sense heritability and genetic gain based on the RI suggest that resistance to red rot can be increased by recurrent mass selection. Resistance selection should not be based on the component traits (IRS, NP, and NR) because of their low narrow-sense heritabilities and potential genetic gains. Although the narrow-sense heritability estimate for nodes passed was almost the same as for the RI, the expected genetic gain for nodes passed was much lower than that for the RI due to its low genetic variance. The results suggest the disease index, which combines aspects of degree of spread and extent of rotting, provides the best evaluation of red rot resistance.

The narrow-sense heritability results were not in agreement with the findings of Azab and Chilton (5) and Chona (8), which suggested that the degree of parental resistance had little effect on the percentage of resistant progeny. In this study, the lowest RI values were obtained for progeny from crosses between the most resistant clones. These results support Abbott (1) and Atchutharamarao and Sarma (4) who concluded that there was a relationship between resistance in the parents and progeny.

The broad-sense heritability estimates indicate that red rot resistance was not repeatable across years. Disease resistance evaluations were conducted under controlled environmental conditions. This suggests the difference between years was due to preconditioning of the stalks determined by the environmental conditions under which they were grown. Environmental conditions might affect sugarcane morphology and physiology and, thereby, resistance. Stalks for the 1992 experiment were collected from plants that had experienced hurricane-force winds and heavy rain, whereas stalks for the 1993 experiment were collected from drought-stressed plants. The results also could suggest that pathogen virulence varied between years. However, tests of individual isolates gave similar results each year (Z. Yin, J. W. Hoy, and S. B. Milligan, *unpublished data*), and a combination of isolates was used in the experiments.

The low repeatability indicated by the broad-sense heritability estimates from the combined-year model and the poor correlation of disease traits between years indicate that single-year evaluation of resistance is unreliable. Comparison of the genetic gain estimates by single- and multiple-stalk analyses demonstrate that clone resistance evaluations should be based on repeated, multiple-stalk inoculations.

The results from variance component estimation for the cross

TABLE 5. Family variance components, means, and genetic coefficients of variation (GCV) for red rot resistance in sugarcane assessed as different traits or a rot index in 1992 and 1993

	Disease trait[b]							
	Internode rot severity		Nodes passed		Nodes rotted		Rot index	
Parameter[a]	1992	1993	1992	1993	1992	1993	1992	1993
$\hat{\sigma}_f^2$	6.6** ± 107[c]	8.8** ± 123[c]	0.07** ± 0.1[d]	0.10** ± 0.1[d]	0.06** ± 0.12[d]	0.12** ± 0.10[d]	1.2** ± 7.3[e]	2.0** ± 6.8[e]
$\hat{\sigma}_{fd}^2$	16.7** ± 138[c]	14.3** ± 125[c]	0.12** ± 0.1[d]	0.08** ± 0.1[d]	0.15** ± 0.15[d]	0.04** ± 0.10[d]	7.6** ± 9.6[e]	5.0** ± 7.9[e]
$\hat{\sigma}_{o(fd)}^2$	65.0** ± 125[c]	87.3** ± 141[c]	0.94** ± 0.1[d]	0.85** ± 0.1[d]	0.93** ± 0.16[d]	0.88** ± 0.14[d]	31.4** ± 9.0[e]	37.2** ± 9.2[e]
$\hat{\sigma}_\varepsilon^2$	62.2 ± 2.9[c]	67.4 ± 1.7[c]	0.56 ± 0.003[d]	0.61 ± 0.00[d]	0.70 ± 0.003[d]	0.62 ± 0.002[d]	21.3 ± 0.2[e]	28.2 ± 0.1[e]
Family mean	26.7 ± 0.4[f]	25.4 ± 0.4[f]	3.28 ± 0.0[g]	2.93 ± 0.0[g]	3.00 ± 0.05[g]	2.67 ± 0.04[g]	98.7 ± 2.8[h]	88.5 ± 2.5[h]
$GCV_{among\ cross}$ (%)	9.6	11.7	8.0	10.7	8.0	12.8	11.0	15.9
$GCV_{within\ cross}$ (%)	30.2	36.8	29.6	31.4	32.2	35.1	56.7	68.9

[a] $\hat{\sigma}_f^2$ = family variance; $\hat{\sigma}_{fd}^2$ = family by test date variance interaction; $\hat{\sigma}_{o(fd)}^2$ = genotypic variance within family by date; $\hat{\sigma}_\varepsilon^2$ = error variance; $GCV_{among\ family} = 100\hat{\sigma}_f$/mean; $GCV_{within\ family} = 100\hat{\sigma}_{o(fd)}$/mean.

[b] Internode rot severity: the average percent discoloration in the inoculated internode and in four internodes on each side of the inoculation point. Nodes passed: the number of nodes passed by internode rot symptoms in each direction from the inoculation point. Nodes rotted: the number of nodes in the assessed area with rot symptoms. Rot index: internode rot severity × nodes passed. Values are given ± standard error. ** = mean squares significant at $P = 0.01$.

[c] Percent squared.

[d] Number squared.

[e] Percent number squared × 10^2.

[f] Percent.

[g] Number.

[h] Percent number.

TABLE 6. Family variance components, means, and genetic coefficients of variation for red rot resistance in sugarcane assessed as different disease traits for 1992 and 1993 combined

Parameter[a]	Disease trait[b]			
	IRS	NP	NR	RI
$\hat{\sigma}_f^2$	4.6** ± 434[c]	0.002** ± 3.39[d]	–0.001** ± 3.36[d]	0.6** ± 214[e]
$\hat{\sigma}_{fd}^2$	10.3** ± 98[c]	0.02** ± 0.10[d]	0.05** ± 0.10[d]	2.3** ± 6.5[e]
$\hat{\sigma}_{fy}^2$	3.0** ± 84.9[c]	0.08** ± 0.10[d]	0.08** ± 0.10[d]	0.9** ± 6.0[e]
$\hat{\sigma}_{fdy}^2$	4.6** ± 0.4[c]	0.08** ± 0.12[d]	0.04** ± 0.11[d]	3.6** ± 7.6[e]
$\hat{\sigma}_{o(fdy)}^2$	77.8** ± 94.9[c]	0.88** ± 0.10[d]	0.90** ± 0.10[d]	34.3** ± 6.4[e]
$\hat{\sigma}_{\varepsilon}^2$	65.7 ± 1.1[c]	0.60 ± 0.001[d]	0.65 ± 0.001[d]	25.9 ± 0.1[e]
Family mean	25.9 ± 0.3[f]	3.07 ± 0.03[g]	2.81 ± 0.03[g]	92.6 ± 1.9[h]
$GCV_{among\ cross}$ (%)	8.2	1.4	0	8.5
$GCV_{within\ cross}$ (%)	34.0	30.5	33.9	63.3

[a] $\hat{\sigma}_f^2$ = family variance; $\hat{\sigma}_{fd}^2$ = family by test date variance interaction; $\hat{\sigma}_{fy}^2$ = family by year variance interaction; $\hat{\sigma}_{fdy}^2$ = family by test date by year variance interaction; $\hat{\sigma}_{o(fdy)}^2$ = genotypic variance within family, date, and year; $\hat{\sigma}_{\varepsilon}^2$ = error variance; $GCV_{among\ cross}$ = genetic coefficient of variation among families; $GCV_{within\ cross}$ = genetic coefficient of variation within crosses.

[b] IRS = internode rot severity: the average percent discoloration in the inoculated internode and in four internodes on each side of the inoculation point. NP = nodes passed: the number of nodes passed by internode rot symptoms in each direction from the inoculation point. NR = nodes rotted: the number of nodes in the assessed area with rot symptoms. RI = rot index: IRS × NP. Values are given ± standard error. ** = mean squares significant at $P = 0.01$.

[c] Percent squared.

[d] Number squared.

[e] Percent number squared × 10^2.

[f] Percent.

[g] Number.

[h] Percent number.

TABLE 7. Correlation coefficients of experiment data within and between years for evaluation of red rot resistance in sugarcane assessed as different disease traits

Correlation	Clones[a]	Disease trait[b]			
		IRS	NP	NR	RI
Among dates within 1992	Parents	0.57**	0.32	0.42*	0.45**
	FM[c]	0.18	0.17	0.15	0.09
Among dates within 1993	Parents	0.48*	0.49*	0.32	0.64**
	FM	0.18	0.24	0.31	0.11
Between 1992 and 1993	Parents	0.07	–0.03	0.13	–0.06
	FM	0.40*	0.07	0.10	0.26

[a] Different progeny from a cross were used on different dates within a year. Although the same 10 progeny were used in the 2 years, their individual identities were not retained. * and ** = significantly different at $P = 0.05$ and 0.01, respectively.

[b] IRS = internode rot severity: the average percent discoloration in the inoculated internode and in four internodes on each side of the inoculation point. NP = nodes passed: the number of nodes passed by internode rot symptoms in each direction from the inoculation point. NR = nodes rotted, the number of nodes in the assessed area with rot symptoms. RI = rot index: IRS × NP.

[c] FM = family mean.

progeny indicated that genetic variances among cross families were low but significantly different, whereas genetic variances within families were high and repeatable over years. This again suggests that red rot resistance could be effectively improved by determining and making the best crosses then focusing selection on progeny within those crosses. However, the low repeatability of clone red rot reactions suggests that effort should be devoted to accurately determining parent resistance reactions and making appropriate crosses rather than large-scale evaluation of red rot resistance in progeny populations.

Considered altogether, the experimental results have important implications for the sugarcane cultivar development program in Louisiana. Development of cultivars by breeding and selecting for only a single trait is not feasible in any crop-breeding program. Sugarcane cultivar selection will continue to be based on multiple traits. Resistance to red rot can be improved by evaluating resistance in potential parents and utilizing the most resistant clones in crosses. However, high levels of resistance are rare in the current parent population, so new sources of resistance need to be identified.

TABLE 8. Broad-sense genetic correlation coefficients of disease traits used for red rot evaluation in sugarcane for 1992 and 1993 combined

Disease trait[a]	Genetic correlation coefficients for disease traits[b]					
	NP		NR		RI	
	Parents	FM	Parents	FM	Parents	FM
IRS	0.61	0.62	0.59	0.61	0.88	0.89
NP			0.86	0.82	0.81	0.80
NR					0.74	0.71

[a] IRS = internode rot severity: the average percent discoloration in the inoculated internode and in four internodes on each side of the inoculation point. NP = nodes passed: the number of nodes passed by internode rot symptoms in each direction from the inoculation point. NR = nodes rotted: the number of nodes in the assessed area that were rotted.

[b] RI = rot index = IRS × NP. FM = family mean. r_g significant at $P = 0.01$ for all values.

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