Inheritance of Resistance to Leaf Freckles and Wilt Caused by *Clavibacter michiganense* subsp. *nebraskense* in Early Maturing Maize Inbred Lines

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ABSTRACT

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The inheritance of resistance to leaf freckles and wilt was studied in two greenhouse and two field trials of a set of diallel crosses between 10 early maturing maize inbred lines. Inheritance of resistance in six F_2 populations derived from resistant × susceptible and intermediate × susceptible crosses was also studied in the greenhouse and field. General combining ability was much more important than specific combining ability for disease resistance. General combining ability effects were generally consistent between trials, whereas specific combining ability effects were not. Additive genetic effects accounted for most of the variation among generation means in populations derived from resistant × susceptible crosses, and less so in populations from intermediate × susceptible crosses. Broad-sense heritabilities were high, and estimates of gene numbers were low in F_2 populations derived from resistant × susceptible crosses. Resistance to leaf freckles and wilt in the more resistant inbred lines appears to be controlled by relatively few genes (3–5) acting in a mostly additive manner. Development of resistant germ plasm using a variety of breeding methods, including pedigree, bulk, backcross, and recurrent selection methods, should be successful, particularly if material is evaluated in more than one environment.

Additional keywords: corn, Goss's wilt, *Zea mays*.

Leaf freckles and wilt, also known as Goss's wilt, is caused by the bacterium *Clavibacter michiganense* subsp. *nebraskense* (syn. *Corynebacterium michiganense* subsp. *nebraskense*) and is a relatively new and potentially destructive disease of maize (*Zea mays* L.) in the United States (14,15,22). First found in Nebraska in the late 1960s, leaf freckles and wilt has since been found in Colorado, Kansas, Iowa, South Dakota, Wisconsin, and Illinois (22). Leaf freckles and wilt has caused grain yield losses of up to 44% in susceptible hybrids in controlled tests (5,6). Although yield losses due to leaf freckles and wilt are significantly correlated with disease severity, not all hybrids with high foliar disease ratings sustain significant losses, indicating possible tolerance to leaf freckles and wilt. In a yield loss study of a closely related set of hybrids, tolerance of hybrids varied between years, indicating that it was not stable (5). Susceptible sweet maize hybrids sustained primary ear weight losses in excess of 17% in 2 yr of a 3-yr yield loss assessment study (13). In a separate study, leaf freckles and wilt reduced the total ear weight of a susceptible sweet maize hybrid by 99% when inoculated at the three- to five-leaf stage (17).

Genetic resistance is the primary means of controlling leaf freckles and wilt in maize. Maize inbred lines and hybrids vary in reaction from resistant to highly susceptible, although no maize genotype is considered immune (2,12,14–16,22). Reactions of maize to artificial inoculation with *C. m. nebraskense* is dependent on plant age at inoculation and inoculum concentration (4,17). Preliminary reports of the inheritance of resistance to leaf freckles and wilt indicate that F_1 hybrids were either intermediate in reaction or approached that of the more susceptible parent, and that probably more than one locus was involved (7,10,18). Subsequent reports indicated additive gene action was responsible for most of the variation in resistance, although dominance for resistance was seen in some crosses involving highly resistant inbreds (19,20).

Because of the apparent spread of leaf freckles and wilt into more northern areas of the U.S. maize belt and because most of the reports of the inheritance of leaf freckles and wilt resistance have dealt with maize inbred lines poorly adapted to the northern maize belt, we decided to study the inheritance of resistance to leaf freckles and wilt in adapted, early-maturing maize inbred lines.

MATERIALS AND METHODS

Ten inbred lines (SD18 and A662 [resistant]; SD24, CM105, W64A, A661, and A619 [intermediate]; A632, A634, and W117 [susceptible]) were crossed in a diallel manner to produce 45 nonreciprocal F_1s, which were evaluated in field and greenhouse trials. The greenhouse trial was conducted twice in the spring of 1982. Each trial consisted of two replications of five single plant subsamples of each F_1 arranged in a completely randomized design. Each plant was grown in an 11-cm diameter plastic pot in a steam-pasteurized soil mix. Seedlings were inoculated 21 days after planting (two- to four-leaf stage) using the pinprick inoculation technique (1,3). The same set of F_1s was evaluated in field experiments in the 1982 and 1983 growing seasons on the South Dakota State University Agronomy Research Farm at Brookings. Agronomic practices (tillage, previous crop, fertilizer, and herbicide applications) were identical in both years. Experimental units consisted of single rows 4.5 m long and 100 cm apart with plants spaced 30 cm apart within rows. The experiment was arranged as a randomized complete block design with three replications. Five plants in each plot were inoculated at the five- to six-leaf stage using the pinprick inoculation technique. An additional five plants in each plot were inoculated at anthesis by injecting 2 ml of a suspension of *C. m. nebraskense* into the second ear node using a 50 cm^3 Vaco pistol grip rubber plunger syringe (Ideal Instruments, Chicago, IL).

Inoculum was prepared by mixing a suspension of six isolates of *C. m. nebraskense* that were maintained as stock cultures at 4 C in slant tubes of nutrient broth-yeast extract (NBY) agar. Each isolate was streaked from the stock cultures onto NBY plates and grown for 48–72 h at 25 C. Cells were washed from the plates with distilled water, and the resulting cell suspension was adjusted to a final concentration of either 1.5×10^7 (seedling

inoculations in the field and greenhouse) or 3×10^8 cfu ml^{-1} (adult plant inoculations in the field) using a spectrophotometer.

The inheritance of leaf freckles and wilt resistance was also studied in the F_2 generation of selected crosses. F_1 crosses of resistant × susceptible (A662 × A632, A662 × W117, SD18 × A632, SD18 × W117) and intermediate × susceptible (A619 × A632, A619 × W117) inbred lines were self-pollinated to produce F_2 seed. The parental inbred lines, F_1, and F_2 generations of each cross were evaluated for leaf freckles and wilt reaction in the greenhouse and field. In the greenhouse experiments, 10 plants each of the parent inbred lines and the F_1 and 210 plants of the F_2 were evaluated in each of four replications in a randomized complete block arrangement during the winter and spring of 1983. The actual number of plants in the F_2 generation evaluated in each experiment varied from 744 to 840 due to poor germination. Planting and inoculation methods were identical to those described previously for the diallel study. In the field study conducted during the 1983 growing season, two rows of the parental inbred lines and F_1, and 20 rows of the F_2 were grown in each of three replications arranged in a randomized complete block design. Actual numbers of plants of each generation evaluated varied due to variable emergence, loss of plants due to cultivation injury, and missed inoculations. The number of plants evaluated ranged from 33 to 70 for parental lines and F_1[S], and from 488 to 599 for F_2[S]. Plot size, plant spacing, and row spacing were identical to that described for the field diallel experiments. Leaf freckles and wilt inoculation methods were also identical to those used in the diallel, except that plants were only inoculated in the seedling stage.

Plants inoculated as seedlings in both greenhouse and field experiments were rated 10–12 days after inoculation. Later disease ratings were not used, because no further disease development was observed. A 0–4 rating scale in which 0 = no symptoms; 1 = some localized spotting around the inoculation site; 2 = extensive wilting of tissue at the inoculation site with some spread to surrounding leaf tissue; 3 = extensive blighting and spread on inoculated leaves; 4 = evidence of systemic infection (seedling stunted, wilted, or killed). Plants inoculated at anthesis were not rated due to poor symptom development.

Data from the diallel crosses were analyzed using method 4, model I of Griffing (8) to estimate general and specific combining abilities (GCA and SCA) for leaf freckles and wilt resistance.

TABLE 1. Analyses of variance for a set of diallel crosses among 10 early maize inbred lines for leaf freckles and wilt reaction in field and greenhouse trials

	Field			Greenhouse		
Source	df	MS (1982)	MS (1983)	Source	df	MS[a]
Replications	2	0.0836	0.0479*			
Crosses	44	0.3661**	0.3761**	Crosses	44	0.6471**
GCA[b]	9	1.1902**	1.2311**	GCA	9	2.3972**
SCA	35	0.1542	0.1563**	SCA	35	0.1971
Error	88	0.1119	0.0116	Error	88	0.1628

[a] *, ** Indicate mean square is significant at the $P = 0.05$ and 0.01 levels, respectively.
[b] GCA = general combining ability; SCA = specific combining ability.

TABLE 2. F_1 cross means (above the diagonal), general combining ability effects (on the diagonal, underlined), and specific combining ability effects (below the diagonal) for leaf freckles and wilt reactions for a set of diallel crosses among early maize inbred lines evaluated in two greenhouse trials

	Inbred line[a]									
	SD18	A662	SD24	CM105	W64A	A661	A619	A632	A634	W117
SD18	−0.34*	1.00	1.65	1.00	1.75	1.75	2.00	2.10	1.90	1.90
A662	−0.39*	−0.25*	1.05	1.75	1.90	2.10	2.00	1.90	2.10	2.00
SD24	0.29	−0.40*	−0.28*	1.25	1.25	2.00	2.05	2.30	2.00	2.00
CM105	−0.38*	0.27	−0.20	−0.25*	1.90	1.45	2.20	2.00	2.10	2.10
W64A	0.21	0.27	−0.35	0.27	−0.10	1.90	2.10	1.90	2.00	2.30
A661	0.09	0.34	0.27	−0.30	−0.01	0.03	2.00	2.15	2.30	2.35
A619	0.10	0.00	0.08	0.21	−0.05	−0.27	0.27*	2.55	2.45	2.55
A632	0.18	−0.11	0.32	−0.01	−0.26	−0.14	0.02	0.29*	2.45	2.70
A634	−0.01	0.10	0.03	0.10	−0.15	0.02	−0.06	−0.08	0.27*	2.65
W117	−0.08	−0.05	−0.05	0.03	0.07	0.00	−0.04	0.09	0.05	0.35*

[a] Indicates estimate is significantly different from zero as its absolute value exceeds twice its standard error.

TABLE 3. F_1 cross means (above the diagonal), general combining ability effects (on the diagonal, underlined), and specific combining ability effects (below the diagonal) for leaf freckles and wilt reactions for a set of diallel crosses among early maize inbred lines evaluated in 1982 field trials at Brookings, SD

	Inbred line[a]									
	SD18	A662	SD24	CM105	W64A	A661	A619	A632	A634	W117
SD18	0.01	1.26	1.80	2.40	1.93	2.00	2.06	1.93	2.13	2.46
A662	−0.30	−0.44*	1.53	1.33	1.53	1.33	2.00	2.06	1.93	1.86
SD24	−0.19	0.00	−0.01	2.13	1.86	1.80	2.13	2.26	2.26	2.06
CM105	0.43*	−0.19	0.18	−0.03	1.46	1.60	1.73	2.46	2.00	2.60
W64A	−0.01	0.04	−0.06	−0.44*	−0.06	2.13	1.80	2.13	2.20	2.40
A661	0.18	−0.04	0.00	−0.19	0.38*	−0.18*	2.00	1.86	1.53	2.26
A619	0.10	0.02	0.19	−0.20	−0.09	0.22	−0.04	2.06	2.26	2.06
A632	−0.30	0.28	0.05	0.26	−0.03	−0.19	−0.13	0.23*	2.33	2.70
A634	−0.01	0.24	0.13	−0.11	0.12	−0.43*	0.16	−0.04	0.15*	2.46
W117	0.10	−0.05	−0.29	0.27	0.10	0.08	−0.26	0.11	−0.05	0.37*

[a] * Indicates estimate is significantly different from zero as its absolute value exceeds twice its standard error.

Data from parental inbred lines, F_1, and F_2 generations from crosses were analyzed by weighted generation mean analysis, by fitting means to a weighted regression model, $Y = m + a_1d + a_2h$, in which Y is the mean of a given generation, m is the intercept, d is the pooled additive genetic effect, h is the pooled dominance genetic effect, and a_1 and a_2 are the relative contributions of these effects to each generation mean (11). Generation means were weighted with the reciprocals of their squared standard errors. Broad-sense heritability of leaf freckles and wilt reactions in the F_2 populations were also estimated in the manner of Kelly and Bliss (9): $h^2 = [V_{F2} - (V_{P1}, V_{P2}, V_{F1}$, pooled)$]/V_{F2}$, in which V_{P1}, V_{P2}, V_{F1}, and V_{F2} are the within plot variances of the parental inbred lines, F_1, and F_2 generations, respectively. The number of genes (K) conditioning leaf freckles and wilt reaction in F_2 populations was estimated according to Mather and Jinks (11): $K = [1/2(P_1 - P_2)^2]/V_{F2} - (V_{P1}, V_{P2}, V_{F1}$, pooled).

RESULTS

Highly significant differences among reactions of F_1 hybrids to leaf freckles and wilt were observed in greenhouse and field trials (Table 1). The F_1 cross × trial interaction term in the combined analysis of the two greenhouse trials was not significant, so data from both trials were combined, and the pooled error term was used to test for significance. The F_1 cross × year term was highly significant in the combined analysis of the field trials, although its magnitude relative to the main effects was small. Because of this significant interaction and the apparent heterogeneity between error terms in the analysis from each year, individual year data were analyzed separately. GCA for leaf freckles and wilt reaction was highly significant in all the analyses, accounting for 76 and 67% of the variation in reaction of the F_1 crosses in the greenhouse and field trials, respectively. SCA for disease reaction was significant only in the 1983 field experiment.

GCA and SCA effects for inbred lines and their F_1 crosses, respectively, are presented in Tables 2–4. There was generally good agreement between trials for estimates of GCA effects for the 10 parental inbred lines, although estimates from the greenhouse and 1983 field trials were more highly correlated ($r = 0.93$, $P = 0.01$) with each other than they were with the estimates from the 1982 field trial ($r = 0.62$, $P = 0.10$; $r = 0.78$, $P = 0.01$, respectively). GCAs of the three most susceptible inbred lines (A632, A634, and W117) were all significantly positive (toward increased susceptibility) in all trials, whereas GCA effects of the two most resistant inbred lines (A662 and SD18) were significantly negative in all trials (with the exception of SD18 in the 1982 field trial). SCAs of the crosses were quite variable, and there was no significant correlation between estimates from the different trials.

Additive genetic effects were highly significant in all six of the populations studied in greenhouse and field trials (Table 5). The distributions of reactions in the F_1 and F_2 generations were intermediate between those of the parent lines, with the exception of the A619 × W117 F_1 in the greenhouse trial, and the A619 × A632 F_1 in the field trials (Figs. 1,2). Significant dominance genetic effects were found in all populations in greenhouse trials and in three of the populations in the field trial. Mean F_1 reactions were significantly less than the midparent value in five of the six crosses in the greenhouse and field (Figs. 1,2). Only the SD18 × W117 F_1 did not follow this pattern. Residual effects were significant in four of the six populations in greenhouse trials and in five populations in the field trial. Broad-sense heritability estimates in the six F_2 populations varied from 21 to 80% (Table 6). Estimates were higher in F_2 populations derived from resistant

TABLE 4. F_1 cross means (above the diagonal), general combining ability effects (on the diagonal, underlined), and specific combining ability effects (below the diagonal) for leaf freckles and wilt reactions for a set of diallel crosses among early maize inbred lines evaluated in 1983 field trials at Brookings, SD

	Inbred line[a]									
	SD18	A662	SD24	CM105	W64A	A661	A619	A632	A634	W117
SD18	−0.21*	2.25	2.36	2.45	2.13	2.50	2.23	2.43	2.45	2.63
A662	0.18*	−0.28*	2.38	2.16	2.00	2.46	2.26	2.08	2.63	2.58
SD24	0.08	0.18*	−0.08*	2.43	2.16	2.33	2.53	2.83	2.76	2.65
CM105	0.30*	0.09	0.16*	−0.21*	2.51	2.42	2.60	2.43	2.06	2.33
W64A	−0.04	−0.09	−0.14*	0.34*	−0.19*	2.62	2.33	2.60	2.53	2.70
A661	0.11	0.15*	−0.18*	0.04	−0.21*	0.03	2.58	2.75	2.85	2.80
A619	−0.25*	−0.14*	−0.08	0.12*	−0.17*	−0.14*	0.12*	3.26	2.95	3.32
A632	−0.17*	−0.44*	0.11*	−0.16*	−0.02	−0.08	0.33*	0.24*	3.10	3.53
A634	−0.12*	0.14*	0.06	−0.51*	−0.06	0.04	0.05	0.08	0.21*	3.45
W117	−0.09	−0.06	−0.20*	−0.39*	−0.04	−0.16*	0.27	0.36*	0.31*	0.36*

[a] * Indicates estimate is significantly different from zero as its absolute value exceeds twice its standard error.

TABLE 5. Analyses of variance of generation means for leaf freckles and wilt reaction in six maize populations in the field and greenhouse

Population	df	SD18 × W117	A662 × W117	SD18 × A632	A619 × W117	A662 × A632	A619 × A632
Greenhouse							
Replications	3	0.006	0.005	0.002	0.007	0.003	0.003
Generations	3						
Additive effects	1	9.680**[a]	9.680**	14.580**	1.445**	15.125**	4.061**
Dominance effects	1	0.162*	1.163**	0.350**	2.108**	0.224**	0.765**
Residual effects	1	0.168*	0.571**	0.029	0.053	0.706**	0.139**
Error	9	0.016	0.008	0.007	0.025	0.013	0.007
Field							
Replications	2	0.010	0.002	0.003	0.002	0.006*	0.004
Generations	3						
Additive effects	1	11.207**	9.779**	19.226**	0.499**	10.088**	0.730**
Dominance effects	1	0.033	0.018*	0.135	0.111*	0.156**	0.778**
Residual effects	1	0.071*	0.376**	0.171*	0.028	0.153**	1.982**
Error	6	0.011	0.003	0.029	0.011	0.001	0.002

[a] *, ** Indicate mean square is significant at the P = 0.05 and 0.01 levels, respectively.

× susceptible crosses than in those derived from intermediate × susceptible crosses. Estimates of the number of effective factors (genes) segregating in the F_2 populations ranged from 0.79 to 4.60 (Table 6).

DISCUSSION

The relative importance of GCA in the diallel analyses and the high proportion of variation among generation means due to additive genetic effects in most of the F_2 populations studied indicate that additive gene action is of primary importance in the inheritance of resistance to leaf freckles and wilt in the early maturing inbred lines studied. Nonadditive gene action (dominance and epistasis) was of minor importance, except in the two F_2 populations derived from crosses of intermediate × susceptible inbred lines (A619 × W117 and A619 × A632). Some partial dominance for resistance was indicated by the significant deviations of F_1 means from midparent values in five of the six crosses studied, which is in contrast to some studies in which partial dominance for susceptibility was reported (7,10,18), but is in agreement with the observations of Treat et al (19,20). Furthermore, GCA estimates (measures of additive gene action) for the parent inbred lines were fairly consistent between trials, particularly for inbred lines at the extremes of the resistance-susceptibility spectrum. In general, resistant inbred lines tended to contribute resistance to the F_1, and susceptible inbred lines contributed susceptibility, whereas the behavior of crosses among intermediate parents was somewhat less predictable. SCA estimates were not consistent between any of the trials. For example, the crosses A662 × SD18 and A662 × SD24 showed significant negative SCA effects in the greenhouse trials, but showed significant positive effects in the 1983 field trial. This indicates that nonadditive genetic effects interact more with the environment than do additive genetic effects. This is consistent with the observations of Treat et al (20), who found significant dominance × year and residual × year effects in two crosses involving the resistant inbred line Mo17. The importance of genotype × environment interaction on leaf freckles and wilt reaction of maize hybrids has been documented previously (5). Taken together, these data indicate that the reaction of the parental inbred lines to leaf freckles and wilt is a good indicator of their behavior in hybrid combination, but that evaluation should be conducted over several environments to get a precise estimate.

The behavior of inbred lines in hybrid combination does not appear to be related to the origin or pedigree of those lines. Four of the parental lines used in the diallel experiments (A632, A634, CM105, and SD24) are closely related, early maturing inbreds with the inbred line B14 representing 75–93% of their parentage. The two most susceptible of these inbred lines, A632 and A634, consistently showed positive GCA effects, whereas CM105 and SD24 showed negative GCA effects for leaf freckles and wilt reaction. A661 and A662 were both developed from the same synthetic variety (AS-A, University of Minnesota), but the more resistant line, A662, showed consistently more negative GCA effects. These observations suggest that genetic variability for leaf freckles and wilt reaction probably can exist in germ plasm with

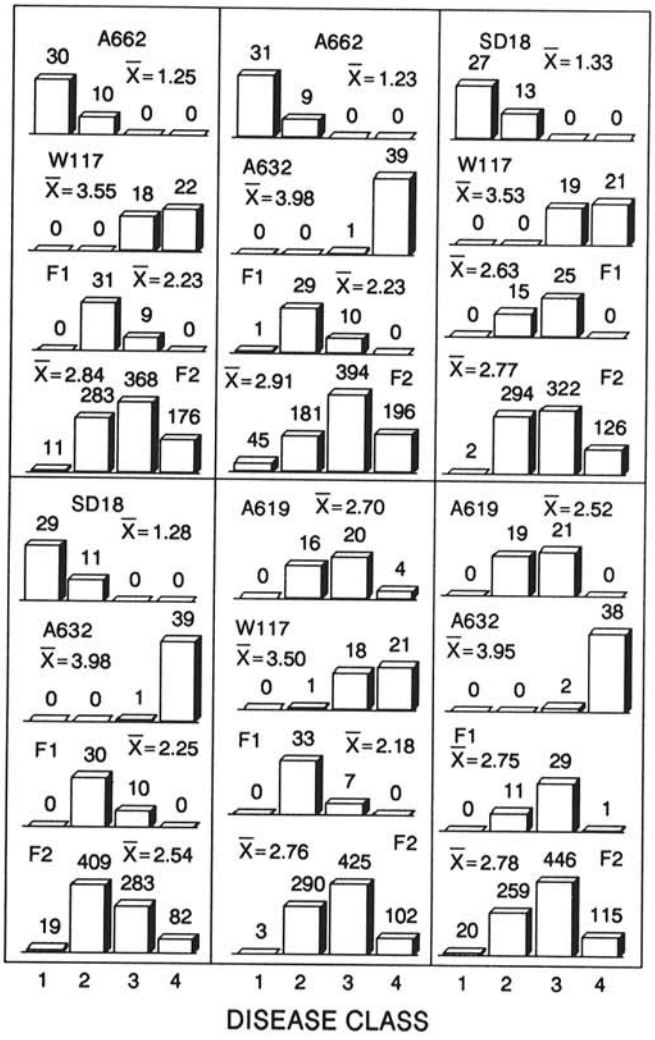

Fig. 1. Frequency distributions and means of leaf freckles and wilt ratings of maize populations in the greenhouse, 1983.

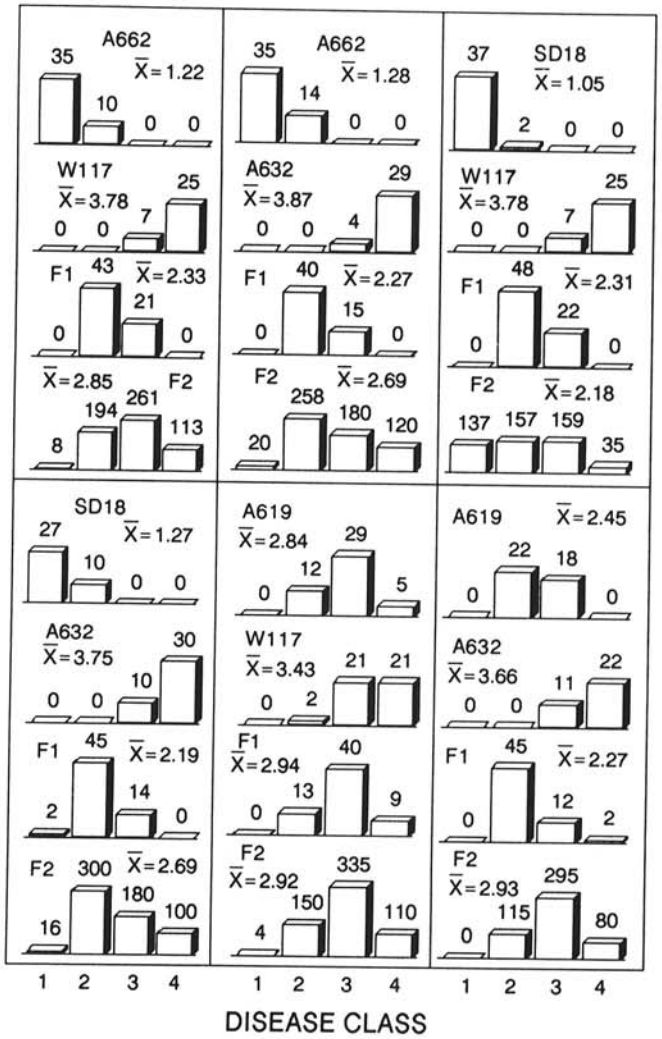

Fig. 2. Frequency distributions and means of leaf freckles and wilt ratings of maize populations in the field, 1983.

TABLE 6. Estimates of broad-sense heritability (h^2) and number of genes (K) governing resistance to leaf freckles and wilt in six maize populations

Population	Environment	h^2	K
A662 × W117	Greenhouse	0.77	3.80
	Field	0.63	4.60
A662 × A632	Greenhouse	0.77	3.72
	Field	0.73	3.27
SD18 × W117	Greenhouse	0.52	4.52
	Field	0.80	2.70
SD18 × A632	Greenhouse	0.71	5.08
	Field	0.64	4.07
A619 × W117	Greenhouse	0.47	0.79
	Field	0.21	0.92
A619 × A632	Greenhouse	0.61	1.69
	Field	0.33	2.83

a narrow genetic base that has undergone selection for other agronomic traits such as yield, early maturity, etc.

The relatively high broad-sense heritability of resistance to leaf freckles and wilt, particularly in the resistant × susceptible crosses indicates that these populations should respond favorably and rapidly to selection. It should be cautioned, however, that these estimates probably are overestimates of actual heritability, because they were estimated from data from only a single environment. Although nonadditive gene action was apparently of minor importance in the crosses studied, genotype × environment interaction was significant, and therefore selection for resistance in a single environment would not be as effective as selection based on reactions measured over multiple environments.

Our estimates of the number of genes for resistance to leaf freckles and wilt from the six F_2 populations evaluated in both the field and greenhouse appear to be in general agreement. Although estimates of the number of effective factors (or genes) segregating in a population are imprecise and may be based on incorrect assumptions, they are reasonable estimates of the relative magnitude of gene number (11). Our estimates of the relatively low numbers of genes conditioning resistance to leaf freckles and wilt is consistent with the observation that so-called "polygenic" disease resistance is usually controlled by a few genes (21). Our estimates are also consistent with the observation that resistance to leaf freckles and wilt has been successfully transferred into a susceptible inbred line (A632) background using a modified backcrossing method in which generations of selfing or sibbing of resistant plants are alternated with backcrossing to the susceptible recurrent parent (5).

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