Inheritance of Resistance to Head Smut Disease in Corn

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Abstract. The inheritance in corn (*Zea mays* L.) of resistance to head smut disease incited by *Sphacelotheca reiliana* (Kuhn) Clinton was studied in the field on crosses of resistant dent corn line N6 with two susceptible sweet corn (*su*) inbred lines. Disease incidence in the resistant parent (Pr) was 0% to 4%, and 83% to 96% in the susceptible parent (Ps). Predisposition of seedlings by clipping just above ground level increased percent infected in progeny populations by as much as 4-fold, but did not affect disease incidence in the parents. At the lower disease incidence of unclipped plots, the F₁, F₂, and BCr means were close to the mean of Pr, suggesting dominance of resistance. At the high disease incidence of clipped plots, the relationship of parent and progeny means "suggested additive inheritance. Epistasis was also generally present with a higher level indicated for unclipped plots. Inheritance was concluded to be quantitative. Reciprocal differences were observed only in backcrosses. In the F₂ and BCs populations, plants grown from dent (*Su*) seed were lower in disease incidence than plants grown from *su* seed.

Head smut, incited by *Sphacelotheca reiliana*, affects corn, sorghum, and sudan grass (Halisky, 1963). The disease is initiated by infection of seedlings, either through the roots or coleoptile (Kruger, 1962). The mycelium becomes systemically established in the meristem and eventually produces large sori that replace the normal ears and sometimes form in the tassel (Halisky, 1963; Potter, 1914). Because loss of the ear is assured when the seedling is infected, degree of host susceptibility is expressed by disease incidence. Corn cultivars differ in potential for infection under a particular set of conditions that involve environmental factors such as soil moisture, level of smut inoculum in the soil, and host predisposition (Ali and Baggett, 1986; Baggett and Koepsell, 1983; Baier and Kruger, 1962; Halisky, 1963; Mack et al., 1984; Matyac and Kommedahl, 1985). Wide differences in cultivar susceptibility to head smut have been reported (Baggett and Koepsell, 1983; Foster and Frederiksen, 1977; Kruger, 1962; Ma et al., 1983; Stromberg et al., 1984).

Several studies of the inheritance of resistance to head smut have been reported. Mei et al. (1982), in China, reported that the inheritance of resistance is quantitative. In 69 of 85 crosses studied, inheritance was mainly additive, with the F₁ close to the midparent in disease incidence. In 14 of 85 crosses, disease incidence was higher than in the midparent, and in 2 of 85 crosses, disease incidence was lower than in either parent, indicating resistance was over-dominant. Ma et al. (1983), also in China, reported that resistance was quantitatively inherited and was dominant, recessive, or controlled by the interaction of several non-allelic genes, depending on the parents. Additive gene effects were also important and there was a positive correlation between resistance in the F₁ and the parents. Stromberg et al. (1984) found that F₁ hybrids were intermediate between Pr and Ps in smut incidence.

Our objective was to elucidate the inheritance of resistance to head smut as expressed by infection incidence in the field under natural conditions but with high infection potential provided by seed furrow inoculation. We also sought to compare

infection data obtained at high and low levels of infection incidence achieved through predisposition of a portion of the plants by clipping in the seedling stage (Ali and Baggett, 1986). An additional objective was to determine whether there is a relationship between resistance level and sweet vs. dent endosperm type in the genetic background.

Materials and Methods

The Pr in these studies was Nebraska 6 (N6), an inbred line of *Su*₁ endosperm type. Ps were SM7, an early maturing *su*₁ inbred line obtained from Crookham Seed Co. (Caldwell, Idaho), and SD-1, a *su*₁ corn inbred line we derived by self-pollination and selection from the highly susceptible commercial F₁ hybrid 'Sugar Daddy'. Crossing and selfing of the F₁ to produce seed were done in either the field or greenhouse using standard methods of pollination control. Crosses were made reciprocally (line to line). Backcrosses BCs and BCr were made reciprocally between the F₁ and Ps and between the F₁ and Pr, respectively. After determination of the extent of reciprocal differences, reciprocals were combined for genetic analysis.

Parents and progenies were tested at the Oregon State Univ. Vegetable Research Farm in 1982 and 1983. A standard plot consisting of a single row 6 m long was used in a randomized

Table 1. Relationship of year, clipping treatment, and endosperm type to head smut incidence.

Comparison ^z	Uninfected (No.)	Infected		χ^2
		Number	Percent	
Year				
1982	5,891	3,998	39.8	653**
1983	14,015	4,717	25.2	
Clipping				
Unclipped	11,968	3,639	23.3	775**
Clipped	7,938	4,976	38.5	
Endosperm type				
(<i>Su</i> ₁)	6,634	3,303	33.2	122**
(<i>su</i> ₁)	4,968	3,472	41.1	

^zAll parents and progenies combined for the year and clipping comparisons. F₂ and BCs combined in the dent-sweet endosperm comparison.

**Significantly related at $P = 0.01$; values derived from contingency table χ^2 test for independence.

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Table 2. Incidence of head smut in unclipped and clipped parents and progenies, endosperm types, and reciprocals combined.

Generation ²	1982				1983			
	Unclipped		Clipped		Unclipped		Clipped	
	No. plants tested ³	Percent infected ⁴	No. plants tested	Percent infected	No. plants tested	Percent infected	No. plants tested	Percent infected
<i>N6 × SD-1</i>								
Pr (N6)	113	4.0 a	94	0.0 a	355	1.8 a	298	1.0 a
BCr	452	3.7 ab	395	11.4 b	988	1.4 a	863	10.2 b
F ₁	117	9.3 b	125	36.2 c	458	3.5 ab	427	28.5 c
F ₂	885	25.6 c	836	40.4 c	1644	8.4 b	1388	24.9 c
BCs	879	63.6 d	759	76.6 d	1730	31.5 c	1437	60.1 d
Ps (SD-1)	229	96.3 e	161	91.3 e	207	90.5 d	132	93.0 e
MP		50.2		45.6		46.2		47.0
<i>N6 × SM-7</i>								
Pr (N6)	113	4.0 a	94	0.0 a	355	1.8 a	304	1.0 a
BCr	482	2.8 a	472	10.4 b	801	1.5 a	633	18.1 b
F ₁	80	1.3 a	82	12.5 b	458	4.5 ab	366	33.3 c
F ₂	905	22.8 b	795	40.3 c	1539	8.9 b	1254	31.3 c
BCs	860	50.2 c	606	66.8 d	1633	33.6 c	1124	61.3 d
Ps (SM-7)	168	90.2 d	87	83.2 e	156	95.0 d	63	95.8 e
MP		47.1		41.6		48.4		48.4

²Pr = resistant parent, Ps = susceptible parent, BCr = backcross F₁ × Pr, BCs = backcross F₁ × Ps, MP = midparent value.

³Number of plants shown is total of four replications.

⁴Means of four replications. Mean separation in columns within crosses by Duncan's multiple range test, *P* = 0.05, using arcsin-transformed percent infection.

Table 3. Analysis of variance for arcsin-transformed percent head smut infection, comparing generations, years, and clipping vs. no clipping; endosperm types and reciprocals combined.

Source	df	<i>N6 × SD-1</i>		<i>N6 × SM7</i>	
		Mean squares	F value	Mean squares	F value
Years ²	1	869.5	9.95*	32.4	0.37 ^{NS}
Replication within years	6	87.4	2.79*	88.3	2.05 ^{NS}
Generations	5	11,007.1	78.53**	10,991.4	42.04**
Clipping	1	1,482.3	6.27 ^{NS}	2,004.2	8.57 ^{NS}
Years × generations	5	140.2	4.48**	261.4	6.09**
Years × clipping	1	236.4	7.55**	233.9	5.44*
Generations × clipping	5	369.6	11.81**	383.2	8.92**
Year × generation × clipping	5	9.05	0.29 ^{NS}	2.0	0.05 ^{NS}
Error	66	31.30		43.0	
Total	95				

²Replications within years used as error term to test years.

^{NS},*,**Nonsignificant or significant at *P* = 0.05 or 0.01, respectively.

complete-block design with four replications. Number of plants per plot varied from 30 to 40 after thinning. All entries in each replication were duplicated to permit clipping (see below) and non-clipping of the seedlings. Clipped and non-clipped plots were randomly distributed. Additionally, generations segregating for dent-sweet endosperm type (F₂ and BCs) included segregated dent and sweet plots in a paired arrangement. In 1982, the Ps and F₂ were planted in double the minimum number of plots to increase population sizes. The reciprocal F₁ was omitted because of insufficient seed. Thus, in 1982, the number of 6-m plots in each replication was two for Pr and F₁, four for Ps, eight for BCr, and 16 for the F₂ and BCs. In 1983, the number of plots of Pr, F₁, F₂, and backcross plots was increased to three or four times the minimum. Total numbers of plots in 1983 were four Ps, six Pr, eight F₁, 16 BCr, 32 F₂, and 32 BCs plots in each replication. Data from plots of the same treatment (clip-

ping and endosperm type) and from the same replication were pooled for analyses.

Head smut inoculum, consisting of spores bulked from field infections of the previous season, was applied with the seed by means of a V-belt planter. About 0.4 liter of inoculum (50 cc teliospores/liter of slightly dampened vermiculite) was used in each 6-m plot as described by Baggett and Koepsell (1983). Rows were 1.1 m apart and previously banded with granular fertilizer. General culture, including weed control, was typical of that used in commercial sweet corn in the area, except for irrigation. Immediately after planting, only enough sprinkler irrigation was applied for plant establishment. Irrigation was then avoided during the early stages to enhance infection (Baier and Kruger, 1962; Mack et al., 1984). After the infection period, water was applied as needed to maintain moderate growth.

Plants in the plots designated for clipping were cut off just

Table 4. Head smut disease incidence in reciprocal backcross progenies of N6 $\times$ SM7 and N6 $\times$ SE-1.

Cross and generation	1982		1983	
	Infected ² (%)	Reciprocal difference ^{3,*}	Infected ² (%)	Reciprocal difference ³
N6 $\times$ SM7				
BCr (N6 $\times$ F ₁)	7.9		12.5	
		2.5 +		5.5*
BCr (F ₁ $\times$ N6)	5.4		7.0	
BCs (SM7 $\times$ F ₁)	64.0		54.4	
		10.9**		13.8**
BCs (F ₁ $\times$ SM7)	53.1		40.6	
N6 $\times$ SD-1				
BCr (N6 $\times$ F ₁)	8.2		7.8	
		1.6 +		4.1**
BCr (F ₁ $\times$ N6)	6.6		3.7	
BCs (SD-1 $\times$ F ₁)	75.2		50.4	
		10.0 +		9.4**
BCs (F ₁ $\times$ SD-1)	65.2		41.0	

²Mean of four replications.

³Reciprocal difference tested by *t* test.

*+ Greater than SE.

*,**Significant at *P* = 0.05 or 0.01, respectively.

above ground level when four or five leaves were visible. Almost all of these clipped plants recovered and grew to maturity.

Infection incidence was determined at crop maturity when disease symptoms were fully expressed. A plant was counted as infected if sori were found on an ear or tassel. There was no attempt to classify symptoms by severity or type on individual plants.

Genetic parameters were estimated by means of a three-parameter joint scaling test adapted by Rowe and Alexander (1980)

from a method described by Cavalli (1952) and Mather and Jinks (1971). The test involves weighted least squares, where the weights are the reciprocals of the SE of the generation means.

Results and Discussion

The incidence of head smut was satisfactory in both 1982 and 1983 for differentiation of resistant and susceptible genotypes. Overall disease incidence was higher in 1982 than in 1983 and higher in clipped than in unclipped plots as shown for the crosses and generations combined (Table 1). High χ^2 values from contingency table tests indicated that year and clipping treatments were not independent of disease incidence. In the F₂ and BCs, plants with *su*₁ endosperm had significantly higher infection incidence than plants with *su*₂ endosperm.

The Pr ranged from 0% to 4% infected, while the Ps were 83% to 96% infected. Disease incidence of the parents was only slightly and inconsistently affected by year and clipping, while all progeny generations of each cross were greatly affected (Tables 2 and 3). F values for the interactions years $\times$ generations and clipping $\times$ generations were significant at *P* = 0.01, but small relative to F values for generation. The significant interactions were due to differences in magnitude of generation means with year and clipping treatment, but did not involve a change in rankings.

Reciprocals of the F₁ and F₂ populations were not significantly different in percent infection and were combined in each case for data presentation. Significant differences existed among some reciprocal backcrosses. In backcrosses to Pr or Ps incidence of infection was generally higher when the original parent was used as female than when the F₁ was used (Table 4). These differences were possibly related to seedling vigor as affected by the seed parent. Because the reciprocal differences were not considered likely to affect the overall interpretation of inheri-

Fig. 1. Incidence of corn head smut among the means of parents and progenies of N6 $\times$ SM7 seedlings, unclipped and clipped, in relation to the percentage of susceptible genotype (1983).

Table 5. Estimates of genetic parameters using joint scaling tests of arcsin-transformed generation means.

Cross	Clipping treatment	Gene effects ^z				
		Mean	Additive	Dominance	χ ^{2y}	P
1982						
N6 × SD1	Unclipped	45.4 ± 3.0	−42.4 ± 2.55	−29.1 ± 5.6	9.2	0.01–0.05
	Clipped	43.2 ± 3.6	−41.5 ± 2.6	−6.5 ± 7.3	8.9	0.02–0.05
N6 × SM7	Unclipped	46.6 ± 3.5	−36.7 ± 3.1	40.6 ± 6.1	16.3	0.001
	Clipped	52.0 ± 4.65	−38.9 ± 5.1	−29.8 ± 7.0	29.9	<0.001
1983						
N6 × SD1	Unclipped	34.9 ± 5.0	−33.0 ± 4.5	−30.6 ± 9.1	38.2	<0.001
	Clipped	41.2 ± 2.7	−36.9 ± 2.7	−10.6 ± 5.2	9.4	0.02–0.05
N6 × SM7	Unclipped	33.9 ± 4.8	−31.1 ± 4.1	−30.0 ± 9.2	33.2	<0.001
	Clipped	37.2 ± 3.5	−34.7 ± 3.3	−8.15 ± 6.4	15.3	0.01–0.001

^z± = SE of the estimate, where applicable.

^yAll χ^2 values indicate significant deviation (at $P = 0.05$) from an additive-dominance model.

tance patterns, reciprocal backcrosses were combined in all other data analyses.

Inheritance patterns. The relationship of the parents and progeny varied with the level of disease incidence, especially as affected by clipping and, to a much lesser degree, by year (Table 1). The relationship of population means is shown graphically (Fig. 1) for N6 × SM7 in 1983 (the graph for N6 × SD-1 would be almost identical). In Fig. 1, a straight-line configuration would indicate that inheritance is entirely additive. Under the lower infection level of the unclipped plots, the relationship of population means showed a pattern expected when resistance is dominant, with the F_1 and BCr mean percent infected almost as low as the mean of Pr and the means of BCS lower than the mid-parent values. In the clipped plots, with the means of both F_1 and F_2 approaching the mid-parent values and BCs means higher than the mid-parent values, the pattern approached that expected for additive inheritance with slight dominance. This change from a dominance to an additive pattern is related to the low response of the parents at the high and low extreme of the percent infection range compared to the large response of the F_1 , F_2 , and backcrosses.

Disease incidence was lower in the F_1 than in the F_2 in every case except for clipped N6 × SM7 in 1983, but the difference was only significant in two cases in 1982 (Table 2). A lower disease incidence in the F_1 was possibly related to a higher level of seedling hybrid vigor.

The generation means analysis (Table 5) generally agreed with the data presented in Table 2 and represented by the graphically presented generation means of N6 × SM7, 1983 (Fig. 1). Additive and dominance effects were usually about equal in the unclipped plots. In clipped plots, the additive effects were usually slightly higher than in unclipped, but the dominance effects were much lower. Chi-square values, which indicated possible epistatic effects, were all significant at $P \leq 0.05$ and were usually lower in clipped than in unclipped plots.

Although the expression of resistance and susceptibility in individual plants is qualitative and the frequency distribution for the progenies includes only two classes, genetic control of susceptibility to infection is quantitative. This conclusion is supported additionally by the continuous range of susceptibility found in corn cultivars (Foster and Frederiksen, 1977; Kruger, 1962; Ma et al., 1983; Stromberg et al., 1984). It is important to recognize that the varying disease incidence, as affected by environment and factors that might predispose the corn plant to infection, can influence data interpretation and thus the apparent

inheritance pattern in a given test. In this study, the level of disease incidence obtained in the clipped plots probably exceeded levels normally encountered in commercial corn production. Thus, from an economic viewpoint, corn breeders may consider resistance to have a degree of dominance over susceptibility. However, at higher disease levels, the responses of all progenies suggest that additive gene action is predominant and corn breeders should attempt to incorporate as much resistance as possible in all inbred lines to be used as parents in F_1 hybrids. Selection for the highest levels of resistance should be practiced in early and later generations under high infection levels.

Plant characteristics, such as dent vs. sweet endosperm type, probably can affect susceptibility in an indirect manner through an effect on seedling vigor. However, unpublished cultivar tests done at this station indicate that sweet corn germplasm with high levels of resistance is available.

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