Yellow- and White-Endosperm Effects on Stewart's Wilt of Maize

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ABSTRACT

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Stewart's wilt of maize (*Zea mays*), caused by *Erwinia stewartii*, has sporadically been an economically important disease in the central corn belt states in the United States. The major objectives of this investigation were to study via a diallel mating design the genetics of resistance to Stewart's wilt in yellow- and white-endosperm maize. Natural Stewart's wilt infection of a diallel cross involving yellow × yellow endosperm (20 single crosses including reciprocals) and white × white endosperm (20 single crosses including reciprocals) indicated that general combining ability effects were more important than specific combining ability effects. The mean Stewart's wilt rating (visual scale: 1 = resistant, 5 = intermediate, and 10 = susceptible) was lower ($P = 0.01$) for white-endosperm single crosses (3.3) than that for yellow-endosperm single crosses (3.8). Heritability estimates from white-endosperm single crosses, yellow-endosperm single crosses, and across both endosperms were 70, 37, and 40%, respectively. We concluded that the white-endosperm gene (*y*) might be associated with some modifier genes that were responsible for greater genetic differentiation of the white-endosperm single crosses. Low heritability estimates for Stewart's wilt suggested that progress from selection in the material studied would be slow.

Additional keywords: bacterial wilt, host-pathogen interaction.

Stewart's wilt or leaf blight caused by *Erwinia stewartii* (E. F. Smith) Dye occurs on both dent corn (*Zea mays* L.) (1,6,7) and sweet corn (1,8). Earlier, emphasis on this disease was in the 1930s (2,9). There was very limited emphasis on Stewart's wilt in the 1960s and 1970s (2). In the 1980s, resurgence of Stewart's wilt occurred as indicated by recent reports (1,7,8). Stewart's wilt has caused almost 100% loss of Golden Bantam sweet corn (9). The disease in dent corn also can result in total yield loss. For these reasons, a better understanding of resistance to Stewart's wilt is needed. The results of genetic studies on resistance to Stewart's wilt are not conclusive. For example, Ivanoff and Riker (5) indicated that resistance to Stewart's wilt was conditioned by a partially dominant gene. Wellhausen (11) reported two complementary, dominant genes (*Sw1* and *Sw2*) to be responsible for resistance. Smith (10) reported that resistance was governed by two major dominant genes (*Sw1* and *Sw2*) and two modifier (also major) genes (*Sw3* and *Sw4*). Blanco et al (2) reported that host reaction to Stewart's wilt was conditioned by additive genetic effects and that the estimated number of genes varied between 5 and 9. Quantification of yield loss in dent corn from Stewart's wilt has not been done.

There are no published reports relative to the effect of endosperm color gene (*YYY* = yellow endosperm, *yyy* = white endosperm) on severity of Stewart's wilt infection. In addition, there are few reports on reciprocal effects. Also, heritability of Stewart's wilt resistance is not known. The present study provides information on the genetic effects conditioning Stewart's wilt in a yellow-endosperm and a white-endosperm diallels, on reciprocal effects, and on heritability estimates for Stewart's wilt reaction in corn (maize).

MATERIALS AND METHODS

A yellow-endosperm line and a white endosperm line were derived from each of the following five sources (a superscript indicates how many dosages of a line are present) by selfing: 1) Mo14W × Oh7B (50% Mo14W and 50% Oh7B genetic complement), 2) $Mo14W^2 \times Oh7B$ (75% Mo14W and 25% Oh7B genetic complement), 3) $Mo14W^3 \times Oh7B$ (87.5% Mo14W and 12.5% Oh7B genetic complement), 4) $Mo14W \times Oh7B^2$ (25% Mo14W and 75% Oh7B genetic complement), and 5) $Mo14W \times Oh7B^3$ (12.5% Mo14W and 87.5% Oh7B genetic complement).

The parental line Mo14W had white endosperm (genetic constitution = *yyy*) and Oh7B had yellow endosperm (genetic constitution = YYY). The 10 lines used in the study were at the S_4 stage of inbreeding. In the summer of 1975, all possible crosses were made among the white-endosperm lines and among the yellow-endosperm lines in the Corn Breeding Nursery at the University of Missouri-Columbia. Each diallel set contained 20 single crosses, including reciprocal crosses. Parental lines were excluded.

The 40 F_1 hybrids (single crosses) were grown in a randomized, complete block design with four replications at two locations at Columbia, MO. The two locations, about a mile apart, differed in soil type and planting date. One location was planted on 20 May 1976 and the other on 26 May 1976. Twenty-six kernels were planted in 13 hills per plot, 30 cm apart in 3.9-m row lengths. Row-to-row spacing was 90 cm. Thirty days after seedling emergence, plots were thinned to one plant per hill.

Because of high levels of the corn flea beetle (*Chaetocnema pulicaria* Melsh.) and *E. stewartii*-inoculated experiments in the vicinity of this study at both locations, natural Stewart's wilt infection occurred in the experimental material. A scale of 1 through 10 was used to rate the disease severity, 1 = little or no leaf tissue destroyed, 3 = 30% leaf tissue destroyed, 5 = 50% leaf tissue destroyed, 7 = 70% leaf tissue destroyed, and 10 = almost 100% leaf tissue destroyed. The visual ratings were made on a plot basis approximately 90 days after planting. The data ranged from 1 to 10 with a mean rating of 3.55. One replication at the second location was not used because of raccoon damage. Data were analyzed according to diallel method 3, model 1 (genotypes or single crosses being fixed effect) (4,12). Locations were regarded as a random effect. The appropriate general linear model for data combined over endosperms was as shown below:

$$\begin{aligned} X_{ijklm} = {} & u + g_i + g_j + s_{ij} + r_{ij} + L_k + b_l + \text{endo}_m \\ & + g_i * L_k + g_j * L_k + s_{ij} * L_k + r_{ij} * L_k \\ & + g_i * \text{endo}_m + g_j * \text{endo}_m + s_{ij} * \text{endo}_m + r_{ij} * \text{endo}_m \\ & + g_i * \text{endo}_m * L_k + g_j * \text{endo}_m * L_k + s_{ij} * \text{endo}_m * L_k \\ & + r_{ij} * \text{endo}_m * L_k + e_{ijklm}, \end{aligned}$$

where X_{ijklm} is the observed value (rating), u is the population mean,

g_i and g_j are the general combining ability effects for parents i and j, respectively; s_{ij} is the specific combining ability effect for the cross between i_{th} and j_{th} parents; r_{ij} is the specific reciprocal effect associated with cross of the i_{th} and j_{th} parents such that $r_{ij} = -r_{ji}$, L_k is the effect of the k^{th} location, b_l is the effect of the l^{th} replication, endo_m is the effect of the m^{th} endosperm, $g_i * L_k$, $g_j * L_k$, $s_{ij} * L_k$, and $r_{ij} * L_k$ are interactions of the given effects with k^{th} location, $g_i * \text{endo}_m$, $g_j * \text{endo}_m$, $s_{ij} * \text{endo}_m$, are the interactions of endo_m with the given effects; $g_i * \text{endo}_m * L_k$, $g_j * \text{endo}_m * L_k$, $s_{ij} * \text{endo}_m * L_k$, $r_{ij} * \text{endo}_m * L_k$ are the three-way interactions involving g_i, g_j, s_{ij}, and r_{ij} effects, endo_m and L_k effects, and e_{ijklm} is experimental error term.

Heritability (broad sense) among F_1 hybrid means was estimated according to Colbert et al (3) and is given below:

$$H = \sigma^2_{F_1} / (\sigma^2_{F_1} + \sigma^2_{F_1} \times L/l + \sigma^2_e/rl)$$

where H = heritability (broad sense), $\sigma^2_{F_1}$ = F_1 variance component, $\sigma^2_{F_1}$ = L/ l = F_1 by location interaction variance component, σ^2_e/rl = error variance component, l = number of locations, and r = number of replications. The above formula is for within-endosperm analysis.

Heritability estimates were determined for each endosperm separately as well as across both endosperm types. A combined diallel analysis across endosperms was computed. For the combined data, heritability was determined by using the following equation:

$$H = \sigma^2_{F_1} / (\sigma^2_{F_1} + \sigma^2_{F_1} \times \text{endo/endo} + \sigma^2_{F_1} \times L/l + \sigma^2_{F_1} \times \text{endo} \times L/\text{endo } 1 + \sigma^2_e/rl \text{ endo})$$

where $\sigma^2_{F_1} \times \text{endo} / \text{endo} = F_1$ by endosperm interaction variance component; $\sigma^2_{F_1} \times \text{endo} \times L/\text{endo } 1$ = three-way interaction of $\sigma^2_{F_1}$, endosperm, and location, and endo = number of endosperm types; and all the other terms were the same as defined above.

RESULTS AND DISCUSSION

A combined analysis of variance for Stewart's wilt ratings across yellow- and white-endosperm color is shown in Table 1. Differences in resistance were associated with endosperm color ($P = 0.01$). Because the endosperm color had a highly significant mean square, we decided to do analyses of variance for the yellow- and white-endosperms separately. The analyses of variance by endosperm color provided additional information on interaction effects not involving endosperm interactions. The general combining ability (GCA) effects also were significant ($P = 0.01$). However, the specific combining ability (SCA) effects and reciprocal effects were not significant (Table 1). When analyses of variance were done within endosperm color types, the GCA effects were larger in white-endosperm hybrids ($P = 0.01$) than in the yellow-endosperm hybrids ($P = 0.05$). SCA effects were significant in the yellow-endosperm single crosses but nonsignificant in the white-endosperm crosses (Table 2). The SCA effects were nonsignificant in the analyses combined over endosperm color (Table 1). The separation of the two endosperm colors in the analyses provided the additional information that SCA was significant in the yellow-endosperm crosses. The GCA * L interaction was significant across endosperm types in the study. However, the separate analyses revealed that GCA * L interaction was significant for yellow endosperm and was not significant for white-endosperm crosses (Table 2). This means that both the endosperm types need to be treated separately for certain effects. The combined analysis of variance should be separated before drawing conclusions across the two groups of endosperms.

The mean Stewart's wilt rating for white endosperm F_1 hybrids was lower (3.3) than that for yellow endosperm F_1 hybrids (3.8). The difference of 0.5 was highly significant (1% probability). The location by endosperm interaction (Table 1) was also highly significant, which indicated that the effect of endosperm color varied with location.

The GCA × endosperm interaction was significant (1% level, Table 1). The GCA × location effect was significant at the 5% level. The GCA effects of line 1 (G1) and of line 2 (G2) across both endosperms were significant. The partitioning of GCA × location interaction revealed that G_2 × location effect was significant (data not shown here or in Tables). The GCA effects of the other four lines were consistent across locations. Partitioning of the GCA × endosperm interaction showed that G_1, G_2, and G_4 effects gave significant interaction with endosperm color.

From the combined analysis (Table 1), heritability (broad sense) of the F_1 hybrid means was calculated to be 40%. Narrow-sense heritability, direct calculation of which is not advisable (because of model 1: fixed effects for genotypes) would be expected to be smaller than 40% because narrow-sense heritability cannot be greater than the broad-sense heritability. Nonadditive genetic effects as well as nongenetic effects were important in determining Stewart's wilt rating.

Because endosperm color of the F_1 hybrids caused a significant variation, analyses of variance were conducted separately for the white-endosperm diallel and the yellow-endosperm diallel (Table 2). The F_1 hybrids were significantly different for resistance to Stewart's wilt in the two endosperms. The SCA was significant only for yellow-endosperm crosses.

In the white-endosperm crosses, G_1 and G_2 effects were significant (Table 3). Resistance to Stewart's wilt was in G_1 and G_2, but may have been lost in the other three parents. In the yellow-endosperm crosses, SCA effect involving lines 1 and 2 (S_{12}) was significant. The G_1 effect in the yellow-endosperm material was

TABLE 1. Combined analysis of variance for Stewart's wilt across white-endosperm and yellow-endosperm diallels

Source of variation	df	Mean square[a]
Locations (L)	1	7.28**
Replications:L	5	12.88**
Genotypes (G)	39	1.86**
Endosperm (EN)	1	20.57**
F_1 hybrids (Hyb.)	19	1.09**
GCA	4	3.60**
SCA	5	0.78
Reciprocal (Recip.)	10	0.24
EN × Hyb.	19	1.65**
EN × GCA	4	5.87**
EN × SCA	5	1.21
EN × Recip.	10	0.19
L × G	39	1.12**
L × EN	1	24.00**
L × Hyb.	19	0.63
L × GCA	4	1.82*
L × SCA	5	0.17
L × Recip.	10	0.38
L × EN × Hyb.	19	0.41
L × EN × GCA	4	0.17
L × EN × SCA	5	0.72
L × EN × Recip.	10	0.36
Pooled error	195	0.54

[a] *, ** Significant at the 5 and 1% level, respectively.

TABLE 2. Analyses of variance for Stewart's wilt from white-endosperm and yellow-endosperm F_1 hybrids

		Mean square[a]	
Source of variation	df	White endo.	Yellow endo.
Locations (L)	1	28.86**	2.42*
Replications: L	5	8.72**	5.33**
F_1 hybrids (Hyb.)	19	1.97**	0.78*
GCA	4	8.39**	1.09*
SCA	5	0.25	1.78*
Reciprocal (Recip.)	10	0.26	0.15
L × Hyb.	19	0.55	0.49
L × GCA	4	0.80	1.19*
L × SCA	5	0.49	0.35
L × Recip.	10	0.49	0.28
Pooled error	95	0.62	0.43

[a] *, ** Significant at the 5 and 1% level, respectively.

TABLE 3. Estimates of general combining ability effects (G_i) and specific combining ability effects (s_{ij}) for Stewart's wilt reaction in white-endosperm and yellow-endosperm F_1 hybrids, and across the two endosperms (combined)

Statistic	White endosperm	Yellow endosperm	Combined
G_1	−1.01*[b]	−0.59*	−1.31**
G_2	0.82*	0.39	1.13**
G_3	0.35	0.28	0.46
G_4	0.22	0.08	0.39
G_5	−0.39	−0.16	−0.67
s_{12}	0.45	−0.94*	1.90
s_{13}	−0.53	0.66	−1.84
s_{14}	−0.31	0.57	−1.30
s_{15}	0.39	−0.28	1.24
s_{23}	−0.19	0.26	−0.53
s_{24}	0.31	0.01	0.74
s_{25}	−0.57	0.68	−2.11
s_{34}	0.27	−0.54	1.04
s_{35}	0.45	−0.37	1.34
s_{45}	−0.28	−0.03	−0.48

[a] Standard error: first, second, and third values, respectively, represent white, yellow, and both endosperms combined. SE (G_i) = 0.109, 0.091, 0.10; SE ($G_i - G_j$) = 0.17, 0.14, 0.16 ($i \neq j$). SE (s_{ij}) = 0.15, 0.12, 0.14; SE ($s_{ij} - s_{im}$) = 0.24, 0.20, 0.23, ($i \neq j, m$). SE ($s_{ij} - s_{mn}$) = 0.17, 0.14, 0.16 ($i \neq j, m, n$; $m \neq n$).

[b] *, ** denote significance from zero at the 5% and 1% level, respectively.

significantly different from zero at the 5% level of probability. No other GCA, SCA, and reciprocal effects were significant (Table 3).

Parental line 1, i.e., (Mo14W × Oh7B) S_4 had a negative GCA effect in both white and yellow-endosperm crosses, indicating that the use of this line in breeding programs could be conducive to reducing Stewart's wilt. In addition, parental line 5, i.e., (Mo14W × Oh7B[3]) S_4 (both white and yellow endosperm versions) might be effective in reducing Stewart's wilt rating. The remaining three lines showed positive GCA effects. Therefore, these three lines in both endosperm color versions appeared to possess genes that contributed to susceptibility to Stewart's wilt (Table 3).

The sign of the GCA effects of a particular line was the same in each endosperm color as well as across endosperms (Table 3). However, the SCA effects, in general, did not have the same sign in the two endosperm versions. When the GCA or SCA effects are not significant, we can assume that the experimental error was large in value.

The broad-sense heritability estimate of the F_1 hybrid means for the white-endosperm material was 70% and for the yellow-endosperm material 37%. This difference indicated that genotypic differentiation was greater in the white-endosperm F_1 hybrids than in the yellow-endosperm F_1 hybrids. This suggested that the white-endosperm gene (*y*) might be associated with some modifier genes that were responsible for the greater genetic differentiation of the white-endosperm F_1 hybrids.

The coefficient of variation for the white-endosperm F_1 hybrids was 28.7% and for the yellow-endosperm F_1 hybrids was 19.5%. The C.V. for the combined data was 23.8%. These C.V.'s were in the acceptable range; therefore, naturally infected material can provide reasonably precise information.

The genetics of resistance to Stewart's wilt probably involves two genetic systems; one may be controlled by one or two genes and the other may be governed by a host of modifier genes with small but additive effect. Further genetic studies are needed to elucidate the genetic systems conditioning Stewart's wilt.

The *y* gene might have a pleiotropic effect, or a block of genes may be closely linked to the *y* allele. This block of genes may be passed on from one generation to the next as essentially a single unit. Effects of single gene on quantitative traits have been reported (6).

Because of relatively low heritability estimates, progress from cyclic selection would be expected to be slow in the germ plasm studied here. However, if one or two genes conditioning Stewart's wilt could be identified, a backcross method could be used to improve resistance to Stewart's wilt of an inbred.

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