Importance of the Husk Covering on the Susceptibility of Corn Hybrids to Fusarium Ear Rot

C. Y. Warfield, Graduate Research Assistant, and **R. M. Davis,** Cooperative Extension Specialist, Department of Plant Pathology, University of California, Davis 95616

ABSTRACT

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The role of the husk covering and the influence of intra-ear thrips infestation on severity of Fusarium ear rot (*Fusarium moniliforme*) were evaluated in 1990, 1991, and 1992 among corn (*Zea mays*) hybrids previously rated as susceptible, intermediate, or resistant to Fusarium ear rot. For the three treatments imposed on individual corn ears in 1990, ears were either left untreated, wrapped with a strip of parafilm at the ear tip to maintain a tight seal, or husk layers were split open along one side of the ear 1 to 2 weeks after pollination to partially expose the developing kernels. In 1991 and 1992, two additional treatments, split husks followed by acephate insecticide application and insecticide application to ears with intact husks, were added. Split husks compromised the natural barrier to insects and fungi and significantly increased Fusarium ear rot severity among all corn hybrids in 1990 and 1991. In 1992, a year with naturally high levels of disease, split husks significantly increased disease severity among the resistant hybrids, but did not affect the susceptible hybrids. Acephate applied to ears with split husks reduced ear rot among all hybrids. Grain yields were significantly less in ears with split husks for all hybrids in 1990 and 1991, and for the resistant hybrids in 1992. Grain yields were higher for all insecticide-treated hybrids each year. These results demonstrate that susceptibility to Fusarium ear rot is influenced by both husk morphology and intra-ear thrips infestation.

Fusarium moniliforme J. Sheld. (Teleomorph: *Gibberella fujikuroi* (Sawada) Ito in Ito & K. Kimura) is the most widespread and economically important Fusarium species on corn (*Zea mays* L.) in the United States (8), and it commonly infects a wide range of crop plants throughout the world. Although the fungus has been associated with both stalk and root rots of corn, in northern California it is responsible for significant losses in both grain yield and quality. Disease symptoms are usually observed first at the tip or base of the ear, or where insects have channeled between rows of kernels. Infection is characterized by intact or split kernels covered with white or pinkish white mycelium. The husk layers often become tightly bound to the kernels by a mat of fungal mycelium. In severe cases of infection, the entire ear may be consumed by the fungus, resulting in a low-quality, lightweight ear (3). *F. moniliforme* may also produce several mycotoxins that affect grain quality (11). This is an important concern from a food-safety standpoint because symptomless kernels may be infested with the fungus.

Corresponding author: C. Y. Warfield
E-mail: cywarfield@ucdavis.edu

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The husk layer has been implicated as a factor in the susceptibility of corn hybrids to infection by *F. moniliforme*. Koehler (7) observed that infection by *F. moniliforme* was increased in relation to injuries at the tip of the ear, or by the failure of the husk to protect the tip. Kommedahl and Windels (8) reported that the percentage of infected kernels was lower when the tips of the developing ears were covered by the husks, compared to ears with exposed tips. Cassini (1) noted that corn cultivars with ears poorly covered by the "spathes" are the most susceptible to ear rot. Farrar and Davis (3) reported that Fusarium ear rot incidence was correlated with husk looseness at the brown silk stage of ear development.

Hesseltine and Bothast (5) suggested that the natural loosening of the husks permits easier entry of insects and consequent fungal contamination. Farrar and Davis (3) correlated Fusarium ear rot of corn to intra-ear thrips population. They reported that reduction of the intra-ear population of western flower thrips (*Frankliniella occidentalis* Perg.) by insecticide treatments reduced disease incidence at maturity. Their work suggested that the husk layer may act as a natural barrier against thrips colonization.

The goal of this study was to evaluate the role of the husk covering and the involvement of thrips in the susceptibility of corn hybrids to Fusarium ear rot in northern California. It was hypothesized that all field corn is susceptible to Fusarium ear rot when the husk, the natural barrier to insects and therefore the fungus, is compromised. To evaluate the importance of the husk covering on ear rot susceptibility, corn hybrids were subjected to treatments designed to alter the husk coverage of developing kernels or to influence thrips colonization within the ear.

MATERIALS AND METHODS

Experimental field trials were conducted at the University of California Armstrong Field Station, Davis, in 1990, 1991, and 1992, and on Staten Island in the Sacramento River Delta Region of San Joaquin County, California, in 1990. Nitrogen (170 kg/ha) was applied as ammonia prior to planting. Rows were planted on 75-cm centers and were furrow irrigated every 2 weeks.

Three Pioneer corn hybrids, 3377, 3295, and 3779, rated as resistant, intermediate, and susceptible to ear rot, respectively, were planted in 1990. Three Pioneer corn hybrids, 3540, 3779, and 3569, rated as susceptible, and two Pioneer corn hybrids, 3377 and 3343, and Asgrow hybrid, RX947, rated as resistant to Fusarium ear rot, were planted in 1991 and 1992. Susceptibility ratings were based on previous field trials (2,6). Planting dates were 12 May (Delta) and 19 May (Armstrong) 1990, 15 May 1991, and 22 May 1992. The experimental design was a randomized complete block, split-plot design with the corn hybrids as the main plots. Each main plot consisted of four rows and was replicated either five (1990) or six (1991, 1992) times. Each replicate was separated by 1.5-m alleys. Main plots were 7.6 m long and seeded at 5.3 seeds per m. Within each main plot, treatments were randomly assigned to single-row subplots.

Three treatments were imposed on corn ears in 1990: (i) nontreated control, (ii) ear tip husk layers wrapped with a strip of parafilm at silk emergence, and (iii) husk layers split along one side of the ear 1 to 2 weeks after pollination to partially expose the developing kernels. Four treatments were imposed on the corn ears in 1991 and 1992: (i) nontreated control, (ii) husk layers split, (iii) split husks followed by insecticide application, and (iv) insecticide application to ears with intact husks. Treatments were applied to 10 primary ears randomly selected from plants within the center of each row. In 1991 and 1992,

four applications of acephate (Orthene 75S, Chevron Chemical, San Ramon, CA) at 0.8 g/liter were made beginning at green silk and repeated at 5- to 8-day (1991) or 12-day (1992) intervals until the late milk to early dough stage of kernel development (22 to 28 days after silking). Silks of the treated ears were sprayed until runoff using a manual pump sprayer. Exposed kernels, as well as silks, were sprayed with acephate for treatments in which the husk layers were split.

At harvest, on 25 October (Delta) and 31 October (Armstrong) 1990, 1 November 1991, and 27 October 1992, husks were completely removed and each ear was visually rated for disease severity using an arcsine pretransformed rating index (9). Ratings were based on the percentage of kernels per ear that exhibited signs or symptoms of *F. moniliforme* infestation, where a rating of 0 = no visually diseased kernels, 1 ≤ 10%, 2 ≤ 35%, 3 ≤ 65%, 4 ≤ 90%, and 5 ≤ 100% kernel infection. Infestation by *F. moniliforme* was confirmed by plating kernels on Nash and Snyder selective medium (10). The mean disease rating for each subplot was calculated and subjected to analysis of variance (MSTATC, Michigan State University, East Lansing). The rating values were then back-transformed to express disease severity as a percentage of total ear infection. In 1991 and 1992, the three susceptible and three resistant hybrids were grouped for analysis.

After rating for disease severity, ears were mechanically shelled. Mean grain yield was calculated from the total weight of kernels collected from the 10 treated ears in each subplot. Yield values were subjected to analysis of variance to derive treatment contrasts.

RESULTS

Ears with split husks and partially exposed developing kernels had significantly ($P \leq 0.05$) higher levels of disease and decreased grain yield for all hybrids at each location in 1990 (Table 1).

There was no significant difference in disease severity or grain yield between the control and the parafilm wrapped ears at either location in 1990 (data not presented). Due to difficulty in maintaining a tight seal, the parafilm offered little or no protection to the tip of the developing ear, and this treatment was omitted in later trials.

In 1991, ears with split husks had significantly ($P \leq 0.05$) more disease for both resistant and susceptible hybrids (Table 2) compared to the control ears. In 1992, ears with split husks had significantly ($P \leq 0.05$) more disease among the resistant hybrid group, but the treatment had no significant effect on disease severity in the susceptible hybrid group (Table 2).

The application of acephate significantly ($P \leq 0.05$) reduced disease severity among the susceptible hybrids in both 1991 and 1992, but did not significantly affect disease severity among the resistant hybrids (Table 2). Applying insecticide to ears with split husks reduced the percentage of infected kernels compared to ears with split husks that received no insecticide application among both resistant and susceptible hybrid groups in 1991 and 1992 (Table 2).

Ears with split husks had significantly ($P \leq 0.05$) reduced grain yield for both resistant and susceptible hybrids in 1991 (Table 3). In 1992, grain yield was significantly ($P \leq 0.05$) reduced among the resistant hybrids when the husks were split; however, there was no effect on yield when the husks of the susceptible hybrids were split (Table 3). Grain yields were significantly ($P \leq 0.05$) increased when both resistant and susceptible hybrids were treated with insecticide in 1991 and 1992 (Table 3).

DISCUSSION

The husk played an important role in the susceptibility of the corn hybrids to Fusarium ear rot. This was consistent with Koehler's (7) report that infections by *F. moniliforme* increased in corn ears with exposed tips compared to completely covered and sound ears. In our study, partially opening the husk layers of both resistant and susceptible hybrids generally increased Fusarium ear rot and decreased grain yield. Disease severity among the resistant hybrids increased when the husks were split, even when acephate was applied to the exposed ears.

Differences in husk morphology were apparent between the resistant and susceptible corn hybrids in our study. The three susceptible corn hybrids were early-maturing hybrids bred to dry down quickly; husk layers were loose and the tip of the maturing cob was often exposed. In contrast, the husk layers were much tighter in

Table 1. Effects of partial husk separation on severity of Fusarium ear rot and grain yield on three corn hybrids in 1990[a]

	Delta site			Armstrong site		
Treatment[b]	**3377**	**3295**	**3779**	**3377**	**3295**	**3779**
	Diseased kernels per ear (%)[c,d]					
Control	5.9	48.7	78.4	9.2	41.9	72.4
Split husk[e]	99.9	99.8	99.0	73.5	76.3	99.2
	Yield (g/ear)[d]					
Control	159	143	124	211	191	139
Split husk	54	42	73	133	144	98

[a] Resistant hybrid = Pioneer hybrid 3377; intermediate hybrid = Pioneer hybrid 3295; susceptible hybrid = Pioneer hybrid 3779.
[b] Treatments were significantly different ($P \leq 0.05$) for all hybrids at each location according to ANOVA.
[c] Percentage of kernels exhibiting visual signs or symptoms of *F. moniliforme* was calculated by % = 100[sine(rating × 18)]^2, based on a pretransformed 0 to 5 rating scale.
[d] Values are means of five replicates of 10 ears each.
[e] Husk layers split 1 to 2 weeks after pollination to partially expose developing kernels.

Table 2. Effects of partial husk separation and insecticide application on the severity of Fusarium ear rot of corn in 1991 and 1992

		Diseased kernels per ear (%)[a]			
		1991		1992	
Treatment		**Susceptible[b]**	**Resistant[c]**	**Susceptible**	**Resistant**
Control		4.5	1.0	81.4	4.5
Split husk[d]		9.4	2.7	65.0	23.9
Split husk + insecticide[d,e]		1.9	0.7	37.2	13.6
Insecticide[e]		0.2	0.1	65.2	4.9
Contrast	df	Sum of squares			
Resistant (R) vs. susceptible (S)	1	3.64*[f]		124.13*	
Insecticide vs. no insecticide	1	4.04*		7.91*	
Control vs. split husk	1	2.39*		0.56	
(R vs. S) × (insect. vs. no insect.)	1	1.45*		2.69*	
(R vs. S) × (control vs. split)	1	0.05		10.74*	

[a] Percentage of kernels exhibiting visual signs or symptoms of *F. moniliforme* was calculated by % = 100[sine(rating × 18)]^2, based on a pretransformed 0 to 5 rating scale. Values are means of six replicates of 10 ears each.
[b] Susceptible hybrids = Pioneer hybrids 3540, 3569, 3779.
[c] Resistant hybrids = Pioneer hybrids 3377, 3343; Asgrow hybrid RX947.
[d] Husk layers split 1 to 2 weeks after pollination to partially expose developing kernels.
[e] Acephate applied four times at 5- to 7-day (1991) or 12-day (1992) intervals beginning at green silk.
[f] * = Significant at $P \leq 0.05$.

Table 3. Effects of partial husk separation and insecticide application on the grain yield of six corn hybrids in 1991 and 1992

		Grain yield (g/ear)[a]			
		1991		1992	
Treatment		Susceptible[b]	Resistant[c]	Susceptible	Resistant
Control		126	166	84	168
Split husk[d]		117	157	84	140
Split husk + insecticide[d,e]		127	162	105	156
Insecticide[e]		134	169	101	166
Contrast	df	Sum of squares			
Resistant (R) vs. susceptible (S)	1	5.06*[f]		14.69*	
Insecticide vs. no insecticide	1	0.15*		0.61*	
Control vs. split husk	1	0.14*		0.34*	
(R vs. S) × (insect. vs. no insect.)	1	0.02		0.12	
(R vs. S) × (control vs. split)	1	<0.01		0.33*	

[a] Values are means of six replicates of 10 ears each.
[b] Susceptible hybrids = Pioneer hybrids 3540, 3569, 3779.
[c] Resistant hybrids = Pioneer hybrids 3377, 3343; Asgrow hybrid RX947.
[d] Husk layers split 1 to 2 weeks after pollination to partially expose developing kernels.
[e] Acephate applied four times at 5- to 7-day (1991) or 12-day (1992) intervals beginning at green silk.
[f] * = Significant at $P \leq 0.05$.

all three of the resistant hybrids, and the tip of the ear was well covered during the early stages of kernel development. Opening the husks of a loosely covered ear would be expected to have less of an effect on disease severity compared to opening the husks of a tightly covered ear. Splitting the naturally loose husks of the susceptible hybrids had no significant effect on ear rot severity in 1992, a year with naturally high levels of disease.

Our results were consistent with those of Farrar and Davis (3), who showed that intra-ear thrips population and husk looseness were related to disease incidence. Thrips were present throughout our experiment, as confirmed by population counts taken in an adjacent field trial. While thrips counts were not made on test ears, acephate applications generally reduced disease severity and grain yield loss among the susceptible hybrids. The insecticide may have provided protection against thrips invasion for the loosely covered ears of the susceptible hybrids and subsequently reduced ear rot infection. When acephate was applied to ears of the resistant hybrids, there was no significant reduction in disease severity. The tight husks of the resistant hybrids may have naturally restricted thrips entry in to the ear. This further supports the role of the husk covering as a barrier to insect entry.

While the husk has an important role in the resistance of corn hybrids to Fusarium ear rot, other resistance factors may be involved. We found that disease levels of the resistant hybrids remained significantly lower than those of the susceptible hybrids, even when the husks were opened. Other maternal tissues (silk, pericarp, placento-chalazal region) and delayed senescence of silks are also involved in the susceptibility of corn to Fusarium ear rot (4,6,12).

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