Relationships Among Ear Morphology, Western Flower Thrips, and Fusarium Ear Rot of Corn

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ABSTRACT

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The relationships among insects, corn (*Zea mays*) ear morphology, and ear rot caused by *Fusarium moniliforme* were studied in 1988 and 1989. Silks on ears of two corn hybrids, one susceptible to Fusarium ear rot and one with an intermediate level of resistance, received applications of the insecticides acephate or carbaryl at the green silk stage before the onset of ear rot symptoms. In both years, insecticide treatments reduced intra-ear populations of western flower thrips (*Frankliniella occidentalis*) at the brown silk stage and reduced disease incidence at maturity. In 1989, 15 corn hybrids, representing a range of susceptibility to Fusarium ear rot, were examined for ear morphology factors that may be correlated with disease incidence. Factors examined were heat units to silking; days

from initial green silk to yellow-brown silk and to brown silk stages; intra-ear thrips populations at the green, yellow-brown, and brown silk stages; and husk looseness at the yellow-brown and brown silk stages. Disease incidence was correlated with thrips populations at the brown silk stage and with husk looseness at the brown silk stage but was not correlated with the other factors measured. Hybrids also could be separated by contrast analysis into susceptible, intermediate, and resistant groups on the basis of thrips populations and husk looseness at the brown silk stage. On the basis of these data, intra-ear thrips populations and husk tightness at the brown silk stage are important in the epidemiology of Fusarium ear rot.

Additional keyword: maize.

Fusarium moniliforme J. Sheld. (teleomorph: *Gibberella fujikuroi* (Sawada) Ito in Ito & K. Kimura) is a fungus of worldwide distribution that causes diseases of various crops. In northern California, *F. moniliforme* causes a serious ear rot of corn (*Zea mays* L.). Ear rot is generally characterized by scattered infected kernels covered with a white to pinkish weft of mycelia, but in California ears are often completely consumed by disease, leaving a light-weight ear with inner husks cemented to the kernels by mycelia. Although *F. moniliforme* has been described as weakly pathogenic to mildly virulent, it can cause large losses due to decreases in grain quality and yield. Fusarium ear rot is also of concern because of its correlation with human esophageal cancer in Africa (7), equine leucoencephalomalacia (19), and other toxic effects on animals (12,17).

Fusarium ear rot currently is controlled by planting resistant hybrids. However, even the most resistant hybrids often are infected with the fungus and occasionally suffer losses in northern California. Current attempts to breed for resistance to ear rot are slow and costly because of the need to screen hybrids in areas with a high incidence of disease (2). High natural disease incidence is necessary because inoculation attempts under natural conditions often fail and inoculation attempts under artificial, nonwounded conditions meet with limited success (5,18). Inoculations in conjunction with kernel or ear wounding are successful, although of limited value because of the compromise of possibly important natural barriers to infection.

The mode of entry of *F. moniliforme* into kernels is still unclear. In a study on Fusarium stalk rot, Foley (4) found *F. moniliforme* systemically within corn plants and proposed, but did not prove, that this infection leads to ear rot. Koehler (6) found *F. moniliforme* in decreasing order of prevalence, in the silks, kernels, pedicels, vascular cylinder, and butt of the cob, and claimed that this indicated the path of infection. He also observed that ears well covered by husks had less ear rot. Scott and King (15) showed that genetic contributions to the embryo and the endosperm by

the pollen have no effect on infection and disease (i.e., resistance to ear rot is under the genetic control of the mother plant). While they suggest that the site of resistance is the pericarp of the kernel, their data do not rule out the possibility that silks or husks are the site of resistance. Studies that positively correlate insect damage to kernels and incidence of ear rot seem to corroborate the pericarp theory because such damage breaches an important defense against infection (10,16).

An increase in ear rot has been correlated with kernel damage from earworm feeding (16). In Minnesota, picnic beetles from buried corn debris and adult picnic beetles captured on standing corn ears were contaminated with *F. moniliforme* (20). In our early studies (2), the two-spotted mite (*Tetranychus urticae* Koch) was hypothesized to be a vector for *F. moniliforme*. Although the two-spotted mite was not an effective vector, high populations of western flower thrips (*Frankliniella occidentalis* Perg.) were observed within the corn ears in fields throughout northern California.

The objectives of this study were to explore the role of insects in the epidemiology of ear rot and to examine several ear morphology and silk aging factors that vary between hybrids, and compare these with the susceptibility of the hybrids.

MATERIALS AND METHODS

Field experimental design and cultural practices. The experiments were conducted at the University of California, Davis (Armstrong Field Station), on loam soil and at the Sacramento River Delta region of San Joaquin County, CA, on peat soil in 1988 and 1989. On the Armstrong site, rows were planted on 75-cm centers and irrigated by furrow every 2 wk. At the Delta site, rows were planted on 90-cm centers and were irrigated as needed by "spud ditch" (a 20- × 60-cm irrigation ditch cut every eight rows). Nitrogen (170 kg/ha) was applied as ammonia before planting at both locations. Each planting generally received a single cultivation for weed control except the 1989 Delta site, which was not cultivated and had to be weeded by hoe and hand-operated mechanical mower.

In 1988, two hybrids (Pioneer hybrid 3295, which has an intermediate level of resistance to Fusarium ear rot (2), and Pioneer hybrid 3779, a susceptible hybrid; Pioneer Hi-Bred International, Inc., Des Moines, IA) were planted in a randomized complete block design. The Armstrong site was planted on 9 May and the Delta site on 19 May. The four-row plots were 8 m long, seeded at 8.3 seeds per meter, and replicated six times. Data were collected from the middle two rows.

In 1989, hybrid 3779 was planted 12 May at the Armstrong site and 24 May at the Delta site. Plots were 10 m long and seeded at 8.3 seeds per meter in a randomized complete block design. Each plot consisted of one row and was bordered by a single guard row of hybrid 3779 on either side. The Armstrong site had six replications and the Delta site had five replications because of salt damage and excessive weed growth in one replication.

In another test, 15 hybrids with a range of susceptibilities to Fusarium ear rot were selected from the 1988 University of California Cooperative Extension Field Screening for Fusarium ear rot (2). The hybrids were planted in randomized complete block designs with five replications at both the Armstrong and Delta sites. The four-row plots were 10 m long and seeded at 8.3 seeds per meter. Several plots at the Delta site, including all of the Cargill hybrid 8707 plots, were eliminated from the experiment because of salt damage, thereby changing the plan to a randomized complete block design with three replications.

Pesticide applications. In 1988, the four treatments at the Armstrong site included carbaryl (Sevin 5% dust; Rhone-Poulenc Inc., Research Triangle Park, NC), acephate (Orthene 75S, Chevron Chemical, San Ramon, CA), carbaryl plus acephate, and a nontreated control. At the Delta site, only the acephate and control treatments were included in the experiment. Ears were treated at the green silk stage (silks newly emerged, 3–5 cm long), which occurred on 24 July at Armstrong and 25 July at the Delta site. Sevin was applied as a light dust, directly from the can, onto the silks. Acephate was applied to the silks at 0.8 g a.i./L with a CO_2 backpack sprayer to runoff. For the combination treatment of carbaryl and acephate, the acephate solution was applied to runoff followed by a light dust of carbaryl on the silks. Only the primary ears were treated.

In 1989, treatments at the Armstrong and Delta sites were identical. The seven treatments were: one application of acephate

(0.8 g a.i./L); two applications of acephate; one application of benomyl (0.6 g a.i./L of Benlate 50WP; E. I. du Pont de Nemours and Co., Wilmington, DE); two applications of benomyl; one application of acephate plus benomyl; two applications of acephate plus benomyl; and a nontreated control. For the combination treatments, acephate and benomyl were mixed and applied to the silks until runoff with a 7.5-L handpump garden sprayer. Ears were treated at the green silk stage (24 July at Armstrong and 31 July at the Delta site) and then again 10 days later (3 August at Armstrong and 10 August at the Delta site) if treated twice. Only the primary ears were treated.

Silk data. Companies providing seed for the 15 hybrids in the experiment were requested to provide heat units to silking for their respective hybrids (3). At pretassel, 10 random plants of each plot at the Armstrong and Delta sites were flagged, checked daily, and recorded as no silk, green silk, yellow-brown silk, or brown silk stages. At the brown silk stage, daily recording ended. Data were converted to days from initial green silk to yellow-brown silk and to brown silk stages.

Thrips enumeration. In 1988, three ears per plot were randomly harvested at the brown silk stage and pooled. In the laboratory, ears were cut into thirds, then submerged and agitated in 1.5–2 L of soapy (Ivory Liquid, Procter and Gamble, Cincinnati OH) water for ~1 min. After ear pieces were removed, the water was filtered through Miracloth (Calbiochem, LaJolla, CA) under vacuum. The Miracloth and filtrate were transferred to a petri plate that was sealed in parafilm, frozen for 1 day at −10 C to immobilize the thrips, and then thrips on the Miracloth were counted under a dissecting scope at ×7. This procedure is hereafter referred to as the wash method. Thrips were enumerated by the wash method at both locations in 1988 and only in the pesticide plot at the Armstrong site in 1989.

In 1989, a faster method was developed for enumerating thrips. In the insecticide trial, two ears per plot were randomly harvested at the brown silk stage. In the hybrid trial, 10 ears were randomly collected at the green, yellow-brown, and brown silk stages from the three unflagged rows. A longitudinal cut though the husks was made from the butt end to the silk channel opening. A lateral cut through the husks was also made halfway around the base of the ear, thereby forming a T-shape. Husks then were separated en masse from the ear. Any thrips that would have had immediate contact with the kernels in the closed ear (including thrips on

TABLE 1. Effects of insecticide treatments on thrips populations and Fusarium ear rot in two corn hybrids (Pioneer 3779 and Pioneer 3295) in 1988

| | Armstrong[w] | | | | Delta[w] | | | |
| | 3779 | | 3295 | | 3779 | | 3295 | |
Treatment[x]	Number of thrips[y]	Percentage of healthy ears[z]	Number of thrips	Percentage of healthy ears	Number of thrips	Percentage of healthy ears	Number of thrips	Percentage of healthy ears
Control	68.3	4.0	33.3	76.7	27.7 a	0.8 a	6.3 a	58.5 a
Carbaryl	36.5	31.0	9.5	91.8	...	...	...	...
Acephate	30.0	50.2	9.7	97.7	12.3 a	81.8 b	5.8 a	94.5 b
Carbaryl + acephate	12.8	43.3	6.7	99.3	...	....	...	...
Sums of squares								
Contrast								
Control vs insecticide	7,887.7*	6,328.1*	2,738.0*	1,687.8*				
Carbaryl vs acephate and combination	1,685.1	453.9	23.5	168.7				
Acephate vs carbaryl and combination	887.5	142.8	27.0	7.7				

[w] The Armstrong site was analyzed by ANOVA and contrast analysis, and the Delta site was analyzed by ANOVA. Values from the Delta site followed by different letters in each column are different at $P \leq 0.05$. Sums of squares from the Armstrong site followed by * are significantly different at $P \leq 0.05$.

[x] Insecticide applications made on silks at green silk stage.

[y] Values represent the number of thrips per three ears and are the means of six replications. Intra-ear populations of thrips were enumerated at brown silk stage.

[z] Percentage of healthy ears (free of ear rot symptoms) in about 20 ears in each of six replications. Ears rated at maturity.

the husks in contact with the kernels, on kernels, and on the silks enclosed by husks) were counted and a rating was assigned. The rating scale was: 0 = no thrips; 1 = 1–5 thrips; 2 = 6–20 thrips; and 3 = >20 thrips. Thrips between the husk layers and in exposed silks were not included in the rating.

Thrips also were captured on the corn plant (excluding ears) and plated on Nash-Snyder media (11). After 7 days, plates were examined for colonies of *F. moniliforme*.

Fluorescein evaluation. At yellow-brown silk and brown silk stages, three ears were harvested randomly from each plot, bagged, and returned to the lab. Ears were dunked, silk end up, in a 18-L bucket of water containing 1 g of fluorescein (fluorescein sodium salt; Sigma Chemical Co., St Louis, MO) for 30 s. After the outer husks were blotted dry with paper towels, the husks were removed, and the ear was examined under long-wave UV light for fluorescence. Ears were scored as positive or negative for fluorescence.

Disease evaluation. At late dent (plants entirely senescent), husks were peeled back and ears were rated for disease. A 0–6 and a 0–5 scale were used to rate disease in 1988 and 1989, respectively. Data were converted to disease incidence (any disease rating greater than 0) for ease of data manipulation. In both years, ear rot associated with earworm damage was disregarded during rating.

Pesticide inhibition of *F. moniliforme* in vitro. Acephate, benomyl, and carbaryl were screened for activity against *F. moniliforme* in the laboratory. Potato-dextrose agar (PDA) plugs (4 mm diameter) of two different isolates of *F. moniliforme* initiated from single conidia (originally isolated from corn ears from commercial fields near the towns of Perkins and Maxwell in Sacramento and Colusa counties, CA, respectively) were placed on Puhalla's minimal media for *Fusarium* (14) amended with 0, 20, 200, or 2,000 μg a.i. acephate or carbaryl per milliliter or 0, 0.2, 2, or 20 μg a.i. benomyl per milliliter. Colony diameter was measured daily for 6 days. Each experiment consisted of five replications of each isolate at each concentration of chemical. Experiments were repeated twice.

The effect of acephate on germination of conidia also was tested on Puhalla's minimal media amended with 0, 20, 200, or 2,000 μg a.i. acephate per milliliter by dilution plating. Twenty-day-old cultures of an isolate of *F. moniliforme* (from corn near Firebaugh in Fresno Co., CA) growing on PDA were flooded with sterile water and scraped with a glass rod. After the conidial suspension was filtered through three layers of cheesecloth to remove mycelial fragments, the concentration of conidia was determined with a hemacytometer and diluted to 2.4×10^3 spores per milliliter. Two serial, 1:11 dilutions of conidial suspension and sterile distilled water were made from this stock solution. The stock solution and serial dilutions were each added to plates amended with 0, 20, 200, and 2,000 μg a.i. acephate per milliliter in three replicates. The number of colonies per plate was determined after 3 days.

Data from all experiments were analyzed in MSTAT (Michigan Statistics Group, Michigan State University, East Lansing, MI) by analysis of variance, regression, and when appropriate, subjected to contrast analysis for group comparisons.

RESULTS

Pesticide evaluation. The acephate, carbaryl, and acephate plus carbaryl treatments significantly ($P \leq 0.05$) reduced the number of thrips in both Pioneer hybrids 3295 and 3779 at the Armstrong site in 1988 (Table 1). Acephate did not significantly reduce thrips populations at the Delta site. Thrips populations at both locations were lower on the nontreated ears of hybrid 3295 than on the nontreated ears of hybrid 3779. The insecticide treatments applied to both hybrids at both locations significantly ($P \leq 0.05$) reduced the incidence of ear rot compared to the nontreated ears. There were no significant differences in the percentage of healthy ears among the acephate, carbaryl, and acephate plus carbaryl treatments in either hybrid at the Armstrong site. The acephate treatment significantly ($P \leq 0.05$) reduced the incidence of ear

rot compared to the nontreated control in both hybrids at the Delta site.

In 1989, acephate but not benomyl significantly reduced the intra-ear populations of thrips compared to the nontreated ears at both locations (Table 2). Two applications of acephate provided greater control of thrips than one application of acephate at the Armstrong site but not at the Delta site. Acephate significantly reduced ear rot at both locations in 1989. Two applications of acephate were more efficacious than one application in reducing the incidence of disease. Benomyl did not reduce the incidence of ear rot. Two percent of thrips captured outside of corn ears were contaminated with *F. moniliforme*.

Hybrid trial. Silk aging characteristics were not correlated with ear rot. Susceptibility to Fusarium ear rot was not correlated with days from initial green silk to yellow-brown silk stages ($r = 0.15$ at Armstrong and -0.23 at the Delta site) nor with days from initial green silk to brown silk stages ($r = -0.04$ at Armstrong and 0.18 at the Delta site). There was also no relationship between heat units to silking ($r = 0.29$) and hybrid susceptibility to ear rot.

Hybrids were grouped by susceptibility to ear rot (i.e., susceptible, intermediate, and resistant) (2) and subjected to contrast analysis based on populations of thrips and percentage of fluorescent ears (Table 3). Both thrips rating and percentage of fluorescent ears were measured at yellow-brown and brown silk stages. Data from the brown silk stage provided better correlations with disease susceptibility than did data from the yellow-brown silk stage. At the Armstrong site, populations of thrips inside ears of the susceptible hybrids were greater ($P \leq 0.05$) than populations of thrips in the intermediate and tolerant hybrids (Table 3). Populations of thrips in the intermediate hybrids were significantly greater ($P \leq 0.05$) than the populations in the resistant hybrids. At the Delta site, populations of thrips were also significantly greater ($P \leq 0.05$) in the ears of the susceptible hybrids than the populations of thrips in the intermediate and resistant hybrids. Populations of thrips in the ears were positively correlated with disease incidence (i.e., ears with a disease rating >0 at both locations) (Fig. 1). Thrips populations and disease incidence in

TABLE 2. Effects of insecticide and fungicide treatments on intra-ear populations of thrips and Fusarium ear rot of corn in 1989

	Armstrong		Delta	
Treatment[w]	Thrips index[x]	Percentage of healthy ears[y]	Thrips index	Percentage of healthy ears
Control	2.2	3.8	0.9	34.6
Acephate	1.6	45.2	0.5	61.6
2× Acephate	0.5	68.6	0.5	83.2
Benomyl	1.6	2.5	1.0	28.2
2× Benomyl	2.4	4.4	1.2	28.8
Acephate + benomyl	1.5	46.6	0.8	63.0
2× Acephate + benomyl	0.5	83.7	0.3	87.2
Sums of squares				
Contrast				
Acephate vs control	10.58*[z]	11,313.9*	1.28*	4,762.8*
Benomyl vs control	0.32	0.3	0.32	124.0
Acephate vs combination	0.03	401.8	0.03	36.4
Benomyl vs combination	12.00*	22,810.6*	3.63*	10,857.8*
1× vs 2× Acephate	13.23*	5,502.4*	0.75	2,622.1*
1× vs 2× Benomyl	0.12	2,279.5*	0.27	768.8

[w] Applications made at green silk stage and again 10 days later if 2×. The hybrid was Pioneer hybrid 3779.

[x] Thrips index: 0 = no thrips, 1 = 1–5, 2 = 6–20, and 3 = >20 thrips per ear.

[y] Values represent the means of six replications at the Armstrong site and five replications at the Delta site. About 20 ears were visually assessed for the presence of ear rot in each replication.

[z] Numbers followed by * are different at $P \leq 0.05$.

TABLE 3. Relationships among Fusarium ear rot susceptibility, intra-ear populations of thrips, and husk looseness as measured by submerging ears collected at the brown silk stage in fluorescent dye

Hybrid	Susceptibility rating[w]	Armstrong[u]		Delta[v]	
		Thrips index[x]	Percentage of fluorescent ears	Thrips index	Percentage of fluorescent ears
Pioneer 3540	S	2.44	93.2	0.70	66.3
Pioneer 3569	S	0.74	73.2	0.50	0.0
Pioneer 3603	S	0.98	93.2	0.12	100.0
Pioneer 3779	S	1.62	100.0	1.10	100.0
Asgrow RX749	I	0.20	19.8	0.0	0.0
Cargill 8707	I	0.72	59.6	. . .[y]	. . .
Pioneer 3295	I	0.44	86.4	0.03	0.0
Pioneer 3733	I	0.20	73.8	0.15	86.7
Asgrow RX807	T	0.30	46.6	0.0	0.0
Asgrow XP9877	T	0.10	13.2	0.0	0.0
NK S4590	T	0.22	46.4	0.13	0.0
Payco 648	T	0.56	59.6	0.33	66.3
Pioneer 3343	T	0.48	52.8	0.34	11.0
Pioneer 3377	T	0.12	6.6	0.09	0.0
Pioneer 3267	T	0.10	19.8	0.0	11.0
Sums of squares					
Contrast					
S vs T		145.8*[z]	38,374.1*	8.2*	22,237.9*
S vs I		119.1*	9,156.7*	10.2*	7,307.0*
T vs I		1.4*	7,733.4*	0.2	1,667.8*

[u] Values from the Armstrong site are the means of five replications.
[v] Values from the Delta site are the means of three replications.
[w] Thrips index: 0 = no thrips, 1 = 1–5, 2 = 6–20, and 3 = >20 thrips per ear.
[x] S = susceptible, I = intermediate, T = tolerant (2).
[y] Hybrid Cargill 8707 was eliminated from the Delta site because of salt damage and excessive weed growth in all replications.
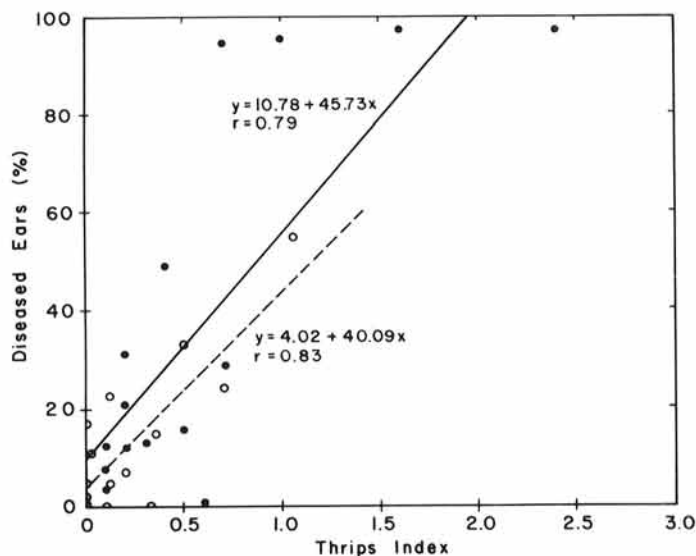[z] Sums of squares followed by an * are significant at $P \leq 0.05$.

Fig. 1. Relationship between intra-ear populations of thrips (population rated at the brown silk stage on a 0–3 scale in which 0 = no thrips; 1 = 1–5; 2 = 6–20; and 3 = >20 thrips per ear) and Fusarium ear rot of corn. Points represent individual hybrids at the Armstrong site (●——●) and the Delta site (○- - -○). Each point represents the mean of six replications at Armstrong and five replications at the Delta site. Both lines are significant at $P \leq 0.05$.

the susceptible hybrids were lower in the Delta site than at Armstrong.

At both locations, the percentage of fluorescent ears was significantly greater ($P \leq 0.05$) in the susceptible hybrids than in the intermediate and resistant hybrids, and the percentage of fluorescent ears was significantly greater ($P \leq 0.05$) in the intermediate hybrids than in the resistant hybrids (Table 3). The percentage of fluorescent ears at the brown silk stage was positively correlated with disease incidence (i.e., ears with a disease rating >0) (Fig. 2), and positively correlated with intra-ear populations of thrips (Fig. 3).

Pesticide inhibition of F. moniliforme in vitro. Growth of F. moniliforme was slightly inhibited (12.3%) by 2,000 μg a.i. acephate per milliliter but was not inhibited by acephate at concentrations less than 2,000 μg/ml (data not presented). Germination of F. moniliforme was not significantly reduced at up to 2,000 μg a.i. acephate per milliliter. Carbaryl had no significant effect on colony growth at up to 200 μg a.i. per milliliter. Colony growth was inhibited at 20 μg a.i. benomyl per milliliter but not at 0.2 and 2 μg a.i. benomyl per milliliter.

DISCUSSION

Applications of acephate and carbaryl at the green silk stage reduced the incidence of Fusarium ear rot of corn. Presumably, insects were controlled by these insecticides, because neither chemical was effective as a fungicide. The most prevalent insects in corn ears were western flower thrips and corn earworm larvae (Heliothis zea Boddie). Other insects present in corn ears at the dough kernel stage and later were the minute pirate bug (Orius tristicolor White), a beetle species in the family Coccinellidae, and several beetle species in the family Nitidulidae. These insects were not as numerous or as widespread in corn ears as thrips and the minute pirate bug is a predator, not a herbivore. Only thrips were common in ears during the yellow-brown silk and milk kernel stages. Windels et al (20) have shown that picnic beetles (family Nitidulidae) are carriers of F. moniliforme in Minnesota cornfields and have suggested that the beetles act as vectors of the fungus by carrying it from buried corn debris to healthy ears. Nitidulid beetles were not numerous in our field areas and occurred after ear rot symptoms first appeared.

Thrips feed by rasping and sucking, thereby wounding the pericarp of young kernels. This wounding may provide an ideal point of entry for the fungus. While the mode of kernel infection by F. moniliforme is unclear, it may be similar to Aspergillus ear rot. In the latter disease, Aspergillus flavus grows down the silks and onto the surface of the kernel and the minor floral
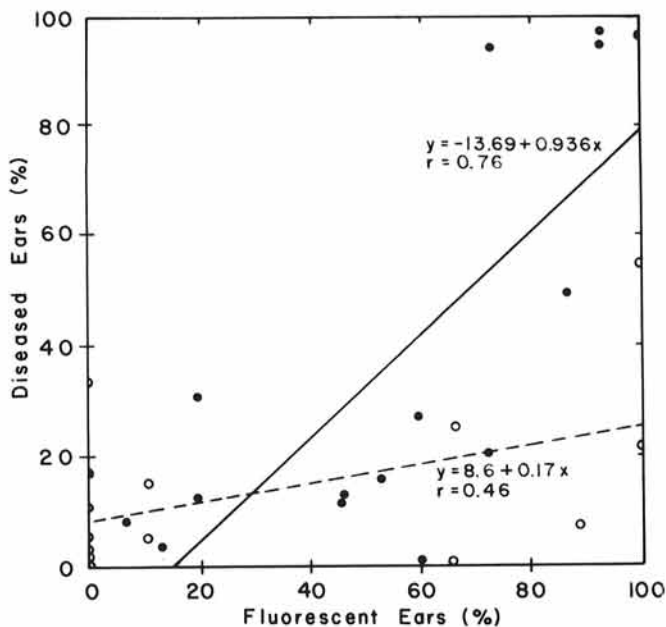
Fig. 2. Relationship between the percentage of fluorescent ears at brown silk stage (used as a measure of husk looseness) and Fusarium ear rot of corn. Fluorescence was determined by dunking an ear into 18 L of water containing 1 g of fluorescein, removing the husks, and examining the kernels under UV light. Points represent individual hybrids at the Armstrong site (●——●) and the Delta site (○- - -○). Each point represents the mean of six replications at Armstrong and five replications at the Delta site. Both lines are significant at $P \leq 0.05$.

Fig. 3. Relationship between the percentage of fluorescent ears at brown silk stage (used as a measure of husk looseness) and intra-ear populations of thrips (population rated at brown silk stage on a 0–3 scale in which 0 = no thrips; 1 = 1–5; 2 = 6–20; and 3 = >20 thrips per ear). Fluorescence was determined by dunking an ear into 18 L of water containing 1 g of fluorescein, removing the husks, and examining the kernels under UV light. Points represent individual hybrids at the Armstrong site (●——●) and the Delta site (○- - -○). Each point represents the mean of six replications at Armstrong and five replications at the Delta site. Both lines are significant at $P \leq 0.05$.

parts (8,9,13). Kernel wounding by insects provides an obvious mode of entry because *A. flavus* is already present on the surface of the kernel.

In many ways, Fusarium ear rot is analogous to Aspergillus ear rot in the southeastern United States. *A. flavus* and *F. moniliforme* have been described as weakly pathogenic, have similar modes of entry into the corn ear (8,9), and are found in asymptomatic kernels (13). Mites, weevils, corn earworms, and corn borers are vectors of *A. flavus* in corn in the southeastern United States (1,10). The two-spotted mite was tested as a vector for *F. moniliforme* in California by Davis et al (2). The incidence of ear rot in treatments using mites and mites dusted with *F. moniliforme* spores was not significantly different from the control treatment. It was therefore concluded that mites are not effective vectors of Fusarium in corn. In the same study, high populations of western flower thrips were discovered inside the nontreated corn ears. Thrips were the intended target of the acephate treatment in this study, and earworms were the intended target of the carbaryl treatment in 1988. Diseased kernels adjacent to earworm damage were disregarded when rating for disease because the correlation between earworms and an increase in ear rot has been documented (16). Carbaryl obviously had an effect on thrips populations that was not intended in the original experimental design and was therefore eliminated from the 1989 experiments. The benomyl treatment was added to the 1989 studies to test the hypothesis that inhibition of *F. moniliforme* spores on exposed silks had no effect on ear rot, which was confirmed here.

Fusarium ear rot of corn was correlated with the population of thrips in corn ears and the looseness of the husks at the brown silk stage. The percentage of fluorescent ears also was correlated with intra-ear populations of thrips. These correlations suggest that the physical exclusion of thrips from the developing ear is a major factor in reducing disease levels and that some action of the thrips is contributing to ear rot. Thrips may be acting as vectors of *F. moniliforme* or as wounding agents by feeding on plant tissue. Thrips may be carrying spores into the developing ear or may be distributing the fungus already present in the ear. R. M. Davis (*unpublished*) found 40% of thrips within corn ears contaminated with *F. moniliforme* in 1987. In contrast, only 2%
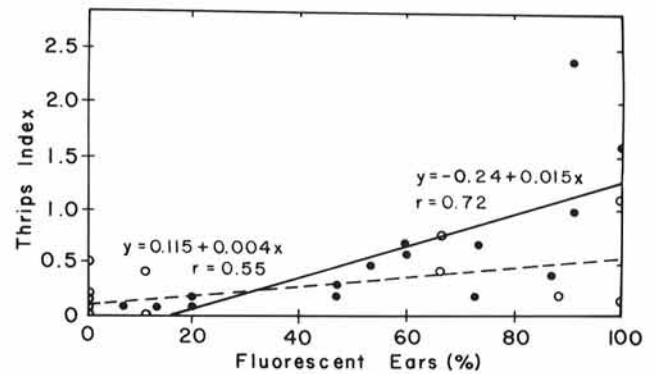
of thrips outside of corn ears in 1989 were contaminated with *F. moniliforme*.

To establish cause and effect relationships among thrips populations, husk looseness, and disease incidence, more work needs to be done under controlled conditions, because thrips and *F. moniliforme* are ubiquitous in California cornfields. Moreover, the correlations among thrips, loose husks, and disease incidence do not account for all of the variability in disease incidence. Other factors such a pericarp morphology, kernel carbohydrates, plant stress, or other resistance factors may play an important role in the disease process.

The correlations among thrips populations in ears, husk looseness, and disease incidence suggest that husk tightness is an important trait in breeding for resistance to ear rot. Historically, corn breeders have had no selectable traits for Fusarium ear rot resistance. Currently, breeders plant corn hybrids in areas with a known high incidence of Fusarium ear rot (2). Hybrids then are rated for ear rot at maturity and a determination of relative resistance is made at that time. Selection for tight husk covering may eliminate some of the trial and error of Fusarium ear rot resistance breeding attempts.

LITERATURE CITED

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