

#### Severe ear rot of corn on lower ear at left was caused by Fusarium moniliforme, a fungus transmitted by western flower thrips (below). Thrips, which enter the ear and wound kernels as they feed, were excluded from the healthy ear at left.



# Fusarium ear rot of corn

R. Michael Davis 🗅 Franz R. Kegel 🗅 Wynette M. Sills 🗅 James J. Farrar

Yearly screenings of field corn cultivars by UC Cooperative Extension help plant breeders and growers in hybrid selection for tolerance to Fusarium ear rot. Researchers have found that thrips feeding carries the fungus into the corn ear.

Fusarium ear rot is the most damaging disease of corn in the Sacramento Valley. Infected kernels are characterized by a white to pinkish decay and may be completely consumed by the fungus. In addition to decreasing yields, ear rot affects grain quality, limits the use of certain cultivars, and causes concern about toxins in corn used for feed. The disease occurs in varying degrees of severity throughout the United States.

Losses to ear rot, which is caused by the fungus Fusarium moniliforme, are kept to a minimum through the use of tolerant corn hybrids. Since mechanisms of resistance are not known and no corn line is immune, the selection of suitable hybrids is largely a matter of screening for resistance in the presence of the disease. To aid seed companies in this process and to help growers select hybrids with acceptable tolerance, the University of California Cooperative Extension annually screens experimental and

differences between susceptible and resistant corn lines. Descriptions of the infection process and the identification of traits that confer resistance may result in the development of superior hybrids and more efficient and rapid screening techniques for disease resistance.

commercial hybrids at two northern Cali-

fornia locations. The program was begun in

1970 by UC Davis Extension specialists with

To further help corn breeders incorporate

resistant traits into new hybrids, a research

program has been established to identify

plant factors that contribute to tolerance to

ear rot. Although the ability of F. monili-

forme to cause disease in corn is well known,

many fundamental aspects of the disease

are not clearly understood. Investigations

are being conducted to explain the mode of

entry of the fungus into corn ears, deter-

mine when infection occurs, and identify

the cooperation of several farm advisors.

# Ear rot resistance screening

Each year, corn breeders are invited to submit seed of commercial and experimental field corn cultivars. (To date, we have not studied sweet corn or popcorn, although both are susceptible to the disease.) Each entry is planted in four replications in a randomized complete block design at two

locations. Each replication consists of 20 feet of two single-row beds. All the ears in one bed per replication, except for those on a few plants on each end of the plots, ears with corn earworm (Heliothis zea) damage, and ears with visible smut are rated for severity of Fusarium kernel infection at the end of the season.

One trial is on peat soils in the Delta region of San Joaquin County and the other is on loam soils on the UC Davis campus. Because disease pressure is high at both locations, our program allows rigorous field screening for ear rot tolerance without artificial inoculations. To our knowledge, these ideal conditions do not occur consistently anywhere else in the United States.

Severity of ear rot is rated as follows: 0 =none of the kernels consumed, broken, or cracked by F. moniliforme infection; 1 = 1% to 10% of the kernels affected, a light infection generally confined to the tip of the cob; 2 = 10% to 40% of the kernels affected, a moderate infection; and 3 = over 40% of the kernels affected, a severe infection. All ears are also evaluated for light-colored streaks in kernels, a symptom of late or arrested Fusarium infection.

Based on the percentage of ears in these categories and the relative performance of two susceptible and two tolerant controls, each entry receives a final rating that describes its susceptibility to ear rot. The following scale is used to summarize the disease rating: highly tolerant = more than 80% of the ears in category 0 and 100% in categories 0 and 1; tolerant = more than 60%in category 0 and not more than 5% in categories 2 and 3; intermediate = ratings between tolerant and susceptible; susceptible = more than 20% in categories 2 and 3; and highly susceptible = more than 30% in category 3. Ears with earworm damage are not included in the evaluation because such damage is almost always accompanied by Fusarium infection. Ears with smut infection are also apparently more susceptible to ear rot and are excluded.

The program evaluates an average of 90 entries for ear rot tolerance each year. From 1970 to 1988, average ratings of the entries were: highly tolerant, 7%; tolerant, 29%; intermediate, 39%; susceptible, 22%; and highly susceptible, 3%. Entries are often submitted more than once to confirm results. The relatively low number of highly tolerant hybrids emphasizes the importance of field screening to avoid serious losses with susceptible hybrids.

## Ear rot research

Our initial experiments were designed to discover the mode of entry of *Fusarium* into corn ears and the growth stage of peak susceptibility. These objectives were accomplished by intensive sampling (at least weekly) from various parts of the corn stalk, ear shank and cob, husk, silk, and kernels. The fungus was apparently present throughout the cornfields soon after planting and was recovered from leaf-blade surfaces, leaf axes, and the air.

Although the fungus was recovered from leaf nodes, it did not infect the corn plant systemically and enter the kernels through the cob. Instead, the kernels were attacked



effective plug, blocking entry to spores or insects into the tip of the ear. Once the silk is pollinated and begins to dry, however, the tip of the ear is vulnerable. In the first year of the study, we found that inoculating the silk with a suspension of spores did not result in ear rot, but injecting the spores through the silk channel opening did. At the same time, we learned that the kernels are most susceptible at about the blister stage of development, the period when they are filling rapidly but are still tender. We assumed that an insect might carry the spores of the fungus through the silk channel opening while the kernels were

primarily though the silk channel opening

of the ear. We based this conclusion on iso-

lations of the fungus from distal parts of the

ear before it could be found at the stem end.

Before pollination, the silk is apparently an

still very young. In the first experiment testing the vector hypothesis, we reared two-spotted spider mites (Tetranychus urticae) on cotton seedlings in a greenhouse, sprayed silks with spores of *F. moniliforme*, and released the mites on the silks while the kernels were very susceptible. No increase in ear rot occurred in the treatments with the mites. In that same experiment, however, we observed heavy infestations of western flower thrips (Frankliniella occidentalis) inside very young ears and readily recovered the fungus from the bodies of the thrips. We made many more similar observations of thrips in developing ears and set up a second experiment designed to exclude thrips during the period of peak disease susceptibility.

In experiments in the Delta and at UC Davis, two sprays of acephate (Orthene 75S) were applied to the silks a week apart, beginning at early silking (the silks were about 1 to 2 inches long). The experiments had a randomized complete block design with six replicated 25-foot plots.



Fig. 1. Insecticide test treatment (acephate 0.8 g a.i./L, applied to ears at green silk stage) that excluded thrips from a highly susceptible field corn hybrid nearly eliminated ear rot, confirming that the insect transmits the disease.



The results were dramatic. Ear rot was significantly reduced when acephate was used. Although very susceptible corn hybrids were used in these experiments, ear rot was almost completely eliminated (fig. 1). In 1 year, yields were increased about 25% in a susceptible hybrid (fig. 2). Such yield losses are not to be expected from commercial hybrids that have tolerance to ear rot. The use of acephate on corn is not registered and may not be economically justifiable, but these results provide insight into the mode of entry of *Fusarium* into ears and the importance of wounds in disease development.

### **Future research**

Research is now directed towards comparing ear and kernel characteristics in a number of corn hybrids with different tolerances to *Fusarium* to determine specific mechanisms involved in ear rot resistance. The relationship between resistance and traits that consistently appear in tolerant corn lines will then be studied in more detail.

Resistance may lie in the silk channel opening, which may prevent entrance of the fungus into the ear, or in physical or nutritional factors that restrict establishment and spread of the fungus within the ear. The following hypotheses, based on information currently available, are under study: (1) the silk channel opening prevents entrance of the fungus and / or insects in tolerant hybrids at least during some critical period; (2) the seed coat or other areas of the kernels in tolerant hybrids are more resistant to infection during peak susceptibility; (3) kernels mature faster in resistant hybrids, and so the period of optimum susceptibility is shorter; and (4) some growth factor, perhaps leakage of nutrients from young kernels, supports greater growth of the fungus in susceptible hybrids.

Our research so far indicates a trend towards a longer period of kernel susceptibility in susceptible than in tolerant hybrids, based on increased kernel-to-kernel spread of *F. moniliforme*. We have also demonstrated the importance of wounds in disease development. These hypotheses will continue to be revised as we learn more about the infection process and disease development. With this new information, we will be able to focus on more specific mechanisms of resistance to ear rot.

R. Michael Davis is Extension Plant Pathologist, Department of Plant Pathology, University of California, Davis; Franz R. Kegel is Farm Advisor, San Joaquin County; Wynette M. Sills is former Farm Advisor, Sacramento County; and James J. Farrar is graduate student, Department of Plant Pathology, University of California, Davis. Research was funded by a grant from Pioneer Hi-Bred International, Inc. The authors thank Trisha Peters for technical assistance.