hybrids in both years. The sheath technique is fairly time-consuming and more damaging to the plant than either the silk spray or whorl methods.

Based on these results, the silk spray technique has been selected as the best technique for screening for resistance to Diplodia ear rot. This technique results in reasonable levels of infection, may be more similar to natural infection (8), is less damaging to the plant, can identify significant differences between inbred lines, and is reasonably easy to apply.

Results from this study support earlier observations that inbreds MBS613 and H111 may exhibit some resistance to Diplodia ear rot, whereas inbred B73Ht is more susceptible. The susceptibility of the elite inbred B73Ht supports the suggestion that susceptible germ plasm may be an important factor in the increased occurrence of Diplodia ear rot (4).

Little or no significant differences were observed between hybrids with any of the inoculation techniques evaluated. This may be attributable to a lack of resistance in the hybrids selected; evaluation of a larger number of hybrids might have shown differences. Although there were no significant differences detected, Pioneer 3192 had the lowest disease incidence and ratings in both years for the whorl and silk spray techniques, which supports earlier observations (K. Byrnes, *personal communication*).

Results indicate that the performance of the inbred lines was related to the method by which the plants were inoculated, resulting in a significant line by treatment interaction. This interaction of genotype and inoculation technique should be considered when choosing an inoculation method and when comparing results from different studies.

Finally, a better understanding of how the ear becomes infected, the time of infection, the role of insects in spreading the disease, and identification of traits that may contribute to disease resistance will be necessary in choosing a technique that most accurately measures plant resistance to *S. maydis*.

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**LITERATURE CITED**

1. Chambers, K. R. 1988. Effect of time of inoculation on Diplodia stalk and ear rot of maize in South Africa. Plant Dis. 72:529-531.
2. Cochran, W. G., and Cox, G. M. 1957. Factorial experiments with main effects confounded: Split-plot designs. Pages 297-299 in: Experimental Designs. 2nd ed. John Wiley & Sons, New York.
3. Kang, M. S., Pappelis, A. J., Mumford, P., Murphy, J. A., and BeMiller, J. N. 1974. Effect of cob and shank inoculations (*Diplodia maydis*) on cell death in stalk internodes of corn. Plant Dis. Rep. 58:1113-1117.
4. Latterell, F. M., and Rossi, A. E. 1983. *Stenocarpella macrospora* (= *Diplodia macrospora*) and *S. maydis* (= *D. maydis*) compared as pathogens of corn. Plant Dis. 67:725-729.
5. Pappelis, A. J., Mayama, S., Mayama, M., BeMiller, J. M., Murphy, J. A., Mumford, P., Pappelis, G. A., and Kang, M. S. 1973. Parenchyma cell death and *Diplodia maydis* susceptibility in stalks and ears of corn. Plant Dis. Rep. 57:308-310.
6. Ritchie, S. W., and Hanway, J. J. 1984. How a corn plant develops. Iowa State Univ. Coop. Ext. Serv. Spec. Rep. 48. 21 pp.
7. Smith, G. M., and Trost, J. F. 1934. Diplodia ear rot in inbred and hybrid strains of sweet corn. Phytopathology 24:151-157.
8. Ullstrup, A. J. 1949. A method for producing artificial epidemics of Diplodia ear rot. Phytopathology 39:93-101.
9. Ullstrup, A. J. 1970. Methods for inoculating corn ears with *Gibberella zeae* and *Diplodia maydis*. Plant Dis. Rep. 54:658-662.
10. Villena, W. L. 1969. Studies of inoculation methods and inheritance of resistance to Diplodia ear rot in corn. Ph.D. dissertation. North Carolina State University, Raleigh. 134 pp.
11. Warren, H. L. 1982. Registration of H110 and H111 maize germplasm. Crop Sci. 22:1270-1271.
12. Warren, H. L., and Onken, S. K. 1981. A new technique for evaluating ear rot resistance. (Abstr.) Phytopathology 71:911.

# Cultural Studies and Pathogenicity of *Pseudocercospora fuligena*, the Causal Agent of Black Leaf Mold of Tomato

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**ABSTRACT**

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*Pseudocercospora fuligena* was studied in pure culture and was inoculated on tomato plants under controlled conditions. Germ tubes were observed most frequently from the tip and basal cells of conidia. Free moisture was not necessary for conidia to germinate, and some conidia germinated at 91% relative humidity. Conidia did not germinate at or below 84.5% relative humidity. The fungus grew slowly in culture on four media tested. The optimum temperature for mycelial growth was 26 C, whereas no growth was observed at 34 C. On tomato-oatmeal agar, $6.1 \times 10^4$ conidia per culture dish were produced after 3 wk, but conidia were not produced on potato-dextrose agar. Tomato plants inoculated with $5 \times 10^3$ conidia per milliliter had 96% leaf area infected 14 days after inoculation. In cross-inoculation experiments, isolates from *Solanum nigrum* infected tomato, and isolates from tomato infected *S. nigrum*.

Black leaf mold of tomato, also commonly referred to as Cercospora leaf mold, is caused by the fungus *Pseudocercospora fuligena* (Roldan) Deighton (syn. *Cercospora fuligena* Roldan). The disease and pathogen were first described from the Philippines in 1938 (12). The disease has also been reported to occur in Cambodia, India, Ivory Coast, Japan, Malaysia, Taiwan, Thailand, Solomon Islands, United States (Florida), and Vanuatu (1,3,5,6,8,9,12,15,16). Although the disease and pathogen appear to be widespread, there have been few detailed reports on the disease or on the biology of the pathogen.

The initial symptoms of black leaf mold appear as pale yellow to light green lesions 1–20 mm in diameter. Lesions on the lower leaf surface are initially covered with white mycelium that turns gray to black as the fungus sporulates (10,11, 13,16). Infected leaves wilt, dry with age, and often drop prematurely. Infection of petioles and stems was reported (11,13), but no reports have indicated the occurrence of infection or symptoms on fruits.

Black leaf mold develops under conditions of warm temperatures and high relative humidity. In Japan, the disease was found to be widely distributed and caused severe reductions in yield (16). Symptoms have been reported to occur from 4 to 35 days after inoculation (1,13,16). There are no reports on how the fungus survives in the field, but it was found that conidia could survive up to 6 mo on dried leaves stored in clay pots indoors (16).

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