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Corn Stunt Spiroplasma[1]

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INTRODUCTION: Corn stunt is one of the most economically important diseases of maize in the US, Mexico, and Central and South America (Bradfute *et al.* 1981; Tsai and Falk 1988). Corn stunt was first named by Kunkel (1946). In the succeeding three decades, corn stunt disease was thought to be caused by several strains of an unknown virus based on symptomatology and transmission by leafhopper vectors (Homoptera: Cicadellidae) (Nault and Bradfute 1979). The helical morphology of the causal agent of Rio Grande corn stunt (Maramorosch 1955) was subsequently established (Davis and Worley 1973) and was named corn stunt spiroplasma, *Spiroplasma kunkelii* (Williamson and Whitcomb 1975). A serious outbreak of a disease complex resembling corn stunt in maize occurred in South Florida from 1979-1980. At least five viral and two mollicute plant pathogens were involved in the outbreak. Corn stunt spiroplasma (CSS) was considered one of the two most important components in the disease epidemic (Bradfute *et al.* 1981).

Fig. 1. Corn plant infected with the corn stunt spiroplasma.

SYMPTOMS AND HOST RANGE: The initial symptoms of Rio Grande corn stunt show characteristic small chlorotic stripes that develop at the leaf bases of young plants after about 25-30 days. The chlorotic stripes become fused and extend further toward the leaf tips in the older leaves with green spots and stripes on a chlorotic back-ground (Fig. 1). The infected plants have much shorter internodes and a proliferation of secondary shoots in leaf axils. Reddening on leaves varies depending on the corn genotype and environmental conditions. The plant hosts of CSS are Z. *mays* L., *Z. mays mexicana* (Schrad.) lltis, Z. *diploperennis* Iltis, Doebley and Guzman, Z. *perennis* (Hitchc.) Reeves and Mangelsd., Z. *mars X Tripsacum floridanum* .pn2 Porter ex Vasey L., and Z. *luxurians* (Durieu and Ascherson) Bird (Nault 1980). In addition, *Vicia faba* L., *Catharanthus roseus (L.) G.* Don, *Raphanus sativus* L., *Sinapis alba* L. *(Brassica hirta* Moench) and *Spinacia oleracea* L. are also reported as experimental hosts for CSS (Markham and Alivizatos 1983; Markham *et al.* 1977).

DISEASE AGENT AND VECTOR RELATIONSHIPS: CSS is transmitted naturally by *Dalbulus maidis* (DeLong and Wolcott), and *D. elimatus* (Ball), and experimentally by *Graminella nirifrons* (Forbes), *G. sonora* (Ball), *Stirellus bicolor* (Van Duzee), *Exitianus exitiosus* (Uhler), and *Euscelidius variegatus* (Kirsch.) (Granados 1969; Granados *et al.* 1968; Nault and Knoke 1981). *D. maidis* is the most common and efficient vector of CSS in South Florida (Tsai 1987a, b). CSS is transmitted persistently by *D. maidis.* With an acquisition access period of 15 min and 7 days, 15 and 100% of the tested *D. maidis* transmitted CSS, respectively (Markham and Alivizatos 1983). There is an incubation period in the vector ranging from 17.5 to 21.2 days (Tsai 1987b). The length of incubation period varies inversely with the length of acquisition access period. CSS is retained up to 45 days by *D. maidis* depending on the individual insect tested (Tsai 19876).

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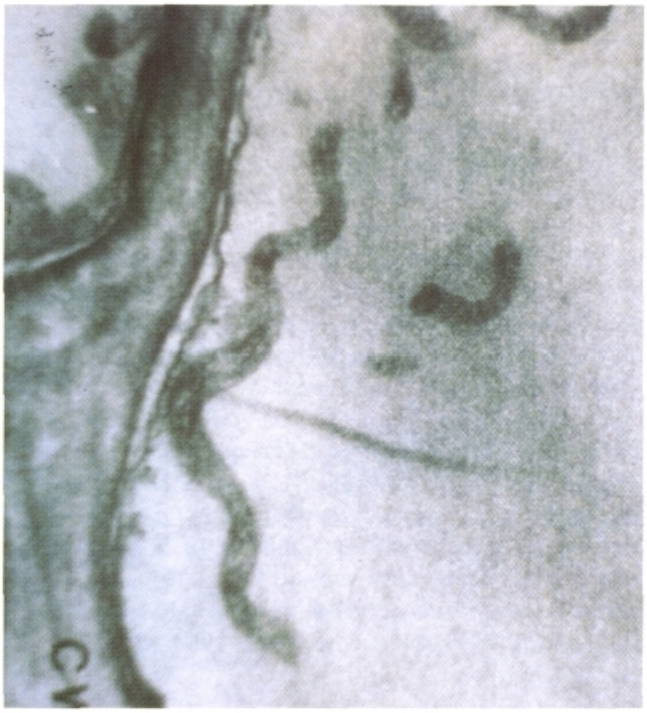
The average development times for nymphal instars I-V range from 11.6 to 33.6 days at 10° C, 6.3 to 13.3 days at 15.6° C, 2.5 to 3.8 days at 26.7° C and 2.4 to 4.4 days at 32.2° C. Both male and female (Fig. 2) longevities are greatest at 15.6° C and lowest at 32.2° C. The average number (x±SD) of eggs per female per day is 3.62±1.09 at 15.6° and 14.18±3.55 at 26.7° C; the average number (x±SD) of eggs per female is 402.33±140.03 at 15.6° C and 611.8±164.9 at 26.7° C. Adult lifespans (x±SD) for mated and unmated females at 15.6 and 26.7° C are 110±14.5 days and I80±26.1 days, respectively at 15.6° C; and 45.5±15.8 days and 112.0±16.5 days, respectively at 26.7° C (Tsai 1987a).

Fig. ***2.*** *Dalbulus maidis. A)* Dorsal view, female. B) Lateral view, female (note ovipositor). C) Lateral view, male. Photography Credit: Jeffrey W. Lotz.

Fig. **3.** Corn stunt spiroplasma in plant cell.



AUSAL AGENT AND DIAGNOSTIC ECHNIQUE: CSS is a motile, helical, cell wall-free prokaryote (Fig. 3) that can readily be seen by phase contrast or dark field microscopy of plant juice or hemolymph and abdominal smears from leafhopper vectors (Davis and Worley 1973). It is a phloem-limited organism. CSS is highly resistant to penicillin in *in vitro* tests (Chang and Chen 1978). Treatment of inoculated plants with tetracycline antibiotic causes remission of symptoms and interferes with leafhopper transmission (Granados 1969). CSS can readily be cultured and maintained *in vitro* (Chen and Liao 1975; Williamson and Whitcomb 1975; Davis *et* al. 1984). Infected corn plants and leafhoppers can be tested for the presence of CSS under the micro-scope or by serological assays using spiroplasma deformation tests and enzyme-linked immunosorbent assay (ELISA). A unique type of cell deformity is found in the stomata of epidermal strips of leaves from the CSS infected plants which can be used as diagnostic feature (Overman *et al.* 1992), with stomata showing rounded guard cells and subsidiary cells fused into adjoining epidermal cells.

SURVEY AND DETECTION: Look for plants that have continuous chlorotic bands of varying width. Some of these symptoms are similar to those caused by nutritional deficiencies or one of the other corn pathogens. Laboratory confirmation is necessary for accurate diagnosis. Adult leafhoppers are usually found singly on leaves or stems; young nymphs may often be found aggregating more on leaves than stems.

CONTROL: Traditional methods of control such as vector control, prompt removal of infected plants. elimination of reservoir hosts such as *Tripsacum* spp. and volunteer corn plants, and breeding for resistance to the insect vector or the pathogen are recommended.

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