Severe Outbreak of Corn Lethal Necrosis Disease in Kansas

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ABSTRACT

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In 1978, corn lethal necrosis disease was epiphytotic in northwestern and north-central Kansas. Assays showed maize chlorotic mottle virus, maize dwarf mosaic virus A and B, and wheat streak mosaic virus, singly or in combinations.

Corn lethal necrosis disease (CLND), a severe disease of corn (*Zea mays* L.), is caused by a synergistic interaction between maize chlorotic mottle virus (MCMV) and either maize dwarf mosaic virus (MDMV) or wheat streak mosaic virus (WSMV) (5). During the 1978 season, surveys were conducted of hybrid corn with CLND in fields surrounding the Kansas communities of Almena (Norton County), Gaylord (Smith County), Alton (Osborne County, and Norway (Republic County) (Fig. 1). Harvest data were not taken from these fields, but losses were estimated at 50% or more based on incidence of CLND. In our hybrid corn-CLND evaluation program, where plants were mechanically inoculated at the 10–12 leaf stage, yields were reduced up to 70% in highly susceptible hybrids (L. E. Claflin, *unpublished*).

MATERIALS AND METHODS

Diseased leaf tissues (leaves with CLND and mild mosaic symptoms) were crushed in buffer (0.01 M phosphate, pH 7.0, containing 0.5% Tween 20 and 1% polyvinylpyrrolidone, mol wt 44,000) at 1:10 and 1:50 dilutions (w/v) and assayed by double immunodiffusion (0.75% Ionagar, 0.85% NaCl, 0.02% NaN_3, in 0.01 M Tris-HCl, pH 7.2) and enzyme-linked immunosorbent assay (ELISA) (2,6), respectively. Antisera against MCMV (prepared to two serotypes), MDMV-A, and WSMV were prepared in our laboratory (J. K. Uyemoto, *unpublished*).

For bioassays, the extract dilutions were combined and inoculated onto Carborundum-dusted seedlings of N28Ht corn, sorghum (*Sorghum vulgare* Pers. 'DeKalb E59+' and 'Asgrow Bugoff'), and wheat (*Triticum aestivum* L. 'Parker'). On corn, single virus infections caused leaf chlorosis and mottle; in contrast, virus combinations of MCMV + MDMV or MCMV + WSMV caused severe chlorosis and plant death. With DeKalb E59+ sorghum, MDMV-A produced a systemic mosaic and MCMV was latent (systemically); this cultivar was immune to MDMV-B and WSMV. Asgrow Bugoff sorghum developed chlorotic mottle and mild red leaf when inoculated with MDMV-A and severe systemic red leaf when inoculated with MDMV-B but was immune to MCMV and WSMV. Wheat developed systemic chlorotic streaks with WSMV and occasional chlorotic spots and, rarely, a faint systemic mottle with MCMV. MDMV did not infect wheat.

RESULTS AND DISCUSSION

Viruses were identified in 168 of 207 field corn samples: MCMV in 116 samples, MCMV + MDMV-A in 7, MCMV + WSMV in 17, all three viruses in 3, MDMV-A alone in 2, and WSMV alone in 23. Also, 13 of 14 sweet corn (*Z. mays* var. *saccharata* [Sturtev.] Bailey) were infected—seven with WSMV, one with MDMV-A, and five with both viruses. In volunteer sorghum plants, two contained MCMV and one, MDMV-A. These findings were for collections made during 7 wk from early July to late August. Initially, the indicator host range consisted of corn, wheat, and DeKalb E59+ sorghum, which would not have shown MDMV-B readily; in later tests, Asgrow Bugoff was included. Relative sensitivity of the assay systems for detecting MCMV showed corn with the highest incidence of positives at 63, followed by ELISA at 54 and double immunodiffusion at 44. Repeated seroassays (in double immunodiffusion) of indicator plants confirmed MCMV transmissions in all 63 samples; at least three infected plants contained a second Kansas-MCMV serotype (Fig. 2).

In several samples where only MCMV was detected (bioassays and seroassays were negative for WSMV and MDMV-A), the corn indicators showed chlorotic mottle and death of plants. Because single virus infection in corn produces relatively mild symptoms, the CLND-like symptom observed suggested concomitant transmission of a second, unidentified virus. We assayed several of the cultures that

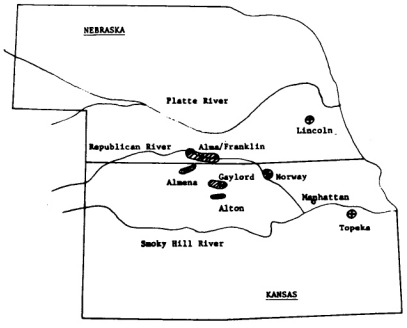

Fig. 1. Distribution map of corn lethal necrosis disease (shaded areas) in Kansas and Nebraska in 1978.

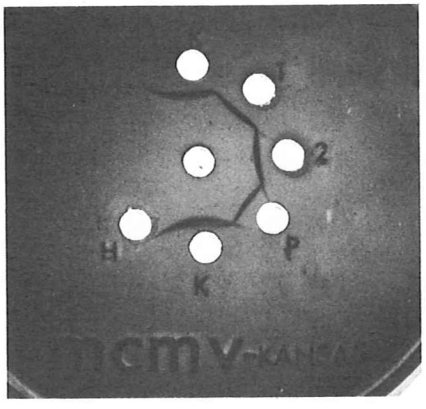

Fig. 2. Results of serological reactions in double immunodiffusion test. Center well contained antiserum prepared to the Kansas strain of maize chlorotic mottle virus (MCMV) (5). Peripheral wells were filled with antigens: K, Kansas strain; 1 and 2, Kansas field isolates, 2 representing a second Kansas serotype; P, Peru strain; H, healthy corn sap.

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behaved in this fashion onto the four indicator hosts; all samples tested induced CLND symptoms in corn and severe red leaf in Asgrow Bugoff sorghum. MDMV-B was confirmed by electron microscopy, which revealed filamentous particles in leaf-dip preparations, and by host range symptomatology (comparisons were made with MDMV-A and MDMV-B cultures supplied by D. T. Gordon, Wooster, OH). Thus, MDMV-B is common in Kansas and has a more significant role in CLND than previously thought (5).

Generally, CLND symptoms are characterized by yellowing and browning of leaves and early death of plants. Around mid-August, however, some corn plants had drying ears (early drying of husk tissues) on otherwise green, healthy-appearing plants. Extracts of husk, shank, silk, and cob pith tissues reacted strongly in MCMV double immunodiffusion tests and were highly infectious on indicator hosts for MDMV-B. These viruses also were detected (by bioassays) in immature kernel, root, and terminal leaf tissues of dry-eared plants. Bioassays of green ears collected in other plantings showed only MDMV-B infections. Thus, double virus infections (MCMV and MDMV-B) were closely associated with early drying of husk tissues, suggesting a causal relationship. The symptoms appeared in the early milk stage; affected ears were fully developed but produced wrinkled, shriveled seeds.

Our results indicate that WSMV and different strains of MCMV and MDMV contributed heavily to the CLND outbreak in Kansas and Nebraska in 1978 (Fig. 1) (3). Except for a reported occurrence in Peru (1), MCMV is currently known only in counties along and near the Kansas-Nebraska border. In 1976, CLND caused moderate losses in Almena (5); in 1977, only trace amounts of CLND were observed in Norton and Republic counties even though MCMV was detected in several plants with mosaic symptoms (D. L. Bockelman, *unpublished*). Apparently, little double infection with WSMV and MDMV occurred in 1977. However, the severity and distribution of CLND observed in 1978, the persistence of MCMV in the same field for three seasons (J. K. Uyemoto, *unpublished*), and the migratory habit of beetle vectors (4) suggest continued spread of MCMV, and hence of CLND, to new areas. Present control recommendations include rotating crops (eg, soybean, sorghum, wheat) and planting CLND-tolerant hybrids.

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