St. Augustinegrass Decline in Arkansas

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ABSTRACT

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St. Augustinegrass plants with viruslike symptoms were observed in a few lawns in central Arkansas in 1981. Assays of affected plants by mechanical inoculation of indicator plants and serologic tests confirmed that the plants were infected with the St. Augustinegrass decline strain of Panicum mosaic virus. This is the first report of this disease in Arkansas.

A few lawns of St. Augustinegrass (*Stenotaphrum secundatum* (Walt.) Kuntze) in central Arkansas showed signs of abnormal yellowing in the spring of 1981. Leaves of many grass plants in four affected lawns had a distinct chlorotic mottle or mosaic pattern similar to that described for the virus-induced St. Augustinegrass decline (SAD) disease (3,4,6). Leaves of some plants were also necrotic, and there were areas of dead and dying turf in three of the lawns. Affected spots of turf varied from 1 to 9 m in diameter, with some spots irregular in shape. In mid-June, sod samples of affected turf from three of the lawns were collected to investigate the cause of the disorder.

MATERIALS AND METHODS

Assay plants that were mechanically inoculated with sap from diseased plants from lawns were German foxtail millet, *Setaria italica* (L.) Beauv.; proso millet, *Panicum miliaceum* L.; pearl millet, *Pennisetum glaucum* (L.) R. Br. 'Tifleaf 1'; crabgrass, *Digitaria sanguinalis* L.; Johnsongrass, *Sorghum halepense* (L.) Pers.; sorghum, *Sorghum bicolor* (L.) Moench; corn, *Zea mays* L. 'Hy × C103'; and St. Augustinegrass. All test plants except St. Augustinegrass were started from seed in steam-sterilized soil in 7.5-cm peat pots. St. Augustinegrass test plants were from stolons of healthy-appearing grass obtained from another area. These plants were determined as being virus-free by serologic testing.

Leaves from plants from three affected St. Augustinegrass lawns were thoroughly ground in 0.05 M phosphate buffer (pH 7.0), a small amount of 600-mesh silicon carbide abrasive was added to the extracts, and each extract was rubbed with cheesecloth pads onto 10 of each of the seeded indicator plants in the three-leaf stage of growth. Leaf sap from healthy-appearing St. Augustinegrass plants was applied to test plants, and an additional set of plants was left uninoculated. Half of the test plants were placed in a greenhouse (22–39 C) and the others in a temperature-controlled room (25 C) 30 cm below a bank of eight 122-cm-long fluorescent lamps (F48T12/GRO/VHO, GTE Sylvania) with a 16-hr light period. Assays on St. Augustinegrass were only on plants maintained in the greenhouse. All plants were examined periodically for symptoms.

For serologic tests, extracts from plants were assayed in immunodiffusion plates with antiserum to a Louisiana isolate of the SAD strain of Panicum mosaic virus (SAD-PMV-II) kindly supplied by R. W. Toler of Texas A&M University. Immunodiffusion plates contained 0.8% agarose, 0.85% sodium chloride, and 0.02% sodium azide in 0.05 M tris-hydrogen chloride buffer, pH 7.2. Leaf tissues for assay were ground in tris buffer, 1:2 (w/v).

RESULTS

Extracts from all three St. Augustinegrass samples with SAD symptoms induced symptoms in German foxtail millet, proso millet, crabgrass, and St. Augustinegrass within 7–23 days. German foxtail millet was the most sensitive assay plant; five of five plants inoculated with each sample and incubated at each temperature regime developed symptoms within 9 days. At 25 C, an average of four of five proso millet and five of five crabgrass plants became infected from each tissue sample. All infected proso millet plants died within 23 days. St. Augustinegrass plants developed local lesions on inoculated leaves after 15 days, and mottling from systemic infection appeared on uninoculated leaves after 31 days. All test plants inoculated with extracts from healthy-appearing St. Augustinegrass and all plants not inoculated were free of symptoms at the end of the test period. Corn, sorghum, Johnsongrass, and pearl millet did not show symptoms and were negative for infectivity when representative groups of plants were collectively back-assayed to German foxtail millet.

In serologic tests, extracts from each of the three samples of St. Augustinegrass exhibiting SAD symptoms formed distinct precipitin bands in immunodiffusion plates with antiserum to SAD-PMV-II. Extracts from healthy-appearing St. Augustinegrass did not produce precipitin lines.

DISCUSSION

Symptomatology, reaction of indicator plants, and serologic tests indicated that SAD-PMV is present in St. Augustinegrass in the Little Rock area of Arkansas. After the initial report of SAD in Texas in 1969 (4,6), St. Augustinegrass lawns in Little Rock and other parts of Arkansas were thoroughly surveyed for the disease in 1971, but it was not detected (2). According to homeowners, lawns that now contain diseased turf were established 8–15 yr ago. Two homeowners, who subsequently added small amounts of sod

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to their lawns in recent years, first noticed a slight yellowing in their lawns in the summer of 1980. It is difficult to determine whether the present occurrence of the disease is the result of previously undetected infection sites or whether the virus has been introduced recently with sod. Because St. Augustinegrass is not produced in Arkansas, the original source of infected turf was probably sod imported from other states.

Control of SAD in Arkansas will probably depend upon use of virus-resistant cultivars. Although the Floratam cultivar is resistant to SAD-PMV (5,7), it is not cold tolerant. The Raleigh cultivar (NCSA 21) is also SAD-PMV resistant (1), and it may be sufficiently winter hardy for use in central and southern Arkansas. It will be tested for SAD-PMV resistance and cold tolerance in central Arkansas.

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