Effect of Sugarcane Cultivar Susceptibility on Spread of Ratoon Stunting Disease by the Mechanical Harvester

KENNETH E. DAMANN, Department of Plant Pathology and Crop Physiology, Louisiana Agricultural Experiment Station, LSU Agricultural Center, Baton Rouge, 70803

ABSTRACT

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The spread of ratoon stunting disease by the mechanical harvester was followed in disease-free sugarcane plants of cultivars susceptible, intermediate, or resistant to the disease. Incidence in a 12-m row of susceptible cultivar L 62-96 preceded by one, two, or four diseased plants was 30, 60, and 75%, respectively, after one harvest and 84, 98, and 99%, respectively, after two harvests. Incidence in intermediate cultivar CP 65-357 was 10, 11, and 9% in the first ratoon and 26, 24, and 44% in the second ratoon. No spread was detected in resistant cultivar L 60-25. Gradients were apparent in consecutive 3-m segments of rows. Little spread occurred beyond the first segments in CP 65-357. In contrast, incidences with L 62-96 were 4, 38, and 43% in the fourth 3-m segment of rows preceded by one, two, or four diseased plants, respectively.

Ratoon stunting disease (RSD) of sugarcane (interspecific hybrids of *Saccharum* spp.) is cited as being the most important sugarcane disease in the world (12). This is due to its cryptic and chronic nature, a classic example of an endemic disease sensu Vanderplank (14). Yield losses in replicated trials averaged 5–32%, depending on cultivar and growing conditions (10). The disease is caused by the xylem-limited fastidious bacterium *Clavibacter xyli* subsp. *xyli* Davis et al (7,8). The disease is spread by planting infected stalks or by using a mechanical harvester or other implements that introduce the bacterium into plant xylem vessels (9). No significant vector other than man has been detected. Disease levels in seed sources in Louisiana in 1986 were estimated at 33% (4). This level of disease occurs despite the availability of heat-treatment methods for control of RSD (3).

There is little information on the efficiency of spread of RSD by the mechanical harvester (13). A 10% average annual increase in incidence occurred across diverse cultivars through the fifth year after heat treatment (4). This was interpreted as being due to harvester spread of the disease back into crops started with heat-treated cane.

Two main factors, both cultivar-related, probably affect the efficiency of RSD spread by the harvester. First, the bacterial concentration necessary to infect one-half of the plants inoculated differs among cultivars, as found through infectivity titration experiments (11). The concentration is higher for resistant cultivars (approximately 10^8/ml) than for susceptible cultivars (approximately 10^4/ml). Second, the concentration of bacteria in vascular extracts of stalks tends to be higher for susceptible cultivars (approximately 10^8/ml) than for resistant cultivars (approximately 10^6/ml) (5). These factors should make RSD more likely to spread in the susceptible cultivars and less likely to spread in the resistant.

Determination of the extent of spread in different cultivars would be useful in establishing disease thresholds for planting material, especially since the 3-yr crop cycle in Louisiana includes three harvests. This study addresses the spread of RSD to the ratoon crops by the bottom blade of the Louisiana whole-stalk sugarcane harvester as affected by cultivar susceptibility and number of diseased source plants. An abstract of this work has been published (2).

MATERIALS AND METHODS

Thirteen stalks (1–2 m long) of sugarcane cultivars L 60-25, CP 65-357, and L 62-96 were planted per 17 m of row at the St. Gabriel Research Station on 2 September 1986. The experiment was replicated three times. Every other row was left fallow to provide easy access for future sampling.

Each stalk of L 60-25 and L 62-96 was numbered before being planted. The basal two-node cutting from each was excised, and the vascular contents of each was removed by centrifugation, then examined with dark-field optics for cells of *C. x. xyli* (1). The bacterium was identified by its coryneform appearance. Cross sections of the cutting were examined for alkaline-induced metaxylem autofluorescence, a putative symptom of RSD (1). Stalks of CP 65-357 were provided by Crop Genetics International (Dorsey, MD) and are commercially marketed in Louisiana as Kleen-Tek CP 65-357. Because these plants were derived from a proprietary meristem-tip culture of disease-free plants and were propagated in nursuries monitored for disease by Crops Genetics International, individual stalks were assumed to be free of RSD.

Diseased stools (one, two, or four) of these cultivars were transplanted in front of the 17-m rows on 27 July 1987. In addition, three rows of CP 65-357 that did not have diseased stool transplants were cultivated during April 1987 with a Lilloston cultivator that had been used in a plot of diseased cv. CP 70-321.

The plots were harvested with a Louisiana whole-stalk, single-row harvester during October 1987. The harvester blade was initially disinfected with Lysol. The order of harvest was rows of 1) the more resistant cultivar L 60-25, 2) the intermediate cultivar CP 65-357, 3) the Lilloston-cultivated CP 65-357, and 4) the susceptible cultivar L 62-96, which minimized carryover effects.

Spread of the pathogen associated with the first harvest on 8 October 1987 was determined by sampling and analyzing the stubble during November and December 1988. The 1-yr delay in sampling after harvest was to allow the pathogen to colonize plants to which it had been dispersed and to increase to readily detectable levels associated with the latter stages of plant growth (6). A tape was stretched from the source plants down the 17-m row. Basal two-node cuttings were taken at 0.3-m intervals and numbered consecutively, from 1 to 40, for the first 12 m of each row; numbers for voids in a row were omitted. Samples were taken to the laboratory and analyzed for *C. x. xyli* in the xylem extracts, and internode cross sections were examined for alkaline-induced metaxylem autofluorescence as described above (1). After the sampling was completed, the plots were harvested in the same sequence as before. The sampling and analyzing of the second stubble crop for RSD were conducted in the same manner during October and November 1989.

RESULTS

The incidence of RSD among stalks in the first and second ratoon crops was affected by the susceptibility of the cul-

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tivar and the amount of inoculum in the form of diseased source plants (Table 1). None of the plants of resistant cultivar L 60-25 had the disease, whereas nearly all of the plants of susceptible cultivar L 62-96 were infected by the time the second ratoon crop had matured. The level of initial inoculum, i.e., one, two, or four diseased source plants, had no effect on incidence of RSD in intermediate cultivar CP 65-357 after one harvest; the plots preceded by four source plants had nearly twice the incidence after the second harvest as those preceded by one or two source plants. The susceptible cultivar, L 62-96, showed a dosage-dependent spread after each harvest.

Gradients of spread from the source plants were evident in consecutive 3-m segments of rows (Tables 2 and 3). With CP 65-357, there was little spread beyond the first segment (Table 2); with susceptible L 62-96, approximately 40% of the plants were infected in the fourth segment of rows with two or four source plants (Table 3).

Cultivation of CP 65-357 with a contaminated Lilloston cultivator in the spring apparently did not spread the disease. No RSD was detected among those plants.

Table 1. Percentage of samples with ratoon stunting disease collected from sugarcane cultivars L 60-25 (resistant), CP 65-357 (intermediate), and L 62-96 (susceptible) in ratoon crops after one (1988) or two (1989) harvests with a whole-stalk, single-row mechanical harvester

No. of inoculum source plants	Percentage of samples with ratoon stunting disease[a]					
	L 60-25		CP 65-357		L 62-96	
	1988	1989	1988	1989	1988	1989
1	0	0	10	26	30	84
2	0	0	11	24	60	98
4	0	0	9	44	75	99

[a]Samples were collected at 0.3-m intervals from the first 12 m following inoculum source plants.

Table 2. Percentage of samples with ratoon stunting disease of sugarcane cultivar CP 65-357 (intermediate) within segments of rows from inoculum source plants after one harvest with a whole-stalk, single-row mechanical harvester

Segment	Percentage of samples with ratoon stunting disease[a]			
	Number of inoculum source plants			
	1	2	4	Average
1	25	36	27	28
2	8	0	0	4
3	0	7	4	3
4	0	0	4	2

[a]Samples were collected at 0.3-m intervals within consecutive 3-m segments of 12-m rows.

Table 3. Percentage of samples with ratoon stunting disease of sugarcane cultivar L 62-96 (susceptible) within segments of rows from inoculum source plants after one harvest with a whole-stalk, single-row mechanical harvester

Segment	Percentage of samples with ratoon stunting disease[a]			
	Number of inoculum source plants			
	1	2	4	Average
1	61	88	96	82
2	25	73	81	60
3	30	37	75	46
4	4	38	43	28

[a]Samples were collected at 0.3-m intervals within consecutive 3-m segments of 12-m rows.

DISCUSSION

Clearly, there was a large cultivar effect on frequency of spread of RSD by the mechanical harvester. The relationship between the populations of *C. x. xyli* in infected plants and the ED_{50} for inoculation of a cultivar appear to be predictive of the spread of the disease. In a sense, this is an infectivity titration experiment done with a mechanical harvester. When pathogen populations are low in the source plant and a high titer is needed for infection, as with L 60-25, little spread would be expected; in the tests reported here, no spread was detected. When the pathogen population exceeds by several orders of magnitude the ED_{50} for inoculation, then significant spread would be expected, as with L 62-96 in this study.

We did not determine the maximum distance of RSD spread from a single infected plant. However, I would expect spread to be beyond 12 m for L 62-96 but not for CP 65-357, judging from the RSD incidence found in the fourth 3-m segments.

Harvester spread of RSD in Australia in the susceptible cultivator Q 110 ranged from 62 to 67% of the first seven stools, with intermittent spread thereafter up to a maximum of 35 stools from the source (13). This is comparable to the results with susceptible L 62-96, where 61% of the samples in the first segment were diseased and some disease was found in the last segment. When the harvester bottom blade was run in the soil after cutting infected stools, the spread among healthy stools was 19% in the first seven stools in the Australian study. Continuous harvesting with the blade set below ground level abolishes spread but is impractical in commercial production.

The results of this study support the hypothesis that spread of ratoon stunting disease is cultivar-specific and related to the bacterial populations a cultivar will support and the ED_{50} for inoculation. Attempts to set thresholds for acceptable levels of RSD in seed cane need to take spread of the disease into consideration.

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