# Relationship Between Resistance to *Clavibacter xyli* subsp. *xyli* Colonization in Sugarcane and Spread of Ratoon Stunting Disease in the Field

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**ABSTRACT**

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The spread of ratoon stunting disease (RSD) of sugarcane resulting from hand-cutting was monitored in plots of six different cultivars that varied in RSD resistance. Cultivar resistance influenced the disease spread. Very little spread was observed among plants of an RSD-resistant cultivar that had low numbers of *Clavibacter xyli* subsp. *xyli*–colonized vascular bundles (cvb) determined in 1-cm-diameter core sample of stalk tissue; whereas the rate of spread and incidence were highest in the most susceptible cultivar, which had approximately 20 cvb per core sample. Disease spread followed the direction of hand harvest within rows from infected source plants (inoculated) to test plants (uninoculated). The incidence of RSD in test plants increased with the number of ratoon crops harvested.

Ratoon stunting disease (RSD) is a major disease of sugarcane (interspecific hybrids of *Saccharum* spp.) and occurs in most sugarcane growing areas (15). In Florida, RSD causes yield losses estimated at 5% over the entire production region (14,19). Elsewhere, losses as high as 30% have been reported (15). Although resistance to RSD is known, the disease is usually controlled by heat treatment of seedpieces (sections of stalk used to vegetatively propagate the crop) or seedcane to eradicate the pathogen followed by phytosanitary practices to prevent reinfection (15). Without strict compliance to phytosanitary practices, plants of susceptible cultivars in commercial fields often become infected within a few years (7,9,15). The difficulty in maintaining disease-free sugarcane in seedcane nurseries and commercial fields is indicated by reports of infected sugarcane in areas with control programs (1,4,9,10,21,22). Soil transmission of the pathogen has been reported (2,3), which may help explain the failure of phytosanitation to control RSD.

RSD is caused by a xylem-limited bacterium, *Clavibacter xyli* subsp. *xyli* (13). The pathogen causes no external symptoms except reduction of plant growth. Reddish colored vascular bundles in basal nodes of infected stalks have been used for diagnosis but are not reliable (15). Reliable diagnosis became possible with phase-contrast microscopy and serological techniques to detect the pathogen. One serological technique, the tissue blot immunoassay (TBIA), allowed the assay of large numbers of plant samples to detect the pathogen and determine relative *C. x.* subsp. *xyli* populations by counting the number of colonized vascular bundles (cvb) in stalk samples (12,16,17). TBIA is useful in determining reactions of sugarcane cultivars to *C. x.* subsp. *xyli*, since resistance is inversely related to pathogen population (11). TBIA has been used in several RSD studies to quantify resistance of sugarcane cultivars to *C. x.* subsp. *xyli* colonization (5,12,20).

Although *C. x.* subsp. *xyli* is spread by the planting of infected seedpieces, another major mechanism of spread is mechanical transmission by implements that wound infected plants and spread pathogen-laden sap to healthy plants. Cutting knives, on machine harvesters or those used by hand, are particularly important. The rate and distance of RSD spread from infected plants during mechanical harvest was high, moderate, and nil in cultivars that were susceptible, intermediate, and resistant, respectively, to *C. x.* subsp. *xyli* colonization (8). The lack of RSD spread is a measure of field resistance. Although absolute field resistance with no disease spread is the most beneficial, reduced spread would enhance control by common phytosanitary practices. No quantitative estimates of the host resistance level required to eliminate the spread of RSD in commercial fields have been made. Our objectives were: (i) to determine the relationship of host resistance, as measured by the extent of bacterial colonization of the plant's vascular system, to rates of disease progress over time and space from an inoculum source, and (ii) to test the prediction that highly resistant cultivars could confer a field resistance that prevents the spread of RSD under field conditions.

## MATERIALS AND METHODS

**Sugarcane cultivars.** Six cultivars varying from highly susceptible to resistant to RSD were used to evaluate the relationship between the level of resistance and spread of the disease. The mean number of vascular bundles colonized by *C. x. subsp. xyli* after inoculation was used to rank the level of RSD resistance of the cultivars. The cultivar with the highest mean number of cvb after inoculation was considered the most susceptible, and the cultivar with the lowest number was considered the most resistant. The mean number of cvb for *C. x.* subsp. *xyli*–inoculated plants in this study ranged from 20.4 for the most susceptible cultivar, CP 53-1, to a low of 0.18 for the highly resistant cultivar, L 60-25. The four cultivars with intermediate RSD resistance were CP 89-2315 (cvb = 7.84), CP 72-1210 (cvb = 6.48), CP 89-2387 (cvb = 1.40), and CP 89-2375 (cvb = 1.29).

**Sugarcane inoculation.** Heat-treated (50°C for 2 h) single-bud seedpieces were inoculated by immersion for 10 min in undiluted juice expressed from RSD-infected stalks of cultivar CP 53-1, a highly susceptible cultivar that supports high *C. x.* subsp. *xyli* populations. Inoculated and uninoculated heat-treated seedpieces were planted in flats and grown in a greenhouse for 2 months, then transplanted to the field. An excess of plants was initially grown in the flats, and the most vigorous plants were used.

**Experimental design and plot layout.** The test was designed to determine the effect of host plant resistance on mechanical spread of RSD from infected plants to healthy plants of the same cultivar during hand harvesting with cane knives. Host plant resistance varied by cultivar and provided different inoculum levels due to the

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number of cvb and RSD incidence of inoculated source plants. Six cultivars replicated seven times were planted in a randomized complete block design. Each three-row plot contained 24 plants, eight per row of a single cultivar. Plants were spaced 1 m apart within rows, and rows were spaced 1.5 m apart. The first three plants in each row were derived from the inoculated seedpieces and served as the inoculum source. The five remaining plants were uninoculated. The uninoculated plants were numbered 1 through 5 starting from the inoculum source. Plants were harvested by hand-cutting, starting from the inoculum source plants and continuing through the uninoculated test plants in each row. Knives were disinfected by swabbing with a 5% solution of commercial quaternary ammonium germicide (Micro-Quat, Economic Laboratory, St. Paul, MN) between plots and rows within a plot but not between plants within rows of a plot.

**Plant assays.** Sections (0.5 m) were cut from the base of a single stalk from each plant 1, 2, 3, and 4 years after planting (the plant-, first-, second-, and third-ratoon crops, respectively) prior to harvest using the same disinfection procedure as described for harvesting. Determinations of cvb were made from the single stalk samples 9 months after transplanting for the plant crop and 11 to 13 months after the previous harvest for the ratoon crops. The TBIA procedure developed by Harrison and Davis (16,17), as modified by Davis et al. (12), was used to determine the number of cvb. Briefly, a longitudinal core of tissue from each stalk sample was removed from a single internode within each stalk sample, using a 1-cm-diameter cork borer, and cut to 1-cm length. The cores were placed within individual wells of a plastic template with one transversely cut surface of each core resting on a nitrocellulose membrane. The membrane had been stacked on a piece of 3mm Whatman paper with an underlying layer of absorbent paper toweling and plastic support on the bottom. The stalk cores were centrifuged to force the sap from the xylem, forming a pellet of bacteria on the nitrocellulose membrane surface directly below the colonized vascular bundles. The liquid was absorbed by the filter paper and toweling. Membranes were dried for 1 h at 80°C and subjected to an enzyme immunoassay to detect the number of cvb. A plant was considered infected if one or more cvb was detected in a stalk sample.

**Statistical analysis.** Square root transformed data were analyzed. The results of these analyses were similar to the results obtained from analyses on the original data; therefore analyses of the raw data are presented. Analyses were performed using PROC GLM and PROG REG procedures in SAS (SAS Institute, Cary, NC). Mean separations were determined using Fisher's LSD ($P = 0.05$).

Mean number of cvb and percent RSD infection of the inoculum source defined the inherent susceptibility of each clone. Incidence in the test plants was analyzed for each year and across all years to determine the overall increase in disease over time and the relative incidence between clones in the test.

Regression analysis was performed to calculate a response surface model to depict the change in disease with increasing distance from the inoculum source over time for each cultivar. This characterization was used to evaluate the potential rate of spread with respect to resistance (mean number of cvb) of a given cultivar to RSD. Linear regression of RSD incidence at the end of the experiment on the mean cvb of the inoculum source was also performed to present a simple illustration of disease incidence versus the inherent resistance of a given clone after 3 years.

## RESULTS

The mean number of cvb and percent RSD incidence of the inoculated source plants varied between cultivars (Table 1). Response surface models describing distance from inoculum source and crop age regressed on percent incidence for each cultivar is presented (Fig. 1). Estimated parameters for the response surface models are listed (Table 2). The mean incidence of RSD for the uninoculated plants that resulted from infection from inoculum source plants is presented for each cultivar by distance from inoculum source (plant position) and crop age (Fig. 1). RSD spread from inoculum source plants to the uninoculated plants in all sugarcane cultivars during hand-cut harvest, with the possible exception of L 60-25 (Fig. 1). The incidence of RSD in the uninoculated plants resulting from transmission during harvest varied among cultivars in ratoon crops and increased with cultivar susceptibility and time (Table 1). There was a direct correlation ($r = 0.93$) between the mean number of cvb of the inoculated source plants of the different cultivars and the incidence at the end of the experiment (Fig. 2). The incidence and the rate of spread were greatest in CP 53-1, the most susceptible cultivar (Fig. 1). Incidence of RSD in CP 53-1 plants was higher than the incidence in all other cultivars in the first through third ratoons (Table 1). The ranking of the cultivars from highest to lowest incidence of RSD across ratoon crops was: CP 53-1, CP 89-2315, CP 72-1210, CP 89-2387, CP 89-2375, and L 60-25. The incidence of RSD did not increase over ratoon crops of the most resistant cultivar, L 60-25 (cvb = 0.18), (Fig. 1 and Table 1).

The disease spread was in the direction of cutting, as evidenced by the gradient of decreasing incidence from inoculum source (plant position down the row) (Fig. 1). This was especially evident in the most susceptible cultivar, CP 53-1, where there was a dramatic drop in disease incidence by plant position from the RSD inoculum source. The disease gradient was apparent in the data for all but one of the six cultivars. A relationship between plant position and RSD incidence was not apparent for the most resistant cultivar, L 60-25 (Fig. 1).

The overall incidence of RSD increased in most cultivars with crop age during each successive ratoon crop (Table 1). CP 53-1, the most susceptible cultivar, had the most important increase in RSD incidence, with 56.9, 72.9, and 76.2% in the first, second, and third ratoon crops, respectively. The incidence of RSD increased to a lesser extent in cultivars CP 89-2315, CP 72-1210, CP 89-2375, and CP 89-2387 during the same cropping cycle. For the most resistant cultivar, L 60-25, no pattern of disease increase was detected. RSD did not reach an equilibrium state during the experiment for any cultivar, with the possible exception of L 60-25.

**Table 1.** Reaction of sugarcane cultivars to ratoon stunting disease (RSD) and spread to uninoculated plants during three harvests

| | | | % RSD incidence in uninoculated plants[z] | | | |
|---|---|---|---|---|---|---|
| | RSD source plants[y] | | Ratoon crop | | | |
| Cultivar | # cvb | % RSD inf. | First | Second | Third | Combined |
| CP 53-1 | 20.40 a | 96.6 a | 56.9 a | 72.9 a | 76.2 a | 67.9 a |
| CP 89-2315 | 7.84 b | 75.8 b | 18.5 bc | 34.6 b | 37.4 b | 29.5 b |
| CP 72-1210 | 6.48 b | 91.8 a | 26.0 b | 25.0 b | 34.9 b | 28.6 b |
| CP 89-2387 | 1.40 c | 41.3 c | 16.7 bc | 31.6 b | 33.8 b | 26.5 b |
| CP 89-2375 | 1.29 c | 54.8 c | 10.5 c | 21.3 b | 23.8 b | 18.0 bc |
| L 60-25 | 0.18 c | 17.5 d | 18.3 bc | 10.1 b | 2.4 c | 10.8 c |
| LSD 0.05 | 5.00 | 15.14 | 14.48 | 31.60 | 16.86 | 15.67 |

[y] Mean number of colonized vascular bundles (cvb) in stalk cross sections and percent RSD infection in inoculated source plants. cvb = number of vascular bundles colonized by *Clavibacter xyli* subsp. *xyli* in stalks sampled in the crop. The higher the number of cvb, the more susceptible the cultivar; conversely, the lower the number of cvb, the more resistant.

[z] Mean percent RSD infection. Infection occurred during harvest as a result of hand-cutting inoculum source plants at the beginning of the plot and continuing to cut the uninoculated plants. Crop number is the number of previous harvests in the cycle. Original data and analysis presented. Similar analysis obtained on square root transformed data. Different letters in columns indicate significant differences according to Fisher's protected least significant differences (LSD) ($P \leq 0.05$).

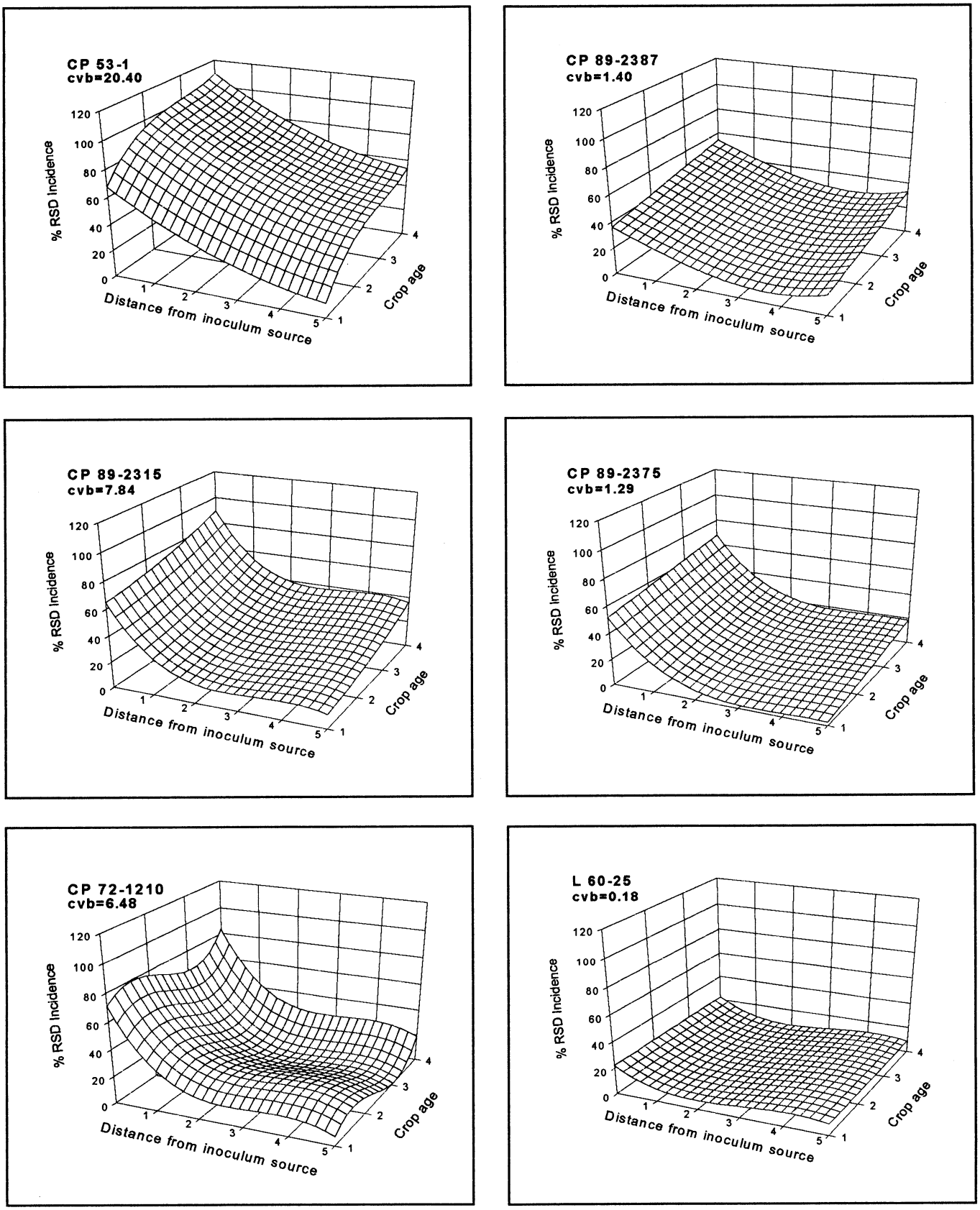


**Fig. 1.** Response surface models of six sugarcane cultivars describing distance from ratoon stunting disease (RSD) inoculum source plants and crop age regressed on percent RSD incidence. Crop age refers to years after planting. The 4-year crop cycle was: year 1 = plant, 2 = first ratoon, 3 = second ratoon, and 4 = third ratoon. Inoculum source plant is at 0, and uninoculated plants are at positions 1 through 5 from the source plants. Plants were harvested yearly by hand-cutting from the inoculum source through plant five. Data were collected just prior to harvest. The number of vascular bundles colonized (cvb) by *Clavibacter xyli* subsp. *xyli* was used to rank RSD resistance of cultivars. The cultivar with the highest the number of cvb is most susceptible, and with the lowest number is the most resistant.

**Table 2.** Estimated parameter values and coefficients of determination ($R^2$) describing distance (D) from ratoon stunting disease (RSD) inoculum source plants and crop age (C) regressed on percent RSD incidence of six sugarcane cultivars[x]

| Cultivar | Regression terms[y] | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | Intercept | C | D | $C^2$ | $D^2$ | $C^3$ | $D^3$ | $R^2$ |
| CP 53-1 | ... | 94.30 | −19.06 | −30.18[z] | 1.56 | 3.37[z] | ... | 0.35 |
| CP 89-2315 | 62.60 | 0.82 NS | −48.74 | 1.47 | 14.70 | ... | −1.42 | 0.27 |
| CP 72-1210 | ... | 119.80 | −55.73 | −56.75 | 17.57 | 8.00 | −1.80 | 0.32 |
| CP 89-2387 | 30.14 | 5.97 | −11.41 | ... | 0.39 NS | ... | 0.28 | 0.08 |
| CP 89-2375 | 46.39 | 5.26 | −38.63 | ... | 9.92 | ... | −0.83[z] | 0.25 |
| L 60-25 | 22.21 | ... | −18.67 | ... | 7.04 | ... | −0.77[z] | 0.04 |

[x] Crop cycle: year 1 = plant, 2 = first ratoon, 3 = second ratoon, and 4 = third ratoon.

[y] Regression terms: C = crop age in years from transplanting plants to the field. D = distance from RSD inoculum source plants. All terms are significant at $P \leq 0.05$ except where indicated. NS = not significant.

[z] Significant at $P \leq 0.15$.

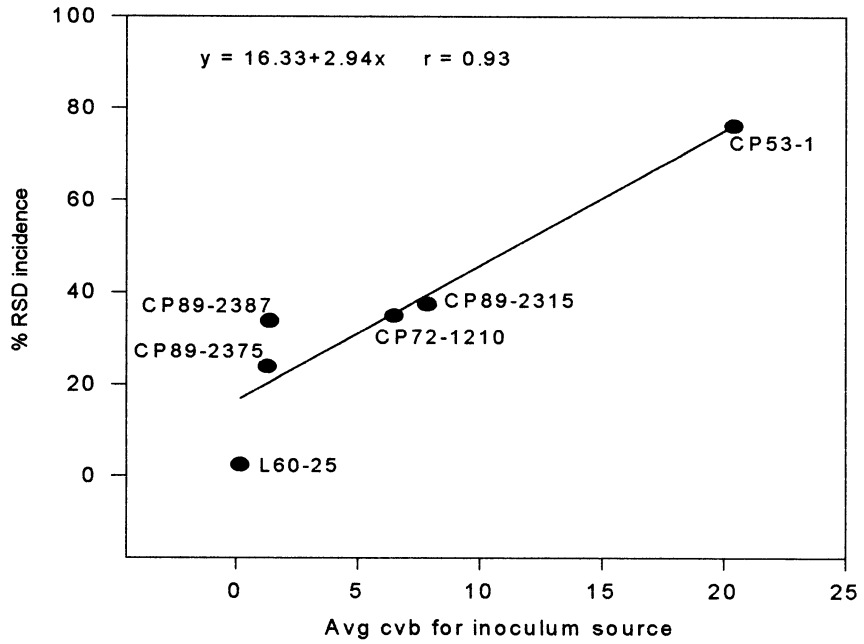


**Fig. 2.** Relationship of ratoon stunting disease (RSD) incidence of uninoculated plants (third ratoon crop) and the average colonized vascular bundles (cvb) in stalks of the inoculated source plants (plant crop) of six sugarcane cultivars. Infection of uninoculated plants resulted from inoculum being spread from inoculum source plants during hand-cut harvesting.

## DISCUSSION

The mean number of cvb of a sugarcane cultivar, a measure of RSD resistance, was a quantitative indicator of the rate of spread in a field of the pathogen within a cultivar. Although the spread of *C. x.* subsp. *xyli* by cutting knives and mechanical harvesters is well-documented, few studies have been designed to monitor disease spread by incorporating cultivars with varying levels of RSD resistance. One test in Louisiana detected no spread by mechanical harvesters from infected plants of the RSD-resistant cultivar L 60-25 (8). Similarly, in our study, only a low rate of disease spread was detected with this cultivar as a result of hand-cut harvest. In the Louisiana test, spread was greater for L 62-92 than for CP 65-357 (8). The mean numbers of cvb for these cultivars were 53 and 16, respectively, as determined in another Louisiana study (M. Grisham, *personal communication*). Although these values were determined at a different geographic location and under different environmental conditions than our study, the cvb values derived from the Louisiana study were consistent indicators of the spread of RSD observed in this test.

Genetic diversity in resistance to RSD is known to occur (11,21,23). A factor associated with RSD resistance has been low populations of *C. x.* subsp. *xyli* within infected sugarcane stalks. *C. x.* subsp. *xyli* populations were reported to be inversely related to RSD resistance with respect to crop yield (11). The number of cvb reflected the *C. x.* subsp. *xyli* population size (16,17). This study indicates that measurement of cvb can provide a valid method to screen sugarcane cultivars for resistance to *C. x.* subsp. *xyli* colonization.

Our objective, to determine the level of host resistance resulting in restricted RSD spread, was accomplished. The level of host resistance resulting either in no or very low RSD spread appears to be less than one cvb. Such a level in this study was detected in L 60-25, where it averaged 0.18 cvb. This agrees with data from commercial fields where the incidence of RSD has been low in cultivars with this level of resistance (cvb levels) (6). Cultivars should be developed with the highest resistance to *C. x.* subsp. *xyli* colonization possible (lowest cvb), since there is a direct relationship between mean number of cvb and RSD incidence. Whether resistant cultivars can be readily infected in mixed plantings with infected, highly susceptible cultivars is not known. There is a direct correlation between inoculum dose and rate of infection (18), which would suggest that such a situation might be possible. However, when a resistant cultivar is planted in a pure stand and provides its own inoculum, disease dissemination is limited.

The relative merits of control by heat treatment versus breeding for resistance are of interest. Roach (21) called attention to the discrepancy between theory and practice in the control of RSD through heat treatment of seedcane. In addition, we would emphasize the difference in economic trends that could reasonably be expected to develop. Sugarcane industries that rely solely on heat treatment and sanitation are locked indefinitely into an expensive control program on every farm. Control through breeding would tend to increase the average resistance of clones released for production and could eventually eliminate the need for control at the grower level. Thus, by putting the burden of control on the breeder and not on the grower, more uniform control would be possible.

Resistance can provide a practical means to control RSD because two commercial cultivars, CP 72-2086 (cvb = 0.15) and CL 73-239 (cvb = 0.03), have been in production for several years without any buildup of the disease even though no heat treatment and special phytosanitary practices have been used (6). In contrast, susceptible cultivars grown during the same period under similar circumstances are now universally infected, with few exceptions. Others have documented that sugarcane cultivars have a wide diversity in their

reaction to RSD (22). Broad genetic diversity and high heritability of RSD resistance should permit resistant cultivars to be developed (20). In fact, CP 72-2086 and CL 73-239 were developed in programs without an active RSD-resistance screening program in place when they were released. Resistant cultivars can be developed by using a screening program for resistance, based on cvb, to increase the frequency of RSD resistance in advanced lines.

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