Epidemiology and Control of Bacterial Canker of Papaya Caused by an *Erwinia* sp. on St. Croix, U.S. Virgin Islands

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ABSTRACT

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A species of *Erwinia* caused angular water-soaked lesions on leaves and firm water-soaked cankers on stems of *Carica papaya*. Infected trees died soon after stem cankers were observed. The pathogen did not survive longer than 2 wk in soil but may survive indefinitely in leaf lesions or cankers of infected trees and as an epiphyte on leaves of suitable nonhosts. Free moisture (except for dissemination and deposition) does not enhance disease severity or pathogen survival on leaf surfaces. Commercial bactericides, antibiotics, and an antagonistic fluorescent pseudomonad failed to control the disease. Resistance was observed in a number of land cultivars from the Virgin Islands and the eastern Caribbean. Commercial cultivars from Hawaii, Puerto Rico, Costa Rica, and Jamaica were highly susceptible to the canker disease.

A previously unknown disorder of bearing papaya (*Carica papaya* L.) was first observed on St. Croix in 1955. The disease was thought to cause a wide variety of unrelated symptoms including greasy or water-soaked spots and cankers on stems; scant, rigid, chlorotic foliage with water-soaked or yellow necrotic spots; bumpy stems; and a "pencil-point" appearance followed by tree collapse in advanced cases. Inoculation studies under controlled conditions tentatively indicated that *Corynespora cassicola* and possibly a *Botryodiplodia* sp. caused the disease (1). At that time, the disease was named "St. Croix papaya decline."

In 1931, von Rant (11) reported a disease of papaya in Java with symptoms similar to those observed by Bird et al (1). The causal agent was determined to be *Bacillus papayae*, later placed in the genus *Erwinia* by Magrou (2). In 1981, Trujillo and Schroth (10) described a decline disease of papaya in the Mariana Islands that displayed symptoms very similar to those reported in Java and St. Croix. Their results showed that the Mariana disease was also caused by an *Erwinia* sp. Subsequent investigations by Schroth (10) on St. Croix showed that the Mariana and St. Croix diseases were not caused by the same bacterium.

This paper reports the characterization of the causal agent of bacterial canker of papaya on St. Croix; the influence of various factors on the etiology, epidemiology, and control of this disease; and the distinction between it and the syndrome known as St. Croix papaya decline.

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MATERIALS AND METHODS

All field and greenhouse trials were conducted at the Virgin Islands Agricultural Experiment Station on St. Croix. The soil is a Fredensborg clay loam, 25–38 cm thick with a pH of 8.1, overlying caliche. The St. Croix climate is warm and dry with an average annual temperature of 26.5 ± 1.6 C and an average annual rainfall of 762–1,270 mm, depending on the location.

Isolation of causal organism. Diseased leaf and stem segments were washed under tap water for 30 min. Tissue chips were excised from advancing margins of water-soaked leaf and stem lesions, dipped in ethanol, flamed briefly, and placed on drops of sterile distilled water (SDW) in sterile petri dishes. After tituration with a glass rod, loopfuls of macerate were streaked onto agar plates containing King's medium B (KB) (7) or nutrient agar (NA). Isolations for fungal pathogens were made in a similar manner. After the tap water wash, however, tissue chips were surface-sterilized for 3 min in 0.54% sodium hypochlorite, washed in SDW, and plated on water agar and potato-dextrose agar.

Pathogenicity tests and identification. Suspensions of 10^8 colony-forming units per milliliter (cfu/ml) were misted with a chromatography sprayer over 8-wk-old papaya seedlings (cultivar Sunrise Solo). Inoculated plants were placed in a moist chamber at ambient temperature (26–32 C) for 24 hr, then returned to greenhouse benches. Wound inoculations were made by inserting toothpicks laden with bacteria into the fifth node from the apex. Wound-inoculated plants were not placed in a moist chamber.

Identification methods used in this study were based on standard determinative morphological observations and physiological tests (2,6,8). Ten isolates of the bacterium were collected from different locations on St. Croix at different times of the year. All isolates were obtained from fresh leaf or stem

lesions. Two cultures of *E. chrysanthemi* (UC107 and UC1067) and two of *E. carotovora* pv. *carotovora* (UC78 and UC94) obtained from M. N. Schroth, University of California, Berkeley, were included as standards.

Selection of rifampicin-resistant marked mutants for epidemiology studies. Spontaneous mutants of the pathogen resistant to 100 μg/ml of rifampicin were screened for similarity to wild types under field and greenhouse conditions. The resistant isolate PVIc1R^r survived on papaya tissues and caused symptoms indistinguishable from the wild type PVIc1.

Because viability and pathogenicity decreased after prolonged storage in SDW or half-strength nutrient broth, inoculum for all tests was obtained from lesions of freshly inoculated 8-wk-old papaya plants. All inoculations were made with suspensions of PVIc1R^r from 48-hr-old KB cultures adjusted to 10^8 cfu/ml and applied to 8-wk-old papaya seedlings unless indicated otherwise.

Determination of effects of free moisture on symptom severity and pathogen survival. After inoculation, symptom development and leaf surface populations of the pathogen were determined for plants receiving 0, 4, 8, 24, and 72 hr of free moisture. Unmisted control plants were sampled at the same intervals. Free moisture was provided by atomizers timed to give a 5-sec misting at 5-min intervals that maintained a film of water on leaf surfaces with minimal runoff. Ambient temperatures were 28–35 C on both misted and unmisted benches.

Leaf surface populations were determined by submerging all the leaves (8–10) from each of four plants per treatment in flasks of sterile phosphate buffer (11.8 g of $K_2HPO_4 \cdot 3H_2O$, 6.25 g of KH_2PO_4, 1 g of peptone, and 1,000 ml of distilled water, final pH 7.0). A ratio of 10 ml of buffer per gram fresh weight (fr wt) of leaf tissue was used. Flasks were submerged in an ultrasonic bath for 10 min (5), then shaken vigorously by hand for a few seconds. The resulting suspension was dilution-plated onto KB + 200 μg/ml of cycloheximide + 200 μg/ml of benomyl (KBCB) and KB + 200 μg/ml of cycloheximide + 200 μg/ml of benomyl + 100 μg/ml of rifampicin (KBCBR). Colony counts were made 3–4 days after plating and incubation at 27 C and expressed as log cfu/g fr wt.

Symptoms were evaluated on eight plants per treatment 2 wk after inoculation on a scale of 0–4, where 0 = no stem cankers or leaf lesions, 1 = scattered leaf lesions, 2 = 1–10 lesions per leaf plus small stem cankers, 3 = >10 lesions per leaf and/or large girdling stem cankers, and 4 = plant dead.

Determination of pathogen survival in decaying plant tissue and papaya rhizosphere. Stem sections of papaya plants inoculated with PVIc1R^r displaying fresh, confluent cankers were placed in nylon mesh bags and buried 5 cm deep in both dry and moist field soil. Samples were removed from both soils 0, 1, 2, and 3 wk later. Bacterial populations in decaying tissue were determined by excising 1 cc from the middle of each stem section, titurating it in 5 ml of phosphate buffer (pH 7.0), and dilution-plating the macerate on KBCBR. Counts of resulting colonies were made 3–4 days later. Each sample from each date was replicated three times.

Rhizosphere populations of PVIc1R^r were assayed by drenching the root zones of four 8-wk-old papaya plants in moist field soil with a suspension (10^9 cfu/ml) of PVIc1R^r. Four subsamples were taken from the root zone at 0, 1, 2, and 3 wk using a No. 5 cork borer. This provided a total sample volume of root and soil of 16 cc per replicate. To this, 160 ml of phosphate buffer (pH 7.0) was added in 225-ml flasks, which were immersed in an ultrasonic bath at 27 C for 15 min, then vigorously shaken by hand; 0.1-ml aliquots were dilution-plated onto KBCBR and the resulting colonies counted 3–4 days later.

Survival of pathogen on leaf surfaces of nonhost plants. Twenty species of monocots and dicots commonly found in association with papaya were assayed for their ability to support epiphytic populations of the canker bacterium. Each plant species was started from a cutting or seed and maintained in a screenhouse. When plants were large enough to provide 15–20 g of leaf tissue for sampling, each was spray-inoculated with PVIc1R^r. Leaf surface populations were determined 1, 3, 7, and 14 days later as described previously.

Effects of various antibiotics, bactericides, and an antagonistic bacterium on symptom severity and disease spread. Streptomycin (Agrimycin 17), oxytetracycline (Mycoshield 17), copper hydroxide (Kocide 101), CGA-78039 50W, and a streptomycin-resistant fluorescent pseudomonad (AVI 194S^r), which displayed in vitro antibiosis toward the pathogen, were applied to papaya plants before and after a single inoculation with PVIc1R^r. Dilute sprays of experimental materials at maximum label rates and AVI 194S^r at 10^9 cfu/ml were applied with a hand-pressurized sprayer until runoff to four trees in each of four replicates. All experimental materials were applied to papaya plants at 2-wk intervals from the time of transplanting at 8 wk of age to the date of inoculation and first fruit set at 18 wk of age. After inoculation, all materials were applied at weekly intervals. The single inoculation of PVIc1R^r was made in a similar manner, using a suspension of

Table 1. Susceptibility of papaya cultivars to inoculation with the bacterial pathogen

Source	Cultivar	Symptom rating[y]	
		Spray-inoculated	Wound-inoculated
Hawaii	Waimanalo Solo	2.92 a[z]	3.61 a
	Kapoho Solo	2.78 a	3.50 a
	Sunrise Solo	2.78 a	3.44 a
	Higgins Solo	2.88 a	3.61 a
	Wilder Solo	2.60 a	3.81 a
	S-64	2.58 a	3.20 a
	P.R. 10-65	1.77 ab	2.55 a
Puerto Rico	P.R. 6-65	2.86 a	3.40 a
	P.R. 6-65 Improved	2.70 a	3.67 a
	P.R. 6-65 Dwarf	2.86 a	3.00 a
	P.R. 9-65	nd	2.57 a
Java	P.R. 7-65	2.78 a	3.40 a
Canary Islands	P.R. 8-65	2.78 a	2.78 a
Costa Rica	CATIE 12914	3.25 a	3.58 a
	CATIE 12915	3.00 a	3.60 a
	CATIE 12917	3.44 a	3.64 a
Virgin Islands	CVI 283-1	2.66 a	2.88 a
	CVI 283-2	2.70 a	2.65 a
	CVI 383-1	2.00 ab	2.10 ab
	CVI 383-2	2.00 ab	2.10 ab
	CVI 483-1	2.66 a	3.00 a
	CVI 583-1	2.41 a	3.00 a
	STT 683-1	1.49 b	1.50 b
	JH 783-1	2.41 a	1.97 ab
Jamaica	Jamaica 183	3.24 a	3.77 a
Trinidad	Trinidad Pink X	2.60 a	1.60 b
	Trinidad Pink	1.97 ab	1.70 b
	Trinidad Yellow	2.66 a	3.20 a
Barbados	Barbados Dwarf 2X	1.50 b	1.46 b

[y] Symptom severity rating based on a scale of 0–4, where 0 = no stem cankers or leaf lesions, 1 = scattered leaf lesions, 2 = 1–10 lesions per leaf plus small stem cankers, 3 = >10 lesions per leaf and/or large girdling stem cankers, and 4 = plant dead.

[z] Means within a column not followed by the same letter are significantly different ($P = 0.05$) according to Duncan's multiple range test.

10^8 cfu/ml applied to a uniform wetness to two of the four 18-wk-old trees in each replicate. Both inoculated and uninoculated trees were evaluated for disease symptoms 14 wk postinoculation based on the rating scale described previously.

Determination of susceptibility of various papaya cultivars to pathogen. Twenty-nine commercial and land papaya cultivars from diverse locations (Table 1) were evaluated for resistance to bacterial canker under greenhouse conditions. All plants were spray- and wound-inoculated, as described earlier, when 8 wk old. A minimum of 18 plants per variety was used for each inoculation method, and each variety was tested at least twice. Evaluations of symptom severity were made 3 wk after inoculation.

RESULTS

Isolation of causal organism. Only one type of bacterium was consistently isolated from diseased leaf and stem tissues. No fungal pathogens were associated with any of the isolations from the margins of water-soaked leaf or stem lesions. Numerous fungi, however, including *Fusarium* and *Alternaria* spp. and *Corynespora cassicola* were isolated from necrotic areas of stem cankers.

Pathogenicity tests and identification. Plants inoculated with the isolated bacterium displayed symptoms identical to those observed under natural conditions. Wound-inoculated plants developed firm water-soaked cankers around the point of inoculation 5–7 days after inoculation. At 2 wk, leaf lesions were observed above but not below the point of inoculation. Wound-inoculated plants usually did not survive longer than 4 wk.

Spray-inoculated plants displayed small water-soaked lesions becoming confluent along midveins and margins 4 days after inoculation. Two to 3 days after leaf lesions were observed, many infected leaves turned yellow and abscised. The remaining infected leaves developed large, oozing, systemic lesions that progressed a short distance into the petiole, causing the leaf blade to wither and hang pendant (Fig. 1B,C). After this was observed, abscission of the petiole usually occurred rapidly. Stem cankers with bacterial ooze were observed at the nodes and internodes of many of the spray-inoculated plants 3–4 wk later (Fig. 1A). Inoculated plants displaying only leaf lesions recovered without further symptoms after infected leaves were dropped. Spray and wound inoculations made with isolated fungi yielded no disease symptoms after 4 wk.

All isolates of the bacterium displayed characteristics that most closely associate it with the genus *Erwinia*. Colonies on KB were mucoid, creamy white, and sporadically produced a soluble blue pigment. Cells were single, straight, gram-negative rods 0.5–1 × 2–3 μm, and facultative anerobes. Other tests used for identification and differentiation are presented in Table 2.

Effects of free moisture on symptom severity and pathogen survival. Symptom severity and pathogen survival were not correlated with the durations of free moisture used in this study ($r = -0.633$ and -0.140, respectively). Mean disease ratings calculated for plants in all treatments ranged between 2.85 and 3.5 and were not significantly different. Plants with disease ratings ≥ 2.5 did not survive.

Leaf surface populations of the pathogen were significantly greater on plants receiving 4 hr of free moisture than on those kept dry for the same period ($P = 0.01$). However, no significant differences were observed in recovery of the pathogen from wet or dry leaves after 8, 24, and 72 hr (Table 3).

Pathogen survival in decaying plant tissue and papaya rhizosphere. Limitations in the accuracy of the dilution-plate method used in this experiment restricted the maximum resolution of pathogen populations to counts of ≥ 100 cfu/cc of buried plant tissue or root per soil sample. Initial populations of the pathogen in both decaying plant tissue and papaya rhizosphere ranged between 7.934 and 8.864 log cfu/cc.

In moist field soil, pathogen populations from both plant tissue and root/soil samples were too low to count after 1 wk. In dry soil, populations of 5.493 ± 1.100 log cfu/cc were recorded after 1 wk but were too low to count after 2 wk.

Survival of pathogen on leaf surfaces of nonhost plants. Measurable populations of PVIc1R^r were recorded 24 hr after inoculation on all species tested. After 7 days, recovery of the papaya pathogen was recorded on only cowpea, melon, and tomato. Resident populations of PVIc1R^r on melon and tomato after 14

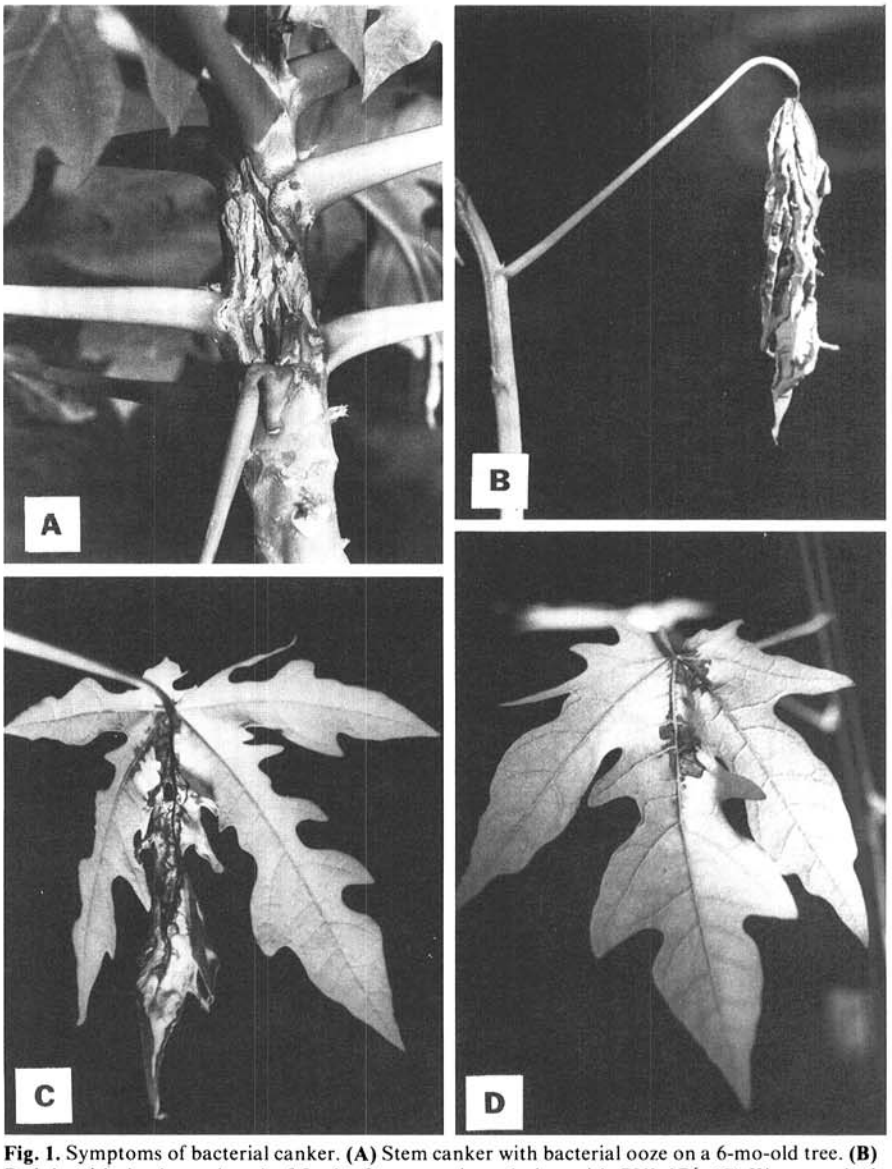

Fig. 1. Symptoms of bacterial canker. **(A)** Stem canker with bacterial ooze on a 6-mo-old tree. **(B)** Petiole with dead, pendant leaf 2 wk after spray inoculation with PVIc1R^r. **(C)** Water-soaked systemic leaf lesion on susceptible cultivar P.R. 6-65 1 wk after spray inoculation with PVIc1R^r. **(D)** Discrete necrotic lesions on resistant Barbados Dwarf cultivar 1 wk after spray inoculation with PVIc1R^r.

days increased from the 7-day sample period (Table 4).

Effects of various antibiotics, bactericides, and an antagonistic bacterium on symptom severity and disease spread. Evaluations of pathogen recovery, mean number of cankers per tree, and symptom severity made on inoculated and uninoculated trees indicated that no significant reduction in symptom severity or disease spread occurred as a result of five bimonthly preinoculation applications and 12 weekly postinoculation applications of the various chemical compounds ($P = 0.01$).

Recovery of the antagonistic bacterium from treated trees indicated that effective colonization of leaf surfaces did occur during the application schedule. However, no reduction in symptom development occurred with this treatment either.

Susceptibility of papaya cultivars to pathogen. Variations in susceptibility were observed for the same cultivars in both spray- and wound-inoculation trials. Least susceptible were the Barbados Dwarf 2X, Trinidad Pink, STT 683-1, and P.R. 10-65. The remaining 25 cultivars were very susceptible and not significantly different from one another (Table 1).

Disease resistance was manifested in spray-inoculated plants by a decrease in the number and severity of leaf lesions and stem cankers. The Barbados Dwarf 2X cultivar, as reported earlier (12), displayed a hypersensitive response to leaf infections, limiting them to discrete necrotic lesions as opposed to the large water-soaked lesions observed on susceptible cultivars (Fig. 1C,D).

Table 2. Biochemical, physiological, and cultural characteristics of the canker pathogens of papaya and related *Erwinia* spp.

Characteristic	Virgin Islands isolates[a]	Mariana Islands isolates[b]	*E. chrysanthemi*[c]	*E. carotovora*	*E. stewartii*[d]
Growth at 36 C[e]	+[f]	+	+	+	−
Pectate degradation[e]	−	v	+	+	−
Sucrose reduction[g]	+	+	v	−	v
Gelatin liquification[g]	−	−	+	+	−
Gas from glucose[g]	−	−	+	v	−
Blue pigment	+	+	+	−	−
Growth in 5% NaCl	+	v	−	+	nd
Sensitivity to erythromycin (50 μg)[g]	+	+	+	−	nd
Urease[e]	−	−	−	−	−
Flagella	−	+	+	+	−
Acid production from[e]					
Citrate	−	−	+	+	+
Dulcitol	v	+	−	−	−
Galacturonate	−	+	v	+	−
Glycerol	+	−	−	v	−
α-Lactose	−	−	−	+	−
Maltose	−	−	−	+	−
Melezitose	−	−	−	−	−
Palatinose	−	nd	−	−	nd
Proline	−	−	−	−	nd
Raffinose	−	−	v	+	+
Rhamnose	−	−	+	+	−
Salacin	+	−	+	v	−
Sorbitol	−	nd	+	+	+
Xylitol	−	−	−	−	nd
Xylose	+	+	+	+	+

[a] Ten isolates of the Virgin Islands pathogen were used in all tests.
[b] Data for Mariana isolates taken from Trujillo and Schroth (10).
[c] Two cultures each of *E. chrysanthemi* and *E. carotovora* var. *carotovora* were included as standards.
[d] Data for *E. stewartii* taken from Bergey's Manual (2).
[e] + = Positive, − = negative, v = variable, and nd = not done.
[f] By the methods of Schroth and Hildebrand (8).
[g] By the methods of Kelman and Dickey (6).

Table 3. Influence of free moisture (FM) on pathogen survival and symptom severity

Duration (hr)	Recovery of PVIc1R[r] (log cfu/g fr wt)		Symptom severity rating[a]
	With FM	Dry	
0	6.430	6.430	3.50[b]
4	5.991	4.000[c]	3.00
8	4.345	4.000	3.50
24	4.360	4.226	3.50
72	5.502	4.867	2.85

[a] Symptom severity ratings made 2 wk after inoculation on a scale of 0–4, where 0 = no stem cankers or leaf lesions, 1 = scattered leaf lesions, 2 = 1–10 lesions per leaf plus small stem cankers, 3 = >10 lesions per leaf and/or large girdling stem cankers, and 4 = plant dead.
[b] Values within column not significantly different ($P = 0.01$) according to analysis of variance.
[c] Value significantly different ($P = 0.01$) from FM treatment according to analysis of variance.

DISCUSSION

Bacterial canker of papaya is caused by an *Erwinia* sp. similar to the bacterium causing the Mariana and Java papaya diseases. The St. Croix isolates, however, possess a combination of physiological and biochemical characteristics that distinguish them from the Mariana isolates as well as other *Erwinia* groupings. Most notable is the absence of flagellae that, with the exception of *E. stewartii*, make this bacterium unique in the genus. As mentioned by Trujillo and Schroth (10), classification of these papaya pathogens by the pathovar system is impractical because it does not take into consideration bacteria that do not fit the characteristics of presently defined species. Species ranking of the St. Croix pathogen will be made by DNA homology matrix typing (9) after further study.

Observations of losses occurring from disease outbreaks show that this disease is most destructive on St. Croix during the short rainy seasons. Symptoms, however, may be observed throughout the entire year. Although rainfall disseminates the pathogen, free moisture following deposition to a susceptible site on the host does not increase pathogen survival or symptom severity. In fact, symptom severity appears to be decreased by 72 hr of free moisture. These findings indicate that the pathogen is well adapted to the semiarid climate of St. Croix and may provide an answer, in part, why this disease is not a significant problem on surrounding, wetter islands.

Under St. Croix conditions, the pathogen does not survive well in association with papaya roots or in decaying, diseased plant material, indicating that it is a transient soil inhabitant (3). The bacterium does survive for indefinite periods in the cankers and leaf infections of affected papaya trees and on the leaves of tomato and cantaloupe. Unlike the "Mariana decline" pathogen, the St. Croix bacterium has not been associated with the African snail (*Achotina fulica*) (10) or insect vectors.

Attempts to control this disease with bactericides, antibiotics, and the locally isolated antagonistic pseudomonad described in this study were not effective. Current trials using a tank mix of copper hydroxide and mancozeb (Dithane M-45) (4) also have shown this treatment inadequate for controlling this disease (*unpublished*).

Table 4. Pathogen recovery from nonhost plants

Plant species	Common name	Recovery of PVIc1R^r (log cfu/g fr wt) 1 Day	3 Days	7 Days	14 Days
Cajanus cajan	Pigeonpea	nd[a]	tftc	tftc	nd
Cenchrus sp.	Weed	2.700	tftc	tftc	nd
Citrus aurantifolia	Lime	2.000	tftc	tftc	nd
Cucumis melo	Cantaloupe	5.644	4.796	3.973	4.815
Euphorbia heterophila	Weed	3.023	3.099	tftc	tftc
Leucaena leucocophala	Tan tan	nd	tftc	tftc	nd
Lycoperscion esculentum	Tomato	nd	4.071	3.357	3.450
Manihot esculenta	Cassava	1.799	tftc	tftc	nd
Melochia pyramidata	Weed	1.958	2.133	tftc	tftc
Moringa oleifera	Horseradish	4.266	5.535	tftc	tftc
Musa sp.	Banana	2.000	tftc	tftc	nd
Nicotiana tabacum	Tobacco	2.903	2.333	tftc	tftc
Parthenium hystrophorum	Weed	2.000	tftc	tftc	nd
Passiflora sp.	Passionfruit	4.517	2.133	tftc	tftc
Portulaca oleracea	Purselane	2.032	tftc	tftc	nd
Sorghum halapense	Johnsongrass	2.133	tftc	tftc	nd
Teramnus labialis	Forage sp.	3.392	tftc	tftc	nd
Vigna sinensis	Cowpea	4.288	2.705	2.023	tftc

[a] nd = Not done, tftc = too few to count.

At present, the most effective control strategy for the canker disease is the use of resistant cultivars. The most promising to date is Barbados Dwarf, which in field trials is often the only cultivar that remains standing after the first year of production.

Another control strategy under investigation is the use of suitable "barrier crops" that do not support epiphytic populations of the pathogen, i.e., cassava, banana, and pigeonpea. Observations of small local farms where papayas are commonly intercropped with a wide variety of fruit and vegetable crops have shown a lower incidence of the canker disease than is found in monocultures.

The term St. Croix papaya decline applies to a variety of factors, acting alone or in concert, that affect the normal development of papaya. In St. Croix, papayas respond to a number of adverse conditions by premature abscission of leaves and cessation of apical growth, which result in a symptom called "pencil-point." This condition has been observed in trees suffering from viral infections, root rot caused by *Pythium butlerii*, bacterial canker, water stress, and nutrient deficiencies caused by low nitrogen levels and/or high soil pH. Greasy spots on papaya stems caused by viral infections are often misdiagnosed by growers as bacterial cankers. Leaf spots caused by *C. cassicola* and the canker bacterium are very different but may be found on the same leaf and are frequently misdiagnosed. *C. cassicola* is also a common secondary invader of cankers caused by the bacterium. In this study, however, it was never isolated from fresh diseased tissues.

Because of the confusion surrounding the term St. Croix papaya decline and the multiple conditions it may represent, I propose that it be dropped from further use and replaced with "bacterial canker of papaya."

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