Variation in the Distribution of Citrus Ringspot and Psorosis Viruses Within Citrus Hosts

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A portion of this work was conducted while the senior author was on study leave at the U. S. Horticultural Research Laboratory in Orlando, Florida, under a cooperative agreement between the U. S. Department of Agriculture, the Division of Plant Industry of the Florida Department of Agriculture and Consumer Services, and the Texas A&I University Citrus Center.

The technical assistance of S. Villarreal and R. Whidden is gratefully acknowledged.

Accepted for publication 22 August 1978.

ABSTRACT

TIMMER, L. W., and S. M. GARNSEY. 1979. Variation in the distribution of citrus ringspot and psorosis viruses within citrus hosts. Phytopathology 69: 200-203.

Some isolates of citrus ringspot virus (CRSV) in Texas were inconsistently graft-transmitted from twig bark of infected mature grapefruit trees to greenhouse-grown citrus indicator seedlings. These isolates usually were transmitted with inoculum from twigs on which foliar symptoms were apparent or from the psorosis-like bark lesions on the primary branches of CRSV-infected trees. In two grapefruit trees with small, initial bark lesions on the primary branches, the virus was transmissible only from the portions of the branches with lesions. Mechanical transmission of some Texas isolates from symptomatic young citrus leaves produced abundant local lesions on *Chenopodium quinoa*, whereas few or no lesions were produced when symptomless leaves were used. Graft transmission of one Florida isolate of CRSV by leaf pieces to citrus seedlings and by mechanical transmission to *C. quinoa* from symptomless young leaves failed, whereas transmission from symptomatic leaves was successful. This isolate was irregularly distributed even within symptomatic leaves, and could not be transmitted to *C. quinoa* from symptomless areas of such leaves. Citrus psorosis virus and two CRSV isolates from Florida were recovered more consistently from symptomless tissue. Although several citrus hosts and *C. quinoa* are excellent indicator plants for CRSV, indexing methods used for other citrus viruses have been useful only to confirm infections that were already suspected from visual inspection.

Citrus ringspot virus (CRSV) was described originally from California (18), and, subsequently, CRSV and apparently related viruses have been reported from other citrus areas (1,3,6,9,13,14). The necrotic strain of CRSV described from Texas produces flecking, chlorotic blotches, and ringspots on leaves; blotches, spots, and gum deposits on fruit; and necrosis of young shoots (13). This isolate produces a shock reaction in graft-inoculated seedlings and is associated consistently with bark lesions in field trees. Garnsey (3) and Garnsey, et al (6) described mechanically transmissible strains of CRSV from a Florida navel orange (*Citrus sinensis* (L.) Osb.) tree and from a grapefruit (*C. paradisi* Macfad. 'Star Ruby') tree imported from Texas into Florida without authorization. All of the above isolates, including the California isolate (18), have been transmitted to *Chenopodium quinoa* Willd. mechanically and produce local lesions on that host (S. M. Garnsey and L. W. Timmer, *unpublished*). Florida and Texas isolates of CRSV produce similar symptoms on a wide range of herbaceous and citrus indicator plants, but individual isolates are distinguishable by minor differences in host range and symptomatology (16). Psorosis A virus produces leaf-flecking symptoms, a shock reaction in graft-inoculated seedlings, and is associated with scaly-bark symptoms on the trunks of affected trees. On the basis of symptomatology, psorosis A virus and CRSV may be related (14); but psorosis A virus has not been transmitted mechanically.

Most citrus viruses are detected in candidate budwood-source trees by graft inoculation of specific indicator plants (2). Generally, sampling of a few bud sticks from various portions of the candidate tree and inoculation of three to six seedlings is sufficient to detect the presence of the virus. In previous studies of CRSV (6,13,15), trees with bark lesions did not always index positive on susceptible indicator seedlings and the virus appeared to be absent from symptomless tissue. The purpose of the present study was to test more fully the effectiveness of indexing procedures for detection of CRSV and psorosis viruses and to determine the pattern of their distribution in citrus hosts.

00031-949X/79/000034$03.00/0

MATERIALS AND METHODS

Plant materials. The following citrus hosts of CRSV were grown from seed: sweet orange (*C. sinensis* (L.) Osbeck 'Pineapple' and 'Madam Vinous'), Mexican lime (*C. aurantifolia* (Christm.) Swingle), sour orange (*C. aurantium* L.), and grapefruit (*C. paradisi* Macfad. 'Duncan' and 'Hudson'). A clonal, virus-free selection (cultivar Arizona 861) of Etrog citron (*C. medica* L. var. *ethrog* Engl.) was propagated by rooted cuttings. *Chenopodium quinoa* was grown from seed and provided with supplemental light to maintain the vegetative state.

All plants were grown in evaporatively-cooled and partly-shaded greenhouses in sterilized potting media and were regularly fertilized and sprayed to maintain vigorous growth. Indexing usually was conducted in the cooler months when the daily high temperatures in the greenhouse did not exceed 27 C. Symptom expression of CRSV did not, however, appear to be as dependent on cool temperature as was that of psorosis A virus.

Graft and mechanical transmission. For routine indexing of CRSV, four bud sticks were collected from separate locations on a tree and were selected at random without regard to the presence or absence of symptoms. One bark patch from each bud stick was grafted into each of three to six 0.5 cm diameter indicator seedlings. For transmission from lesion or nonlesion bark from the trunk or scaffold branches, triangular bark patches 2 cm long and 1 cm wide at the base were grafted into 1 cm diameter indicator seedlings. Leaf-piece grafts were made by the double-bladed knife technique (5). Seedlings were cut back immediately after inoculation and periodically thereafter to force new growth. Symptoms usually appeared in the first flushes of growth, but symptomless plants were kept at least 6 mo and inspected for symptoms. Citrus ringspot

virus produced severe shoot necrosis followed by flecking, chlorotic spots, and ringspots on subsequent flushes on all species except citron on which it produced only mild chlorotic blotches and etching (3,6,13,16). Fruit peduncles were inoculated by grafting two bark patches just above fruit (2–3 cm in diameter) on mature field trees. Chlorotic spots and ringspots typical of CRSV (13) usually appeared within 3 mo.

Inoculum for mechanical transmission was prepared by triturating young leaf tissue of citrus in a mortar and pestle in 0.05 M Tris (tris(hydroxymethyl)aminomethane) buffer, pH 8.0, containing 0.5% 2-mercaptoethanol (1 g of tissue/10 ml of buffer). Inoculum was applied by cotton swab to *C. quinoa* leaves predusted with 22-μm corundum. Local lesions appeared 3–6 days after inoculation.

Isolates. The Texas isolates derived from individual trees are listed by number in Table 1. Isolate Txr-1 from tree 2-15-2 was the isolate described previously as the necrotic strain of CRSV (13). All of the Texas isolates of CRSV except Txr-6, Txr-10, and Txr-13, which were not tested and are no longer available, produced local lesions when mechanically transmitted to *C. quinoa* (Table 1).

Florida isolates used were: CRSV-2(ZN), from a Zatima navel orange tree propagated in Florida from budwood originally imported from Algeria (3); CRSV-4(FSR), from a Star Ruby grapefruit tree imported into Florida from Texas without authorization (6); and CRSV-5(FN), a previously undescribed isolate from an old-line Florida navel orange tree (Table 2). Psorosis virus isolate P3 was derived from a Pineapple sweet orange seedling from Florida which was naturally infected by unknown means; psorosis virus isolate P1 was from infected field sweet orange trees in Florida which were propagated from infected budwood. Neither of the psorosis virus isolates was mechanically transmissible to *C. quinoa*. The psorosis virus isolates produced flecking on the young leaves of sweet orange, but we have no evidence that either psorosis virus isolate was capable of producing bark lesions. The possibility remains that the psorosis virus isolates may be isolates of concave gum virus (CGV) or mixtures of psorosis A virus and CGV (14).

Experimental procedure. Field trees in Texas were indexed periodically from 1971–1975 by utilizing various indicator plants and the procedures described above (Table 1). All trees 12 yr of age or older had bark lesions when indexing began. None of the 2 yr old trees had bark lesions when indexing began, but all occasionally had shown foliar symptoms of CRSV on some branches. However, as these trees matured, all except trees 2-14-8 and 3-7-6 developed bark lesions (Table 1).

For in-tree virus distribution studies, a single Mexican lime indicator seedling was inoculated with three bark patches from each sample site on the scaffold branches of two 15 yr old grapefruit trees, 2-19-13 (Fig. 1) and B-5-13-24. Seedlings that were symptomless after the first inoculation were reinoculated 3 mo later with bark from the same sites. The experiments with both trees were repeated using Pineapple sweet orange indicator seedlings.

The virus concentrations in symptomless and symptomatic young leaf tissue of greenhouse-grown sour orange seedlings graft-inoculated with CRSV-2(ZN), CRSV-4(FSR), and CRSV-5(FN) were compared in a randomized, complete block experiment with six replications on half-leaves of *C. quinoa*.

To determine the distribution of virus in greenhouse-grown sour orange seedlings graft-inoculated with CRSV-2(ZN), CRSV-4(FSR), and CRSV-5(FN), a 10-mm diameter disk was cut from each symptomless and symptomatic leaf tissue area (Fig. 2), triturated in Tris buffer, and rubbed on six half-leaves of *C. quinoa* in a randomized complete block experiment. The distribution of each isolate was determined in a separate experiment.

Leaf-piece grafting was used in a similar experiment to determine the distribution of CRSV-2(ZN), CRSV-4(FSR), CRSV-5(FN), and P3 psorosis in citrus hosts (Table 2, Expt. 1). A single leaf piece from each of the various types of tissue was grafted into separate Duncan grapefruit indicator seedlings. Symptomless tissue was collected from leaves similar to those illustrated in Fig. 2; viz, from symptomless leaves on symptomless shoots, from symptomless leaves on shoots with symptoms, and from symptomless areas on symptomatic leaves.

The presence of psorosis virus in symptomless and symptomatic

TABLE 1. Results of indexing of field trees for the presence of citrus ringspot virus. Twig inoculum collected at random was compared with that selected from symptomatic branches[a]

Tree no.	Isolate no.	No. positive / no. inoculated: Randomly selected inoculum[b]	Symptomatic inoculum[c]
Nucellar 2 to 6-yr-old Hudson grapefruit:			
2-15-2	Txr-1	3/3[d], 6/10[e]	6/6[d]
2-2-9	Txr-2	0/3[d], 0/6[d], 0/10[e], 3/3[f], 0/3[g]	3/3[d]
2-14-8	Txr-3	2/3[d], 3/3[f], 2/3[g]	8/10[e]
5-7-1	Txr-4	6/6[d], 3/3[f], 1/3[g]	7/7[e]
3-7-6	Txr-5	3/3[d], 3/3[d]	...
Nucellar 12 to 16-yr-old Webb Redblush grapefruit:			
B-5-3-3	Txr-6	4/6[d], 1/3[g], 3/10[e]	...
B-5-6-14	Txr-7	0/6[d], 0/6[d], 0/3[f], 2/10[e]	5/5[h]
B-5-16-1	Txr-8	0/6[d], 0/6[d], 0/3[f]	5/6[h]
B-5-18-9	Txr-9	3/3[d], 3/3[f], 1/10[e], 1/6[d]	2/6[h]
2-30-7	Txr-10	1/6[d], 0/3[g]	7/10[e]
2-19-13	Txr-11	0/3[d], 4/6[d]	10/12[h]
Old-line 25 to 30-yr-old Webb Redblush grapefruit:			
D-3-2-1	Txr-12	3/3[d], 4/6[d]	6/6[d]
C-3-3-11	Txr-13	3/3[d], 3/3[f], 0/20[e]	...
D-3-1-17	Txr-14	3/3[d], 3/3[f]	...

[a] Summary of tests conducted at various times from 1971-1975 with several indicator plants.
[b] Collected from four sides of field trees without regard to presence or absence of symptoms.
[c] Collected from twigs with foliar symptoms or from principal branches with bark lesions.
[d] Indexed on sweet orange seedlings by grafting bark patches from twigs.
[e] Indexed by inoculating grapefruit peduncles with bark patches from twigs.
[f] Indexed on Mexican lime seedlings by grafting bark patches from twigs.
[g] Indexed on rooted cuttings of Etrog citron by grafting bark patches from twigs.
[h] Indexed on sweet orange seedlings by grafting lesion-bark inoculum.

TABLE 2. The occurrence of citrus ringspot virus (CRSV) and psorosis virus in symptomless and symptomatic tissue as determined by leaf-piece graft inoculation of Duncan grapefruit (Expt. 1) or sweet orange (Expt. 2) seedlings

Expt. no. and isolate	Tissue source	Symptoms present	No. positive / no. inoculated
Experiment 1[a]:			
CRSV-4(FSR)	Duncan grapefruit	+	2/2
	Duncan grapefruit	−	0/6
CRSV-5(FN)	Sour orange	+	1/1
	Sour orange	−	3/4
CRSV-2(ZN)	Duncan grapefruit	−	2/2
	Sour orange	−	2/2
P3 (psorosis)	Mexican lime	−	2/2
	Duncan grapefruit	−	2/2
Experiment 2:			
P-1 (psorosis)	Sweet orange (tree 1)	+	1/1[b]
	Sweet orange (tree 1)	−	4/4[b]
P-1 (psorosis)	Sweet orange (tree 2)	+	1/1[b]
	Sweet orange (tree 2)	−	2/3[b]
P-1 (psorosis)	Sweet orange (tree 2)	+	1/1[c]
	Sweet orange (tree 2)	−	4/5[c]

[a] Each seedling was inoculated with a single leaf piece.
[b] Each seedling was inoculated with three leaf pieces each from a separate leaf on a single shoot.
[c] Each seedling was inoculated with three leaf pieces from a single leaf; all leaves taken from a single shoot.

tissue of field-grown sweet orange trees infected with isolate P1 was determined by leaf-piece grafting (Table 2, Expt. 2). One symptomatic and four symptomless shoots and one symptomatic and three symptomless shoots were collected from trees 1 and 2, respectively. Three leaf pieces from separate leaves on each shoot were grafted into each sweet orange seedling (one seedling/shoot). A single shoot with one symptomatic leaf from tree 2 was indexed for psorosis virus by grafting three leaf pieces from each leaf into single sweet orange seedlings.

RESULTS

Indexing and internal distribution of CRSV in field trees in Texas. Repeated indexing of field trees in Texas over a 4 yr period indicated that CRSV was distributed erratically and difficult to detect in some trees, but was uniformly distributed and readily detectable in others (Table 1). Most of the 2 to 6 yr old trees consistently indexed positive, regardless of the indicator used. However, tree 2-2-9 frequently indexed negative, except when symptomatic tissue was used as inoculum (Table 1). The virus was difficult to detect in naturally infected 12 to 16 yr old nucellar trees. In tree B-5-16-1, for example, CRSV was detected only when lesion-bearing bark was used as inoculum. However, CRSV was detected readily in 25 to 30 yr old Webb Redblush grapefruit trees which probably had been infected during propagation (Table 1).

In addition, six old-line, 25 to 30 yr old Webb Redblush grapefruit trees, which had been originally budded from certified psorosis-free sources but which had subsequently developed bark lesions, were indexed on single sweet orange seedlings using inoculum from symptomless twigs. Only one indexed positive. Also, nine nucellar Webb Redblush grapefruit trees were indexed using twig or lesion-bark inoculum on three Mexican lime or sweet orange seedlings. With twig inoculum, five of the sources produced 100% infection, two 67%, one 33%, and one 0%. With lesion-bark inoculum, six sources produced 100% infection, two 67%, and one 33%.

In the studies of the distribution of the virus in mature field trees with bark lesions, CRSV was recovered only from the branch with the bark lesion and from one adjacent scaffold limb on tree 2-19-13 (Fig. 1). The virus was not detected in any of the four sites indexed on a branch directly above the lesion. Tree B-5-13-24, which had a bark lesion on the main trunk about 3 m above the ground, was indexed similarly. The virus was recovered from the bark lesion and from the main trunk 2 m below the lesion, but was not recovered from two other scaffold branches, or from twigs from two locations on the tree.

The absence of detectable virus from symptomless tissue was confirmed by indexing twig inoculum from the tree illustrated in Fig. 1 on six seedlings of tangor (*C. reticulata* Blanco × *C. sinensis* 'Dweet'), a more sensitive indicator for leaf-flecking viruses (12). All were negative. Madam Vinous sweet orange seedlings that had been inoculated with twig inoculum from the tree in Fig. 1 and remained symptomless were challenge inoculated with bark lesion inoculum from a CRSV-infected tree. These seedlings and three seedlings previously grafted with bark patches from a healthy field tree and challenged with lesion-bark inoculum developed typical bark lesions. In contrast, three other seedlings previously graft-inoculated with twig inoculum of CRSV and showing foliar symptoms were protected against formation of bark lesions when challenged with bark lesion inoculum.

Mechanical transmission of the CRSV isolates from trees 2-15-2 (Txr-1) and 2-2-9 (Txr-2) (Table 1) to *C. quinoa* was attempted with symptomatic and symptomless young leaf tissue of graft-inoculated, greenhouse-grown seedlings. Symptomatic tissue from satsuma (*C. reticulata* 'Owari'), tangelo (*C. reticulata* × *C. paradisi*

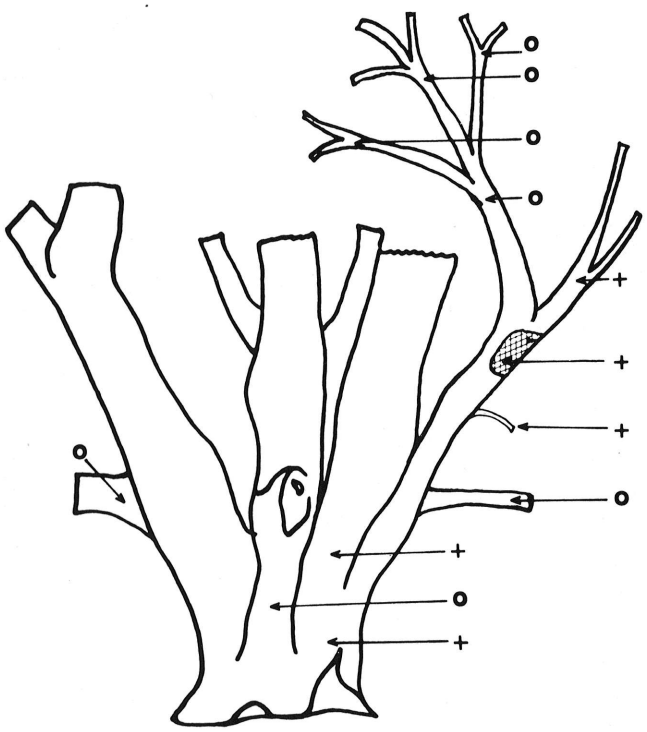

Fig. 1. Distribution of citrus ringspot virus in grapefruit tree 2-19-13 with a small bark lesion (cross hatched area). Plus (+) indicates a location from which the virus was graft-transmitted to indicator seedlings and (0) indicates an area from which no transmission was obtained.

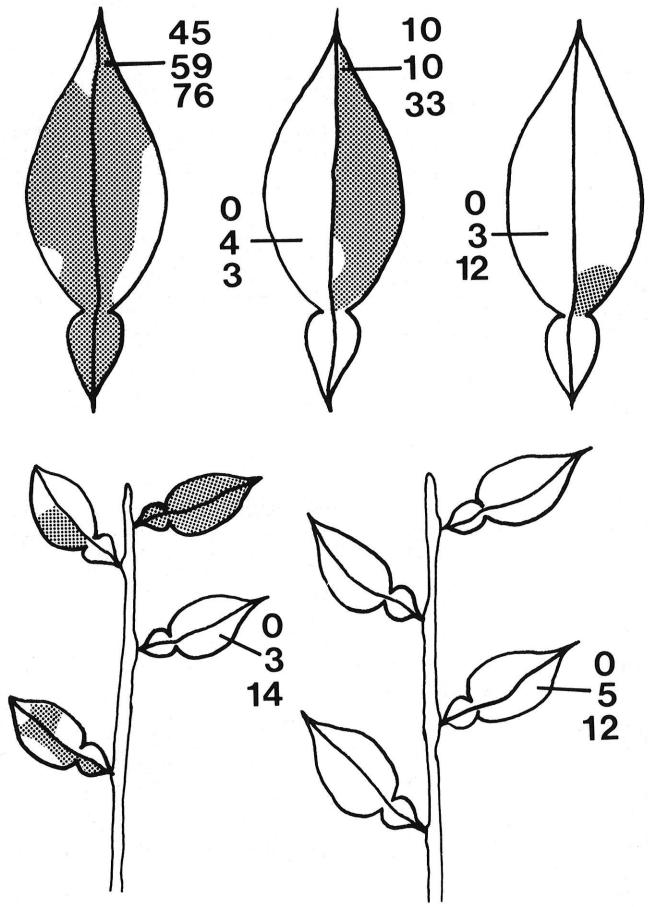

Fig. 2. Distribution of citrus ringspot isolates in infected sour orange leaves. Stippling indicates the areas with visible symptoms. Assays were conducted on separate seedlings for each isolate. The diagrams presented are indicative of the symptom distribution, but distribution patterns were not identical for all isolates. Figures indicate the number of local lesions per half-leaf when tissue from these areas was ground and rubbed onto *Chenopodium quinoa*. In each case, the top number is for isolate CRSV-4(FSR), the middle number for CRSV-5(FN), and the bottom number for CRSV-2(ZN).

'Orlando'), and Mexican lime infected with isolate Txr-1 produced abundant local lesions on *C. quinoa*, whereas symptomless tissue from tangelo and satsuma produced few local lesions; symptomless tissue from lime produced none. Symptomatic tissue of sour and sweet orange infected with isolate Txr-2 (tree 2-2-9) produced abundant local lesions on *C. quinoa*, whereas symptomless tissue produced none.

Internal distribution of Florida isolates of CRSV and psorosis virus. Assay of leaf tissue of sour orange seedlings inoculated with CRSV-2(ZN), CRSV-5(FN), and CRSV-4(FSR) produced 62, 76, and 85 local lesions per half-leaf of *C. quinoa*, respectively, when symptomatic tissue was used and 24, 13, and 0 lesions per half-leaf, respectively, when symptomless tissue was used. Assay of various types of symptomless tissue of sour orange seedlings with CRSV-2(ZN) and CRSV-5(FN) likewise indicated that these isolates consistently were present in symptomless tissue even when entire shoots were free of symptoms. In contrast, CRSV-4(FSR) was not recovered from any symptomless tissue, even from that adjacent to symptomatic areas on the leaf (Fig. 2). In other tests, CRSV-4(FSR) occasionally was recovered from very young symptomless tissue. Since CRSV symptoms often do not appear until the leaves are nearly fully expanded, these leaves probably would have developed symptoms, if they had been allowed to expand.

Leaf-piece grafting consistently transmitted CRSV-2(ZN), CRSV-5(FN), and psorosis virus P3 from symptomless tissue (Table 2, Expt. 1). In contrast, CRSV-4(FSR) could only be graft transmitted from symptomatic tissue. Psorosis virus from field sources was transmitted by leaf-piece grafting from symptomless leaves in a high percentage of the attempts, and always was transmitted from symptomatic leaves (Table 2, Expt. 2).

DISCUSSION

Because of the uneven distribution pattern of some CRSV isolates in citrus trees, standard indexing methods are unreliable for detection of the virus in field trees. In the present study, trees with bark lesions sometimes indexed negative when twig inoculum was used. Such trees repeatedly indexed negative when tested several times over a 4-yr period and the virus probably would be even more difficult to detect in trees without bark symptoms. The virus probably can be detected best in candidate budwood-source trees by frequent field inspection of trees for foliar, fruit, or bark symptoms. Citrus indicators, such as grapefruit, sweet orange, sour orange, and Mexican lime, or *C. quinoa* then can be used for confirmation of suspected symptoms of CRSV in field trees.

Although viruses commonly are distributed throughout most tissues of infected plants, other cases of erratic distribution pattern have been reported. For example, graft transmission of sharka virus from infected peach trees was erratic and caused problems in the indexing of potential parent trees (8). Tobacco ringspot virus was distributed unequally in cherry trees (17). In many mosaic diseases, the dark-green areas of leaves frequently contain no infectious virus, or considerably less virus, than the yellow areas (7,11). Tatter-leaf (citrange stunt) virus in the citranges is restricted to the areas of the leaf with visible symptoms (4). Similarly, at least some strains of CRSV are limited to the parts of the plant that show symptoms.

Psorosis virus and some strains of CRSV consistently are present in symptomless tissue, although the titer of CRSV in symptomless tissue usually is low. Uneven distribution, however, appears to be a common trait especially of Texas isolates of CRSV. The Florida isolate of CRSV that was not uniformly distributed, CRSV-4(FSR), may have been imported from Texas (6). Other leaf-flecking viruses, probably related to CRSV (14), also may be distributed unequally in citrus hosts. In Argentina, Pujol and Beñatena (10) obtained 100% transmission of a naturally spread form of psorosis virus from symptomatic tissue but were unable to transmit the virus by grafting symptomless tissue. Planes and Martí (9) indicated that the virus causing 'leaf variegation with ringspots' was present only in symptomatic tissue. In grapefruit trees graft-inoculated with impietratura, the virus does not become systemic in the trees but often is localized in the inoculated branch (M. Bar-Joseph, *personal communication*).

The uneven distribution of some isolates of CRSV in citrus hosts may explain discrepancies in symptomatology, host range, and cross-protection tests conducted on what appear to be similar viruses from several citrus areas (14). Inoculation of a citrus host range with symptomless tissue could lead to erroneous conclusions about the hosts susceptible to the virus. For example, Planes and Martí (9) reported no symptoms on sour orange inoculated with 'leaf variegation with ringspots' virus and Broadbent (1) found no symptoms on sour orange or Mexican lime inoculated with Monak psorosis B virus. However, all Florida and Texas isolates of CRSV produce severe symptoms on these species. Cross-protection studies with erratically distributed CRSV isolates may be valid only when challenge inoculations are made on symptomatic shoots of protected plants.

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