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Evidence That Xyloporosis Virus Does Not Pass Through Seeds of Palestine Sweet Lime

TRANSMISSION OF CITRUS VIRUSES through seed has not been demonstrated and many citrus clones have been freed of viruses by growing nucellar seedlings (6). There is, however, a single report based on observations that the causal agent of xyloporosis was apparently transmitted through 66% of 30 Palestine sweet lime (*Citrus limettioides* Tanaka) seed (2).

Grant *et al.* (4) noted that false symptoms of xyloporosis accompanied purple scale (*Lepidosaphes beckii* Newm.) infestations on sweet lime seedlings. Childs (3) noted that the experimental evidence of seed transmission of xyloporosis through sweet lime seed was less adequate than was desirable (3). Nevertheless, as Childs (3) pointed out, the possibility of seed transmission can scarcely be ignored because of its importance to budwood-certification programs.

Transmission of viruses through seeds of other tree fruits is known. Avocado sunblotch virus, for example, is seed-transmitted from some Mexican-race trees that are symptomless virus carriers (5).

The purpose of the present trial was to follow up Childs' lead and determine whether xyloporosis virus is seed-transmitted from xyloporosis-infected Palestine sweet lime trees to their seedlings. In this report, xyloporosis is considered to be identical with cachexia.

Methods and Materials

ORLANDO TANGELO SELECTED AS INDICATOR PLANT.—Orlando tangelo (*C. paradisi* Macf. x *C. reticulata* Blanco) was selected as an index plant for xyloporosis, because xyloporosis symptoms develop more rapidly and reliably in it than in sweet lime indicator plants. Calavan *et al.* (1) also

preferred Orlando tangelo to sweet lime as a xyloporosis-indicator plant. Childs (2) noted no seed transmission of xyloporosis virus in Orlando tangelo.

SEED COLLECTION.—Sweet lime plants of several varieties were included in a variety collection set out in November, 1953, at Rio Farms, Inc., Monte Alto, Texas. In 1957, the Palestine sweet lime trees showed characteristic xyloporosis symptoms (pits-and-pegs, brown-stained bark, healed-over bark cracks) and the trees were stunted. Butwal and Columbian sweet lime trees on Cleopatra mandarin rootstock in the same planting showed no xyloporosis symptoms and made vigorous, normal growth.

In November of 1957, several hundred seed were collected from the Palestine sweet lime trees that showed xyloporosis and their seedlings were grown during 1958.

INDEXING PROCEDURE.—On September 12, 1958, scions from various sweet lime sources listed in Table 1 were side-grafted 4 inches above the bud-union to one-year-old old-line Orlando tangelo indicator trees growing on Cleopatra mandarin rootstock. As shown in Table 1, the sweet lime scions were from 3 general sources: (a) 3 apparently healthy 8-year-old trees, (b) 5 8-year-old seed-source trees with xyloporosis symptoms, and (c) 113 different 10-month-old seedlings of the 5 xyloporosis-infected Palestine sweet lime trees in group (b). About a month later, the Orlando test trees were inspected and all trees on which the graft had failed were destroyed. The remainder were left in the nursery row until termination of the experiment.

The grafted scions never made significant growth; so the tops of the test trees remained old-line Orlando tangelo, with a fragment of living sweet lime tissue grafted to the trunk.

The Orlando tangelo virus-indicator trees were inspected for xyloporosis symptoms in October, 1961, slightly more than 3 years after inoculation. Trees with characteristic brown-stained inner bark and pitted wood were considered to be infected with xyloporosis. In January, 1962, the test plants were badly frozen by temperatures that reached 12°F in nearby areas. Some trees were killed back to the bud-union with Cleopatra mandarin rootstock and were dug but the remainder were pruned and continued in the test. In April, 1963, 4 years and 7 months after inoculation, the Orlando tangelo trees were again inspected for xyloporosis symptoms.

Results

When the Orlando tangelo test plants were examined 3 years after

TABLE 1. OCCURRENCE OF XYLOPOROSIS SYMPTOMS IN ORLANDO TANGELO TREES IN RELATION TO SOURCE OF SCIONS WITH WHICH THEY WERE GRAFTED

Source of scions	Orlando tangelo trees with xyloporosis symptoms[a]	
	After 3 years	After 4 years and 7 months, subsequent to severe freeze
Apparently healthy 8-year-old control trees:		
Columbian sweet lime 16-1	0/5	0/3
Columbian sweet lime 16-2	0/5	0/4
Butwal sweet lime 16-7	0/5	0/5
Total	0/15	0/12
Control 8-year-old Palestine sweet lime trees with xyloporosis symptoms:		
PSL 16-13	2/5	1/1
PSL 16-14	4/5	2/2
PSL 15-6	3/5	1/1
PSL 15-2	4/5	1/1
PSL 15-7	4/5	2/2
Total	17/25	7/7
10-month-old seedlings of indicated xyloporosis-infected trees:		
PSL 16-13	0/40	0/16
PSL 16-14	0/12	0/7
PSL 15-6	0/33	0/19
PSL 15-2	0/13	0/5
PSL 15-7	0/15	0/9
Total	0/113	0/56

[a]Numerator=number of trees with symptoms; denominator=number of trees in group.

grafting (Table 1) no symptoms were present, indicating that no seed transmission of xyloporosis virus had occurred in 113 seedlings from the 5 xyloporosis-infected Palestine sweet lime trees. Fifty-six of the test trees survived a severe freeze and 19 months later were still free of xyloporosis symptoms. Orlando tangelo test plants of the same series inoculated with xyloporosis virus from the seed-source trees developed characteristic xyloporosis symptoms, indicating that the mother plants were infected and that the incubation period in the test plants was sufficient.

Discussion

Virologists have had difficulty in determining the presence of xyloporosis virus in sweet lime seedlings. In this work, sweet lime seedlings were grown from 5 known infected Palestine-sweet-lime mother trees and were indexed on Orlando tangelo, a better indicator plant for xyloporosis than sweet lime, to determine the presence of the virus. Similar Orlando test plants inoculated with xyloporosis virus from the seed-source mother trees as controls developed symptoms, indicating that the incubation period was sufficient.

The fact that no seed transmission occurred in these tests indicates that Childs' report of transmission in 1956 should be explained on some other basis. Since the symptoms reported by Childs were mild, they might have been confused with the results of scale injury such as described by Grant *et al.* (4).

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