A BUD-UNION DISORDER OF CALAMONDIN TREES IN CALIFORNIA[1]

L. G. Weathers and E. C. Calavan[2]
University of California Citrus Experiment Station,
Riverside, California

INTRODUCTION

The calamondin, *Citrus mitis* Blanco, belongs to the mandarin group of citrus, or is very closely related to it (*1, 8, 10*). It is probably a hybrid of *C. reticulata* var. *austera* ? × *Fortunella* sp. ? and therefore should not be considered a valid species (*8*). Calamondin is widely grown in the Philippine Islands and to some extent in Hawaii and the United States. Calamondin has considerable merit as an ornamental for several reasons: It is cold-resistant and very easy to grow. The tree tends to grow tall and columnar in form with dense, bright-green foliage. It is a prolific bearer and is generally in flower or fruit the year round. The flowers are white and very fragrant. The fruits are orange-colored, about 1 inch in diameter, and highly prized for marmalade. The juice makes an acceptable acid drink.

A bud-union disorder affecting calamondin was first called to our attention in 1953 in a citrus variety collection in Santa Barbara, California. Affected trees were found on rootstocks of grapefruit, *Citrus paradisi* Macf.; sweet orange, *C. sinensis* (Linn.) Osbeck; and Troyer citrange, sweet orange × *Poncirus trifoliata* (Linn.) Raf. Subsequently, the disorder was found on calamondin growing on *P. trifoliata;* sour orange, *C. aurantium* Linn.; and lemon, *C. limon* (Linn.) Burm., in other citrus areas in California.

The number of trees affected with this disorder is unknown, but in surveys conducted to date almost all calamondin trees over four years old and on roots other than their own show this abnormality. Calamondin trees growing on their own roots, either as seedlings or cuttings, show little or no evidence of disease.

A similar bud-union disorder of both old-line and nucellar-line Red Blush grapefruit and Valencia orange trees on calamondin and other kumquat hybrid rootstocks has been described in Texas (*3, 6, 7*). Many other instances of the failure of calamondin as a rootstock for various citrus varieties have been recorded and are reviewed by Olson (*7*) and Webber (*10*).

A bud-union disorder with Nippon orangequat, *Citrus reticulata* × (*Fortunella japonica* [Thunb.] Swing × *F. margarita* [Lour.] Swing.), on sweet orange rootstock, closely resembling the calamondin bud-union disorder, has been noted in California. Similar bud-union abnormalities have been observed with Eustis limequat, *F. japonica* × *C. aurantifolia* (Christm.) Swing., on Sunshine tangelo, grapefruit × *C. reticulata*, and sour orange rootstocks (*7*); Eureka lemon on *Poncirus trifoliata* and Troyer citrange rootstocks (*9*); sweet orange on Rough lemon rootstocks (*2, 4, 5*); and Pera sweet orange on *P. trifoliata* rootstocks (*4*).

In preliminary observations it was determined that fungi or bacteria had no causal

[1] Paper No. 1110, University of California Citrus Experiment Station, Riverside, California.
[2] Assistant Plant Pathologist and Associate Plant Pathologist, respectively, Citrus Experiment Station, Riverside.

relation to the calamondin bud-union disorder. It was also considered unlikely that it might be the result of stress of climatic influence or nutritional deficiencies because of the wide range of conditions under which it occurred. The stionic reaction exhibited by the disorder suggested that some type of uncongeniality between scion and rootstock or a virus infection might be the cause. Accordingly, experiments were started in 1954 to determine whether the calamondin bud-union disorder is caused by a virus.

SYMPTOMS

Affected calamondin trees can generally be detected by their conspicuous top symptoms, which usually become apparent within the second or third year from the time the trees are budded and become more noticeable with additional time. The first symptoms are a suppression of new flushes of growth, followed by a light-green color or chlorosis associated with a general flaccid appearance of the leaves (fig. 1). Severely affected trees die so rapidly that the leaves wilt and dry while still green in color. Most of the trees blossom heavily, however, and usually set a heavy crop of fruit, then wilt very slowly. The leaves turn yellow and drop. Many such trees die, but those less severely affected generally continue to live indefinitely in a state of reduced vigor (see fig. 1) or until killed by overproduction, pests, or other factors of stress. Twig dieback is very common on declining trees.

The bud unions of affected calamondin trees reveal a marked abnormality in the wood and bark, which appears to precede the onset of top symptoms. Bud-union symptoms apparently vary only slightly with different rootstocks. A horizontal groove appears in the wood at the union. The bark may split horizontally at that point and pull apart. Gum accumulates in the bark and in the groove in the wood (fig. 2).

The understock roots of affected calamondin trees are always damaged. The small

Fig. 1. Calamondin trees on sour orange rootstock two and one-half years after budding: (left) declining tree following development of bud-union disorder; (right) normal tree prior to development of bud-union disorder (height of markers 3 feet). (Photographs by R. G. Platt.)

rootlets are usually dead by the time symptoms appear in the top. The injury to the roots appears to follow the breakdown of tissue at the bud union. Treatment with iodine showed starch depletion in the rootstocks of affected trees. Destruction of the phloem at the bud union apparently restricts the translocation of carbohydrates from the leaves to the roots and results in starvation and death of feeder roots. A root system so injured fails to function adequately and thus causes the tree to wilt. Affected roots die progressively from their tips toward the trunk and may involve larger roots. The degree of wilting and rate of death seem to be proportional to the amount of root injury.

EXPERIMENTAL METHODS AND RESULTS

Transmission Tests with Seedlings. In this test of the transmission of the disorder, scions were taken from severely affected calamondin trees and indexed for virus by inserting the scions into 5 to 10 potted seedlings each of sweet orange, sour orange, calamondin, West Indian lime (*Citrus aurantifolia* [Christm.] Swing.), Orlando tangelo, Sunshine tangelo, Rangpur lime, and Palestine sweet lime. The scions were not allowed to grow. Equal numbers of seedlings were left uninoculated to serve as checks. At frequent intervals after inoculation the seedlings were cut back to force additional flushes of foliage for the purpose of leaf-symptom expression.

Inspection over a three-year period revealed no leaf, bark, or wood symptoms of any kind in any of the inoculated seedlings of sweet orange, sour orange, calamondin, West

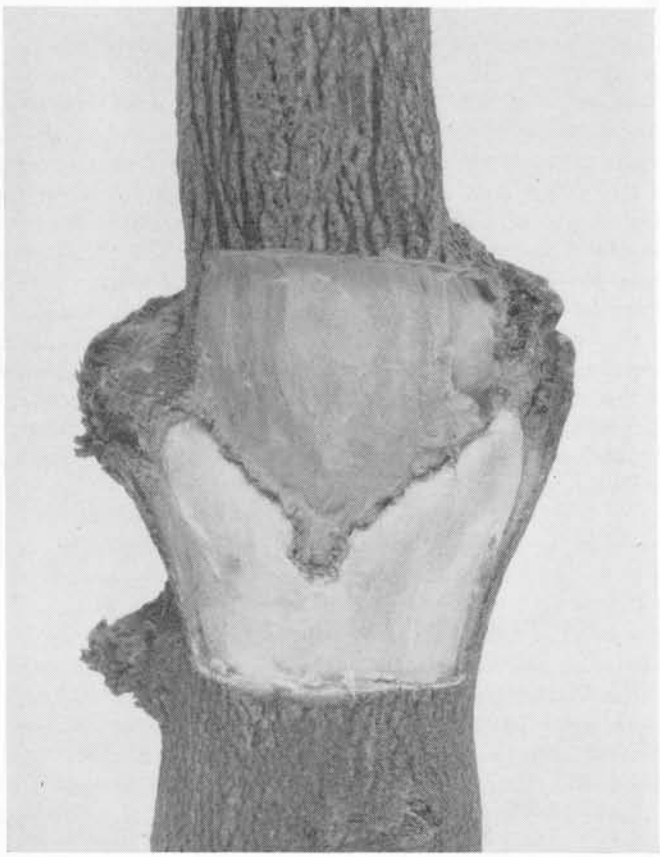

Fig. 2. Bud-union disorder of calamondin scion on grapefruit rootstock three years after budding. Bark removed to show groove and dark line at the union. (Photograph by K. L. Middleham.)

Indian lime, Orlando tangelo, Sunshine tangelo, Rangpur lime, and Palestine sweet lime.

Transmission Tests with Budded Trees. In the first test of transmission of the disease to budded trees, nucellar seedlings were grown from seed of calamondin trees growing on their own roots. Buds were then taken from the nucellar calamondin seedlings and propagated on potted seedlings of sour orange, sweet orange, grapefruit, Troyer citrange, Morton citrange, *P. trifoliata*, Willow-Leaf mandarin, Wekiwa tangelo, and Rangpur lime. The calamondin buds were forced to grow. Five calamondin trees on each of the above-listed rootstocks were then inoculated by inserting scions from affected calamondin trees into both the scion and the rootstock of each plant. Five other plants of each combination were grafted with tissue from nucellar seedlings of calamondin to serve as checks. In addition, scions from affected old-line calamondin trees were propagated on 5 seedlings each of sour orange, sweet orange, grapefruit, Troyer citrange, Morton citrange, *P. trifoliata*, Willow-Leaf mandarin, Rangpur lime, Wekiwa tangelo, Sampson tangelo, Ponkan mandarin, and Rough lemon and forced into growth.

In a second test with budded trees, scions were taken from uniform seedlings of Wekiwa tangelo and Sunshine tangelo and grafted into potted nucellar seedlings of calamondin and forced into growth. Five trees each of Wekiwa tangelo and Sunshine tangelo on calamondin roots were then graft-inoculated by inserting scions from affected calamondin trees into each. Five other plants of each combination received scions from nucellar seedlings of calamondin to serve as checks.

A few months after inoculation, all the trees were removed from pots and transplanted into the field to await development of symptoms. From time to time thin sections of bark were removed at the bud union to expose any discoloration or abnormality that might occur.

In the nucellar-line calamondin trees on sweet orange, grapefruit, Troyer citrange, Morton citrange, *P. trifoliata*, and Rangpur lime rootstocks which were inoculated by inserting affected scions, no bud-union symptoms or other evidence of disease have appeared during three years of observation. However, in the nucellar-line calamondin trees on Wekiwa tangelo, Willow-Leaf mandarin, and sour orange, both the inoculated trees and the check trees on these rootstocks developed a bud-union disorder and declined severely two and one-half to three years after inoculation. In the group of trees in which scions from affected old-line calamondin trees were propagated on the various rootstocks, all the trees except those on Rough lemon developed severe bud-union symptoms and wilted from two to three years after propagation. Many of these trees died. No symptoms have thus far been observed in trees of affected calamondin scions propagated on Rough lemon.

In the group of Wekiwa tangelo and Sunshine tangelo trees on calamondin, which were inoculated with scions from affected calamondin trees, none of the trees have expressed any bud-union or decline symptoms after three years of growth.

DISCUSSION AND CONCLUSIONS

The bud-union disorder of calamondin trees exhibits a complex series of scion-rootstock reactions which make a current interpretation of the cause almost impossible. The disorder is shown to be bud-perpetuated by the fact that scions from affected trees propagated on most other rootstocks invariably produce affected trees two to three years after propagation. The absence to date of any positive results from attempts to transmit the disorder to supposedly healthy nucellar-line calamondin trees indicates that it is not graft-transmissible. This is strengthened by the fact that on Wekiwa tangelo, sour orange, and Willow-Leaf mandarin rootstocks both the inoculated and the uninoculated nucellar-line calamondin trees developed the disorder. Any virus that

might be involved, therefore, must be seed-transmitted and carried in these rootstock seedlings or in the calamondin seedlings, or is readily transmissible by mechanical means or by a vector.

It can be argued that the disorder is the result of incompatibility between stock and scion or that some substance produced in calamondin tops is lethal to most other citrus varieties. This explanation would seem to be at variance with the view expressed by Webber (*11*), that the degree of congeniality follows closely the nearness of genetic constitution and that, in general, plants that can be successfully hybridized can be successfully worked together in propagation. Neither is this explanation in accord with all the manifestations of the disorder that have thus far been observed. In the case of propagations on sweet orange, grapefruit, Troyer citrange, Morton citrange, *P. trifoliata*, and Rangpur lime rootstocks, only the old-line calamondin trees have developed the disorder, whereas, with Wekiwa tangelo, sour orange, and Willow-Leaf mandarin rootstocks, both old-line and nucellar-line calamondin trees are affected. Moreover, no disease symptoms have developed in the Wekiwa tangelo and Sunshine tangelo trees propagated on calamondin rootstocks. It is possible, however, that sufficient time may not have elapsed for the disorder to develop in all of the combinations or under all of the conditions tested, and that it may develop in these trees at some later time.

It is the belief of the writers that the true nature of the calamondin bud-union disorder has not been demonstrated, that a complex situation exists in regard to this disorder, and that more work is necessary to determine its nature. In the light of the absence of any evidence of transmissibility and until more information regarding its cause is gained, it seems most logical at this time to refer to it as a bud-perpetuated, nontransmissible disorder.

ACKNOWLEDGMENTS

The authors gratefully acknowledge the cooperation of G. E. Goodall, J. H. LaRue, K. W. Opitz, and R. G. Platt of the University of California Agricultural Extension Service; Marcus E. Cravens of the Agricultural Commissioner's Office, Santa Barbara, California; and C. J. Barrett of the Agricultural Commissioner's Office, Ventura, California. They also wish to express their appreciation to Citrus Field Research, Inc., Santa Barbara, California.

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