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Response of Stubborn-Infected Trees to Iron Chelates

WHILE STUBBORN DISEASE affects several varieties of sweet oranges and grapefruit in Arizona, it is most widely prevalent in the Washington Navel oranges. Surveys show from 4 to 43 per cent affected trees. Several commercial groves planted since 1948 have more than 20 per cent infected trees. Budwood for these groves came from highly productive old-line trees without recognizable stubborn symptoms. This high incidence indicates that stubborn is the most serious citrus disease in Arizona and suggests that it is becoming worse.

Although stubborn disease was not designated and reported as such until 1944 (3), many Arizona growers recognized these off-type "runt" trees between 1920 and 1930 and attempted to improve them by various fertilizer, pruning, and top-working treatments. Records of the development of the disease and its effect on fruit production have not been reported.

Diagnosis of Stubborn Disease

The chief characteristic for the identification of stubborn disease is abnormal growth which produces atypical development of shoots, leaves, and fruit. This is an uncertain basis for diagnosis. Acorn-shaped fruit associated with stubborn in other areas occurs infrequently in the desert area. Young trees may develop vegetative symptoms before the trees begin to set fruit, so that only leaf and shoot abnormalities can be used.

The writer distinguishes two types of stubborn disease in the Wash-

ington Navel orange. *Type A* includes trees with a general restriction of growth that produces a stunted tree. Symptoms may develop on trees 3 years old or may be delayed until the tree is 25 to 30 years old. In old trees, symptoms usually develop on one part of the tree and gradually spread. Characteristic symptoms are as follows: small, upright spring leaves; short, stubby summer shoot growth with round type, thick leaves with prominent veins; growth from multiple buds and twig dieback. Other symptoms are: leaf abscission in December; reduced sucker development; less tolerance to cold; increased injury from citrus thrips and off-season blossoming.

Type B is characterized by abnormal vigor of primary shoots which develop into main scaffolds. Secondary shoot growth is restricted and similar to that of *Type A* so that an open type tree develops. As the disease advances, individual limbs may defoliate badly, weak shoot growth then follows or the limb may die back. Iron chlorosis is prevalent. Acorn-type fruit are more likely to occur than on *Type A* trees.

Development of Stubborn Disease

Observations have been made on 417 trees budded on sour orange rootstock and planted in 1933 in Block F at the Citrus Experiment Station in Tempe, Arizona. The original owner of the grove obtained the trees from two commercial nurseries and they are typical of the commercial trees planted at that time. Yield records began in 1943 when the University obtained the grove and observations on stubborn disease were started in December, 1948.

The development of *Type A* stubborn disease symptoms and yield records from 5 affected trees are presented in Table 1. These records illustrate typical variations in the development of the disease and emphasize the difficulty in evaluating treatment responses.

Tree 1 is considered a normal healthy tree. Varying yields apparently are caused by climatic conditions.

Tree 2 is a typical tree that developed stubborn disease early in its life so that yields were always low.

Tree 3 was never fruitful, but failed to develop definite stubborn symptoms until 1951. After many low producing years it produced a moderate crop in 1954, and then declined rapidly.

Tree 4 is a tree with delayed deterioration from stubborn disease. In 1954 two main limbs developed symptoms rapidly. The opposite side of the tree remained normal for 2 more years before stubborn became general and yields decreased. This type of disease manifestation is be-

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coming more prevalent at the Citrus Experiment Station. Fourteen trees, which were all considered normal prior to 1956, were rated questionable in 1957 and definite first or second stage stubborn in 1960. Yields on these trees, which averaged 12 per cent below 14 paired normal trees prior to 1956, dropped to 29 per cent below these normal trees in the 1958-1960 period.

Tree 5 is a moderately fruitful tree which has had some stubborn symptoms since 1951. The tree has failed to develop definite serious stubborn symptoms. Such trees probably carry an infection which interferes with fruit production but not sufficiently with growth to manifest the disease.

TABLE 1. DEVELOPMENT OF STUBBORN DISEASE AND EFFECT ON FRUIT PRODUCTION

Year	Tree number									
	1		2		3		4		5	
	Rate (a)	Yld. (b)	Rate (a)	Yld. (b)	Rate (a)	Yld. (b)	Rate (a)	Yld. (b)	Rate (a)	Yld. (b)
43		247		96		95		134		204
44		485		155		206		337		351
45		270		100		213		344		418
46		162		92		98		191		310
47		151		32		104		45		158
48	N	335	1	?	93	N	113	N	98
49		733		108		290		455		368
50	N	786	1	244	?	122	N	422	N	537
51	N	195	2	55	2	46	N	102	?	172
52	N	426	2	27	2	25	N	161	?	138
53	N	348	2	121	2	97	N	254	1	342
54	N	592	3	174	3	244	N	239	?	388
55	N	683	3	18	4	43	1	428	1	273
56	N	497	3	18	4	32	2	98	N	269
57	N	581	5	9	3	17	2	163	?	207
58	N	247	3	0		0		29		24
59	N	339	4	0	5	11	3	133	N	256

^aVisual classification of trees.

N—Normal.

?—Growth suggests stubborn because of a few round curled leaves and stubby short summer shoots, slight flat top.

1—Stubborn disease; definite early stage with few stubby shoots.

2—Stubborn disease; moderate number stubby shoots, multiple bud shoots, flat top.

3—Stubborn disease; reduced growth and mild dieback.

4—Stubborn disease; severe growth reduction with moderate dieback.

5—Stubborn disease; severe growth reduction with severe dieback.

^bYields in fruit per tree.

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The development of stubborn disease symptoms in two groups of trees at the Citrus Experiment Station is summarized in Table 2. Of the 417 trees in Block F, 57 trees had definite symptoms in 1948. A marked increase in trees showing disease symptoms occurred between 1948 and December, 1950, following severe freezes in January, 1949, and 1950. Thereafter a gradual increase in the number of infected and suspected trees took place.

In Block D, which contained 53 trees, 11 trees were infected with stubborn in 1948. In 1952, 19 infected trees and 1 suspected tree were removed to determine whether the removal of the diseased trees would influence the development of the disease in the remaining ones. In February, 1960, 11 trees had definite symptoms and 5 were questionable. Thus the removal of diseased trees did not reduce disease development in adjacent trees.

TABLE 2. ACCUMULATIVE TOTAL NUMBER OF TREES WITH SYMPTOMS OF STUBBORN DISEASE IN TWO BLOCKS

Year	Block D ^a			Block F ^b		
	normal	possibly stubborn	stubborn	normal	possibly stubborn	stubborn
1948	36	6	11	332	28	57
1950				272	9	136
1952	33	1	19	248	29	140
1954				217	36	164
1957				140	99	178
1960	18	5	30			

^aTotal original trees 53.

^bTotal original trees 417.

Effect of Freezing on Stubborn Development in Young Trees

Between 1948 and 1950, infected trees were removed from Block F and replaced with trees obtained from a commercial nursery. Of 27 trees planted in 1948, only 10 survived since freezes in January, 1949, and in January, 1950. Five trees on which the head survived developed into normal trees. Of the 5 remaining trees which were frozen to the trunk, one developed stubborn disease, 2 were considered probable stubborn, and 2 normal. Fourteen of the 25 trees planted in 1949 survived the 1950 freeze. One tree survived with the head intact, and made excellent normal growth. Thirteen were frozen to within 2 to 8 inches of the bud union, so the trees arose from dormant buds. Of these 13 trees,

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8 definitely had stubborn disease, 2 had probable stubborn symptoms, and 3 made moderate normal growth. Of the 20 trees planted in 1950, 3 had probable stubborn symptoms, 2 definite stubborn disease, and 15 were considered normal.

It may be postulated that the stubborn factor was present in the trees when obtained from the nursery. Freezing of tops back to the trunk may have weakened the tree so that the new growth soon manifested stubborn symptoms. It is also possible that the stubborn factor may become more concentrated in dormant buds and normally inhibit their growth. When such buds emerge to form a tree, stubborn symptoms quickly develop. The superior growth of the least frozen trees suggests stubborn may not have been present in these trees.

Experiments

TOPWORKING STUBBORN DISEASE TREES.—In 1953, Washington Navel trees in Block F at stage 2 or 3 stubborn were topworked with 4 old-line varieties that had not been affected with recognizable stubborn symptoms. Five trees budded with Eureka lemon made very weak growth. Of the 6 trees budded with Lisbon lemon, 1 made very weak growth, 1 was fair, and 4 grew vigorously. Two Kinnow mandarin trees remained healthy but growth was stunted. One Clementine mandarin made fair growth and 5 others made very weak growth. During the period of observations, it was established by budding on Rangpur lime that the Eureka and Clementine carried exocortis virus and the Lisbon did not. The Kinnow mandarin budded on old-line Marsh grapefruit developed mild cachexia symptoms.

The results suggest that when the stubborn factor is combined with exocortis virus a strong inhibition of growth occurs.

ROOTSTOCK REACTION.—*Buds from stubborn trees.*—In October, 1953, buds from a severely-affected stubborn tree (Table 1, tree 2) were budded on 5 different rootstocks and planted in the field in 1955. The condition of the trees in September 1960 was as follows. Cleopatra root: 7 trees; all with severe stubborn; 4 died during the first 3 years; very slight honeycomb in the bark on 1 tree. Troyer root: 5 trees; 3 with severe stubborn; 2 normal and vigorous; slight honeycomb in bark on 4 trees. Sour orange: 4 trees; 2 vigorous, 1 possible stubborn, 1 stubborn; small pegs in bark on 2 trees, slight honeycomb in 1 tree, stubborn tree with normal bark. Rough lemon: 3 trees; 1 very severe stubborn,

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2 normal; stubborn tree top overgrew union, with slight stem pitting; 1 normal tree with bark pegs, 1 normal tree with no abnormal condition. Rangpur: 3 trees; all normal, vigorous, no exocortis, no abnormal conditions at bud union.

This test shows that variation occurred in the transference of the stubborn factor with all rootstocks except Cleopatra mandarin. All trees on this rootstock reacted violently to stubborn disease. The exocortis virus was not in the stubborn-infected tree used in this test.

Buds from normal trees.—In 1954, from 4 to 21 trees of 16 different rootstocks were planted in the field. Budwood for these trees was taken from 4 Washington Navel trees planted in 1939 which had been grown from buds from a single tree planted about 1922. None of these parent trees had stubborn symptoms in 1958. Bark inspection of rootstocks show cachexia present in susceptible varieties, so it is assumed that cachexia virus is present in all trees. A severe freeze in December, 1954, defoliated all trees and killed part of the heads.

The percentages of trees rated stubborn or possible stubborn in September and December, 1960, were as follows: rough lemon, 62; Rangpur lime, 75; sour orange, 67; Oklawaha sour orange, 67; Sacaton citrumelo, 40; Troyer citrange, 55; citrangor, 70; Savage citrange, 100; Kara mandarin, 37; Oneco mandarin, 40; Batangas mandarin, 60; Willow leaf mandarin, 55; Dancy tangerine, 70; Kinnow mandarin, 80; Cleopatra mandarin, 88; Wilking mandarin, 100. These data suggest that Savage citrange, Cleopatra mandarin, and Wilking mandarin could be used as index plants for stubborn disease.

RESPONSE FROM NUTRITIONAL SPRAYS, AND SOIL MANAGEMENT.—Stubborn-infected trees frequently develop zinc mottle, iron chlorosis, and other mottle leaf patterns. The possibility that correction of nutritional deficiencies would alleviate stubborn disease has been a tantalizing idea. Beginning in 1949, nutritional sprays of zinc, copper, manganese, and phosphorus were applied to 16-year-old trees 3 times each year and continued for 3 years. There was no improvement. Between 1949 and 1956, soil fertilization with manure, or large amounts of nitrogen combined with cultural programs of bare soil nontillage, cover crops disked, and bermuda grass sod mowed had no effect on the disease.

RESPONSE FROM CHELATED METAL COMPOUNDS.—In May, 1957, two 24-year-old trees in the very early stubborn stage were treated with 2 lb. of RA 157 (an experimental iron chelate of unknown formula supplied by Geigy Agricultural Chemicals), 1 lb. Zn DTPA, and 1 lb. Mn.

EDTA. New summer growth was deep green, and vigorous shoots developed in the tops of the trees. Yields of both trees increased slightly when compared with either adjacent control trees or previous yields. Also, stubborn symptoms decreased both with respect to previous condition and to the controls. Two additional trees in stage 4 of stubborn disease were treated with chelates; they produced improved growth for 2 years, but did not regain fruitfulness and declined.

A more extensive test was started in 1960 at the Experiment Station on 10- and 27-year-old trees and on 9- and 11-year-old trees in two commercial groves. Treatments were (a) 1 lb. HFe EDDHA; (b) 1 lb. HFe EDDHA and 1 lb. Zn DTPA; (c) 2 lb. Zn DTPA; (d) control. In the 2 commercial groves the trees selected showed first-stage definite symptoms or questionable symptoms. Materials were applied in April or May, 1960. In the commercial groves the iron chlorosis has been completely corrected by the iron chelate. The zinc chelate had no effect. Iron chlorosis in the zinc-treated and control trees remained approximately constant throughout the summer. Improvement in stubborn disease was based on the amount and length of the summer shoot growth, trunk enlargement, and defoliation during the fall. These ratings indicated that a rather well-defined improvement occurred in 11 of the 24 trees treated with 1 lb. HFe EDDHA either alone or combined with zinc; none of the 8 zinc-treated trees improved; 2 of the 12 control trees improved. These results are preliminary.

Discussion

The highly variable behavior of stubborn disease with respect to symptom development, transference by buds, and rootstock reactions suggests that the stubborn factor is present in most old-line trees in Arizona. Unfavorable environmental conditions, particularly freezes, apparently change the normal physiological condition of the tree so that the factor can become active. It is postulated that the stubborn factor acts by interference with the normal physiological action of auxin or other growth-regulating substances. The variable transference by buds from an infected tree suggests that all buds do not contain the same quantities of the stubborn factor, which may also be reflected in the time of appearance of the disease.

Some conclusions can be drawn regarding the possibility that stubborn disease involves a combination of other virus diseases. Because the rootstock experiment trees show a wide prevalence of stubborn without

exocortis symptoms present on Rangpur lime rootstock it is evident that stubborn disease can occur in the absence of the exocortis virus. Reichert, who initially considered stubborn (little leaf) a specific disease (6), now suggests that it is another manifestation of xyloporosis (5). Xyloporosis-infected Shamouti trees when budded on rough lemon in Israel and in Arizona develop stem pitting and a necrotic "ring" at the bud union. Stubborn-infected trees growing on rough lemon usually have a smooth bud union without serious stem pitting, and a necrotic "ring" has never been observed. Many stubborn trees are free of marked bark and wood abnormalities. The writer has observed honeycomb development in the bark with accompanying small pegs concentrated at the bud union and spreading through the trunk and upper roots only on trees which have shown serious stubborn symptoms for many years.

It appears that stubborn represents a specific virus and that this virus may induce bud mutations. This would explain some of the extremely abnormal trees observed. Fawcett (3) points out that "large trees affected might be mistaken for 'Australian' type navel." The Type B stubborn-affected trees in Arizona are larger than normal trees.

While stubborn is considered to be latent in the tree, the possibility of transference either by insects or root grafts from infected trees cannot be dismissed. In many instances, trees adjacent to stubborn trees have developed the disease and a general enlargement of the area of affected trees has taken place. However, one outstanding exception to this observation has been noted, in which the disease has not spread during 17 years after stubborn was evident in a single tree.

The value of iron chelates has not been established. At present it appears that symptom development may be arrested in certain trees by application of iron chelates to the soil if made in the earliest stages of symptom manifestation.

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