

Effect of Exocortis on Production and Growth of Valencia Orange Trees on Trifoliolate Orange Rootstock

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STUDIES on the effect of exocortis on growth (1-6) and yield (4, 6) of citrus trees, based mostly on young trees or on trees planted for purposes other than the study of exocortis, have been reported. This paper reports results of an investigation started in 1955 to determine the effects of exocortis virus on Valencia orange [*Citrus sinensis* (L.) Osb.] trees on trifoliolate orange [*Poncirus trifoliata* (L.) Raf.] rootstock.

Materials and Methods

In 1955, 20 experimental trees were propagated in the greenhouse by grafting buds from a healthy Campbell Valencia tree (nucellar clone) onto trifoliolate orange seedlings. Soon after propagation, 10 trees were inoculated with exocortis by bud grafts from 5 infected Eureka lemon [*C. limon* (L.) Burm. f.] trees. Inocula from each source were placed in 1 to 3 trees with no tree receiving inocula from more than 1 source. Indexing for other citrus viruses was negative in all 5 inoculum source trees. Seven of the 10 control trees were grafted with buds from thoroughly indexed, apparently virus-free lemon trees.

In April, 1956, the experimental trees were planted at Riverside, California in 24 x 10 foot spacing, on land where no citrus grew previously. Although tristeza and vein-ensation viruses were spread naturally throughout the plot from 1958 to 1963, the control trees remained exocortis-free for the duration of the experiment.

Results

PRODUCTION.—Precocious fruit production was noted on the diseased trees during the first few years, but the yields were too small to be economically significant. Production records for 1962 through 1966 show that the diseased trees produced only 58 per cent as much fruit by weight and 53 per cent by number, as the controls (Table 1). On the weight basis, yield differences between diseased trees and controls were significant at the 0.001 level for the 1962-1966 period, and for 4 of the 5 individual years at the levels shown in Table 1. In 1966, a low production year, weight differences were not significant. Preliminary estimates

TABLE 1. TOTAL YIELDS AND AVERAGE WEIGHT PER FRUIT FROM 10 CONTROL (C) TREES AND 10 EXOCORTIS-INFECTED (I) VALENCIA ORANGE TREES ON TRIFOLIATE ORANGE ROOTSTOCK, 1962 THROUGH 1966. RELATIVE YIELDS AND AVERAGE WEIGHT PER FRUIT OF INFECTED TREES TO CONTROLS IS SHOWN IN PER CENT (I/C)

Year	Yield			Avg. wt./fruit		
	C	I	I/C	C	I	I/C
	kg	kg	per cent	g	g	per cent
1962	536	371*	69	156	157	101
1963	728	397**	55	166	182	110
1964 ^a	341	168***	49	116	139*	120
1965	592	236***	40	200	262***	131
1966 ^b	259	241	93	166	173	104
1962-1966	2456	1413***	58	161	175*	109

a. Most mature fruits split and dropped prior to harvest.

b. Poor production year in the Riverside area.

***, **, *. Significantly different from C at the 0.001, 0.01, and 0.05 levels, respectively.

indicate that the controls should produce approximately twice as many fruits as did the exocortis-infected trees in 1967. The average weight of individual fruits of exocortis-infected trees was significantly higher than that of controls in 1964 and 1965, but not in other years.

GROWTH.—The effect of exocortis virus on growth of the young trees was apparent within 2 years after planting. After 4 years in the field the degree of stunting of the virus-infected trees (2) averaged 46 per cent. In July, 1966, the average cross-sectional area of the trunks, 6 in. above the bud-union, was 170 cm² for controls and 71 cm² for the infected trees; the difference (58 per cent) is significant at the 0.001 level. The average height of the exocortis-infected trees in July, 1966, was 2.7 m and that of the controls 4.3 m; the difference is significant at the 0.001 level.

Discussion

These data indicate a downward trend in fruit production of all trees; this trend was caused by poor production conditions in 1964-1966. In 1964, all trees lost a large portion of the mature crops from fruit splitting before yield data could be obtained. However, in 1966 the exocortis-infected trees yielded relatively well for their size and condition despite adverse production conditions. Inasmuch as other Valencia trees in the area experienced a poor fruit set in 1965, it seems probable that exocortis favorably affected the setting of fruit under adverse conditions. Differences in yield between diseased and healthy Valencia trees increased through several years, as did the difference between exocortis-free and exocortis-infected Satsuma mandarin (*C. unshiu* Marcovitch) in Louisiana (6). Doubtless, the difference in yield will increase again in 1967, following the reversal in 1966.

Conclusions

From the above results it was concluded that: 1) exocortis reduces the growth and fruit production of Valencia orange trees on trifoliolate orange rootstock, at least during their first decade; 2) exocortis causes larger fruit size some years; and 3) the tendency to alternate bearing, noted in the controls, is absent or less prevalent in the exocortis-infected group.

These data show that under the conditions of this experiment the effect of the virus was greater on tree growth than on fruit yield.

The relationship between tree size and fruit production suggests that exocortis reduced production by stunting trees and reducing the fruit-bearing surface. It must be assumed in this experiment that severe strains of exocortis virus were responsible for the typically severe lesions on the trifoliolate orange rootstock and for the severe stunting of the trees because: 1) no other virus could be detected by thorough indexing; 2) inoculated and control trees were equally exposed to natural spread of tristeza and vein-enation viruses; and 3) the exocortis virus from trees used in the experiment and severe strains of exocortis virus transmitted mechanically caused similar stunting in Etrog citron (*C. medica* L.), whereas mild strains of exocortis caused little or no stunting of citron and little or no bark cracking or stunting of trees on trifoliolate orange rootstock.

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