Limited Systemic Spread of Impietratura and Psorosis-A in Graft-Inoculated Grapefruit Trees

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ABSTRACT

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Four grapefruit trees were inoculated with impietratura-diseased buds on one or two secondary canopy branches per tree. Fruit symptoms characteristic of impietratura developed 3–6 mo after the secondary branches were inoculated. On a budded branch, symptoms were observed only near the inoculation site; even 8 yr after inoculation, affected fruits could not be found on branches originating about 1 m below the inoculation site. Indexing on indicator plants indicated the presence of inoculum causing leaf flecking symptoms only in branches originating less than 90 cm basipetally from the inoculation site and in almost all acropetal parts. Budwood from two grapefruit trees with psorosis-A showed the same infection pattern.

Impietratura (Imp) and psorosis-A (Ps-A) are two citrus diseases caused by graft-transmissible but as yet unidentified agents. However, the causal agents of these diseases are generally thought to be viruses (5,9). Ps-A induces bark scaling, and Imp is associated with gum deposition in fruit albedo. Grafting Ps-A-diseased budwood on indicator seedlings induces leaf flecking and a shocklike leaf drop. Symptoms were similar on indicator seedlings inoculated with budwood collected from several Imp isolates (2). Recently, during work on cross protection between Ps-A and Imp, we observed that 6–8 yr after graft inoculation the causal agents of these diseases appeared to spread systemically mainly acropetally; little systemic spread in other directions could be established.

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MATERIALS AND METHODS

Budwood for Ps-A inoculations was from a 40-yr-old Marsh cultivar seedless grapefruit (*Citrus paradisi* Macf.) with severe bark scaling. The Imp inoculum was an isolate from Bet Dagan (Imp-BD) described previously (1).

Two Marsh seedless grapefruit trees, about 30 yr old, were graft-inoculated with Imp-BD in March 1970 with 10 chip grafts per tree, all on one side of the tree. Grafts were made about 1.5 m from the ground on flowering branches 1–2 cm in diameter. Two years later, two other grapefruit trees, about 20 yr old, were similarly inoculated with Imp-BD and two more with Ps-A.

Seedlings of sour orange (*Citrus aurantium* L.) and sweet orange (*C. sinensis* (L.) Osb.) cultivars Valencia and Madam Vinous were grown in 3-kg pots containing soil, sand, and peat (2:1:1). Inoculations were done by two chip grafts. After the grafts were established, the plants were topped to force new growth and transferred to a greenhouse chamber kept at 22 ± 4 C.

RESULTS AND DISCUSSION

Imp fruit symptoms developed on each of the four Imp-BD-inoculated trees 3–6 mo after inoculation. Diseased fruits were reduced in size, with large gum deposits under the calyx. In the first year after inoculation, fruit with symptoms appeared only on the inoculated branches, acropetally from the inoculation sites. Fifty randomly distributed fruits collected from the top branches and from the outer canopy of each of these trees were carefully examined for Imp symptoms. In addition, fruits thought to be diseased (eg, smaller than normal) were separately sampled and checked for gum deposition. Fruit symptoms, including reduction to about one-half normal size, recurred annually during each of the following years. Even 8 yr after inoculation, however, affected fruits were not found on other branches located more than 1 m below the inoculation site.

Results of indexings 5 and 7 yr after inoculation also indicated that the disease agent remained localized in the inoculated sites. Of five sites per tree sampled on the periphery of the canopy and indexed with four indicator plants per site, only the inoculated site gave positive reactions (14 of 16 sour orange seedlings used for indexing). All remaining sites tested on 64 indicator plants gave negative reactions. Inoculum taken acropetally and from branches originating less than 90 cm basipetally from the area of inoculation caused leaf flecking on young sour orange leaves, whereas budwood taken basipetally beyond this point and from other uninoculated secondary branches did not produce symptoms.

Budwood collected from two grapefruit trees 5 yr after inoculation with Ps-A and indexed on Madam Vinous sweet orange seedlings revealed the same pattern of infected budwood.

Only a few trees were tested for each disease, but each of the trees that received 10 graft inoculations reacted similarly. This strongly supports the hypothesis of limited systemic spread for both disease agents.

Indications of transmission of the Ps-A agent via root grafts were obtained by Bittancourt and Fawcett (3). Other studies (8,10) indicated that a disease agent associated with psorosislike bark lesions appears to spread naturally in Argentina and Texas in a pattern indicating dispersal by factors other than direct root grafts. Uneven distribution patterns in citrus trees of some citrus ringspot virus isolates belonging to the citrus psorosis complex (10) were recently demonstrated by Timmer and Garnsey (11). The virus could be readily detected in bark lesions of mature field trees but only erratically in symptomless parts.

The citrus psorosis complex includes several viruses that cause leaf flecking symptoms on susceptible indicators. Some of them have been identified as ilarviruses (10). The pattern of spread of the citrus ilarviruses has not been conclusively established, but some ilarviruses that infect *Prunus* trees reportedly are transmitted through pollen (6) and are nonuniformly distributed in infected trees.

Matthews (7) described two kinds of systemic movement of viruses in plants: relatively slow cell-to-cell spread with rate of spread closely connected to virus multiplication and rapid long-distance spread through conducting tissue. With citrus tristeza virus (CTV), a phloem-transported virus, Burnett (4) reported that 13½ mo elapsed from the time the first test plant gave a positive CTV reading until all parts of a tree were systemically infected. The Imp and Ps-A agents spread systemically at a much slower rate than that of CTV, especially basipetally, which indicates differences in the type of vascular tissue used for transport or that there are undefined difficulties in entrance of the infective material into the conducting tissue. Considering the limited systemic spread and uneven distribution of several components of the psorosis complex, including Imp, and some indication for pollen transmission (12), we suggest that in addition to frequent indexing and inspection for scattered fruit symptoms, budwood source trees should be isolated from diseased trees.

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