CHAPTER 4

Exocortis and Related Diseases

Review of Recent Research on Exocortis Disease

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In 1948, Fawcett and Klotz (8) described and gave the name exocortis to a bark-shelling disorder of Poncirus trifoliata (L.) Raf. rootstocks, and in 1949, Benton et al. (2) in Australia, demonstrated that the causal agent is a virus. Indexing by numerous investigators in many parts of the world indicated that the causal virus is widely distributed in citrus varieties. However, no obvious symptoms are produced on non-sensitive rootstocks.

The use of rootstocks sensitive to exocortis virus has increased in recent years. This is especially true since tristeza-tolerant rootstocks such as trifoliate orange, Rangpur lime (Citrus reticulata var. australis hyb.), and Troyer citrange (P. trifoliata x C. sinensis) have been substituted for sour orange (C. aurantium L.) rootstock in areas where tristeza occurs. When budwood infected with exocortis virus is propagated on these rootstocks, bark shelling, retarded growth, and reduced yields result. In many areas considerable losses have resulted because propagators ignored the general but fundamental rule of propagating only budwood from vigorous, productive, long-lived trees growing on the same rootstock as that used for propagation.

The effects of exocortis virus on tolerant rootstocks are more subtle. For example, grafts of lemon [C. limon (L.) Burm. f.] infected with
exocortis, but free of other citrus viruses, caused measurable stunting of
trees on the “tolerant” rootstocks: sweet orange, grapefruit (C. paradisi
Macf.), and sour orange (5). Exocortis-infected Washington navel
orange trees on Cleopatra mandarin (C. reticulata Blanco) and sweet
orange rootstocks showed no obvious bark symptoms, but grew more
slowly than virus-free trees, and infected trees on sweet orange rootstock
produced less fruit than did virus-free trees (20).

Exocortis virus also affects sweet lime [C. aurantifolia (Christm.)
Swing.], sweet lemon [C. limon (L.) Burm. f.], Cuban shaddock, pos-
sibly a lemon-citron hybrid, and Tahiti lime (15, 17, 23).

Indexing for Exocortis

Since exocortis virus causes no obvious symptoms on many citrus
varieties, its presence in symptomless carriers may be determined only
by indexing, i.e., the procedure of grafting tissue of the suspected host
onto sensitive indicator plants. Likewise, failure to transmit exocortis
virus by grafting tissue of a tree to the most sensitive indicator is the
best proof that the tree under test is free of exocortis virus.

Benton et al. (2) in 1949, in Australia, were indexing sweet orange
selections when they showed that exocortis is graft-transmissible and that
symptoms appear 4 to 8 years after infected sweet orange tissue was
budded onto P. trifoliata. Moreira (12) in Brazil, and Olson and Shull
(14) in the United States, showed that exocortis virus caused bark shell-
ing of Rangpur lime within 44 months after budding. Thus, Rangpur
lime provided a more rapid test for exocortis virus than did P. trifoliata.
Childs et al. (7), in the United States, developed a test for infection
based upon histochemical examination of phloem ray cells of P. trifoli-
ata. It too was replaced by newer tests.

Moreira (13) showed that yellow blotching of Rangpur lime and P.
trifoliata bark was a symptom of exocortis infection and that it occurred
4 to 6 months after infection in vigorous plants. In some areas blotching
from unidentified causes reduces the reliability of this test.

In the United States, Calavan and Weathers (3) included citron (C.
medica L.) among a list of species with symptoms resembling those pro-
duced by exocortis. In Brazil, Salibe (16) showed that some varieties of
citron reacted severely to exocortis infection within 200 days. In 1964, a
team of Californians (4, 10) developed an indexing procedure based on
selected seedlings of Etrog citron. Leaf symptoms of exocortis infection
developed in the selections five to ten weeks after inoculation. This test
is currently the best one for indexing exocortis virus in possible bud-
wood sources. Thus, in the period 1949 to 1964, the time required to index for exocortis virus was reduced from eight years to ten weeks. This development resulted from cooperation and exchange of data among research workers in Australia, Brazil, and the United States.

Different strains of exocortis (6, 18, 23) exist, and some strains stunt trees in the absence of bark-scaling symptoms (9). Strains of exocortis virus that cause no recognizable symptoms on Rangpur lime and *P. trifoliata* are detected by the citron test (4).

Plant nutrition also affects the effectiveness of indexing methods. Weathers *et al.* (24) obtained better symptom development and shorter incubation periods on *P. trifoliata* plants by using high levels of nitrogen and phosphate fertilizer.

**Virus Spread**

Reports by many observers suggest that exocortis virus is spread mainly through propagation of infected plants. However, Weathers (21, 22) transmitted the virus experimentally from citrus through dodder (*Cuscuta subincisa*) to citrus and *Petunia hybrida*. He also transmitted exocortis from petunia to petunia by grafting and by mechanical transfer in sap. Natural spread from citrus to citrus in the greenhouse has been reported (4). No evidence of seed transmission of exocortis has appeared in seedlings from infected trees in Australia (9), South Africa (1), and California. However mild strains of exocortis occurred in non-inoculated nucellar seedlings of exocortis-infected Baianinha navel orange in Brazil (19). Since this review was presented in 1966, Garnsey and Jones (11) have shown that exocortis virus was transmitted mechanically on grafting tools to 26 of 30 plants in three tests.

**Current Problems**

The principal unanswered questions affecting the problem of exocortis virus infection are as follows: In what way do weak strains of exocortis affect citrus varieties on different commercial rootstocks? Does seed transmission of exocortis virus occur? Does a virus other than exocortis virus cause stunting of trees on *P. trifoliata* rootstock? How do variations in environment affect expression of symptoms?

**Literature Cited**

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