

## Citrus Dieback in India—the Contribution of Greening Virus

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DIEBACK DISEASE of citrus has caused increasing alarm to horticulturists and growers in India since it was first observed over 20 years ago. It has now been recorded in all citrus-growing areas of the country. As a result of numerous investigations from many angles, it had come to be generally accepted that the disorder involved tristeza virus, zinc deficiency, and some fungal parasites of twigs (1).

None of the above conditions can adequately explain the disease or its spread. Tristeza is widespread in Indian citrus, but is not universal, and is not invariably present in dieback-affected trees. Moreover, many of the species affected in India are tolerant of tristeza in other countries. Deficiencies of zinc and other minor elements have been implicated on a symptom basis, but application of zinc with or without other minor elements failed to cure the condition, although sometimes slight and temporary improvement resulted. The fungi associated with dying-back branches are well known as invaders of moribund or weakened tissues.

A study was made of citrus dieback disease in all major citrus areas of India, during April, May, and June, 1966.

### *Observations*

As a result of the observations made, and a review of the experimental work in progress at research institutions in several States (3), it is concluded that dieback is caused by the virus responsible for greening disease of citrus in South Africa (3).

DISTRIBUTION AND MEANS OF SPREAD.—The dieback disease was seen in all citrus-growing districts in India, and almost all citrus appeared to be infected. At present, all nurseries are taking budwood from infected trees and much of the spread has been due to that practice. Insect transmission is obviously taking place since seedling trees of all ages in private plantations and in research institution orchards were infected. Infected unbudded seedlings only nine months old were seen in nursery seedbeds.

SYMPTOMS.—Symptoms vary in intensity with the variety. In sweet orange [*Citrus sinensis* (L.) Osb.] the range of symptoms is exhibited most clearly in young, vigorously growing trees. In trees which appear to have been infected after reaching maturity, symptoms are less marked. In spring-formed leaves, tissues along the main vein turn yellow after

leaves reach maturity, and chlorosis spreads over lateral veins and involves much of the leaf in severe cases. Such leaves usually fall. Heavy blossoming with poor setting of fruit may also be evident. Later growth is less vigorous; many twigs are upright and produce smaller leaves. The leaves develop patterns more or less resembling those of zinc and iron deficiency; they may also exhibit round or angular yellowish blotches or become entirely yellow. Frequently, small green spots occur on chlorotic leaves.

Reduced vigor is associated with sparse foliage, dieback of branches, and a steep decline in cropping. Final deterioration and death occur through secondary invasion by wood-rotting fungi.

McClellan and Oberholzer (3) noted that leaf symptoms of greening are often restricted to one or several limbs in early stages of the disease. This is also a striking symptom of the dieback disease in India and suggests that to some degree the reaction of susceptible varieties to infection is a hypersensitive one, and that the metabolic status of the host may be important in determining virus multiplication and spread. Leaf symptoms are masked by high temperatures, and growth made at the hottest time of the year is generally symptomless as noted by McClellan and Oberholzer (3) for greening disease.

Sudden wilting and death of some affected trees occurs during late spring and early summer in several districts of central and southern India. All gradations between this sudden collapse and the normal spring sequence of symptom development were seen. Occasionally, young trees showing the first symptoms of greening developed large vigorous leaves with numerous small circular pale green spots of a ringspot type (Fig. 1).

In species other than sweet orange, the general pattern of symptom development is similar, although intensity of chlorosis, amount of dieback, and degree of yield reduction is less in the more tolerant types.

Fruit symptoms as described by McClellan and Oberholzer were not seen. A type of sunburning was common in the Mosambi and Sathgudi sweet orange varieties, mandarins (*C. reticulata* Blanco), Meyer lemon [*C. limon* (L.) Burm. hyb.], and pummelo [*C. grandis* (L.) Osb.]. Typically, a yellow patch with abrupt margin occurs on the exposed side of the fruit, while the rest of the fruit remains conspicuously green. In several tangelo varieties, and occasionally in mandarin, sweet orange, and grapefruit, lopsided fruits with poorly developed seeds were observed, but this is not a feature of the disease.

HOST RANGE AND REACTION.—All sweet orange varieties grown are highly susceptible and none show sufficient tolerance for commercial pur-

poses. Sathgudi, when grown on Sathgudi stock, persists longer than most others, but cropping eventually becomes uneconomic with this variety also. No stock exerts a worthwhile ameliorative effect on the disease in sweet orange.

Mandarins vary in reaction with strain, locality, and stock. The seedling varieties grown in hill areas (Kalimpong, Assam, Coorg, et cetera),

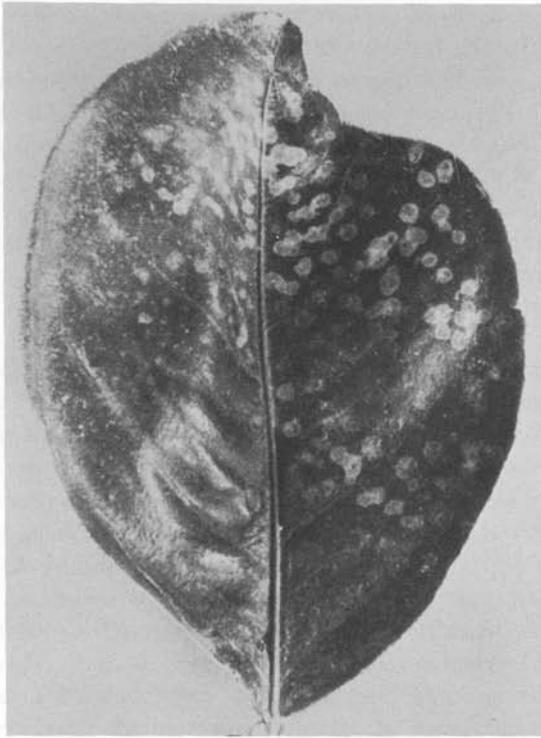


FIGURE 1. *A large vigorous leaf with small pale green spots of the ringspot type.*

if affected at an advanced age, exhibit reduced cropping and vigor. Finally, twig dieback occurs, and the trees gradually die from secondary wood rots. Seedlings infected at an early age react more severely. Apparently the yellowing of veins and adjacent tissue in mature leaves occurs near the point of infection. Chlorotic leaf patterns develop in subsequent growth, and considerable leaf fall results in sparse foliage. Dieback and death ensue within a few years. Cleopatra-type mandarins appear to be less severely affected.

Many strains of Rough lemon (*C. jambhiri* Lush.) exist in India and most appear somewhat tolerant of the disease, but in Assam, the Sohmyndong strain is quite susceptible. The strains of Rough lemon in use as stocks at Coorg and at Nagpur show a useful degree of tolerance and have the effect of prolonging productive life of the Coorg and Nagpur mandarins, as compared with seedling mandarin trees.

Grapefruit (*C. paradisi* Macf.) appears almost as susceptible as sweet orange, and tangelo (*C. reticulata* x *C. paradisi*) varieties appear extremely susceptible. On the other hand, lemons, Rangpur lime (*C. reticulata* var. *austera* hyb.), sweet lime [*C. aurantifolia* (Christm.) Swing.], and *C. karna* Raf. show considerable tolerance. Symptoms in sweet lime and *C. karna* Raf. were restricted to mild leaf blotching in young trees.

Moderate symptoms were shown by *C. hystrix* DC, Meyer lemon, *C. ichangensis* Swing., citron (*C. medica* L.), and sour orange (*C. aurantium* L.). Mild symptoms were exhibited by *Poncirus trifoliata* (L.) Raf., *C. maderaspatana*, *C. assamensis*, wild lemon, and *C. latipes* (Swing.) Tann. Only one species, *C. macroptera* Montr., showed no symptoms which could be related to greening.

AGE AT WHICH SYMPTOMS APPEAR.—Although all budwood is now of necessity taken from infected trees, generally less than 5 per cent of young budded trees in commercial nurseries show visible evidence of infection. Young commercial blocks of sweet orange varieties propagated from infected sources usually grow well for four or five years. Major development of symptoms occurs after the first crop of fruit and appears first in only a few trees. However, by the time the block is seven to eight years old, most or all of the trees show severe dieback. It is generally noted that when the virus is spreading through a virus-free plantation, the first symptoms of dieback disease are seen following heavy cropping. McClean and Oberholzer (3) have reported a variable percentage of transmission of greening and postulate uneven distribution of the virus in the tree so that some buds do not carry it. Possibly, virus multiplication is checked until the physiological state of the tree, coincident with heavy cropping, favors its extension.

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