

## Psorosis

### Relationships of Viruses of the Psorosis Virus Complex

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VIRUSES SO FAR ascribed to the psorosis virus complex are those of psorosis A, psorosis B, crinkly leaf, infectious variegation, concave gum, and blind pocket. Symptoms induced by them have been described in detail by others (3, 4, 5, 7).

Symptoms typical for psorosis B were observed in leaves and fruit of 30–40 trees of Washington Navel (old Mildura line) and Valencia Late (Leng strain) sweet orange in a block of 200 trees in the irrigation district of Monak, New South Wales. However, the conspicuous bark gumming and scaling described as features of psorosis B were not present. Their absence prompted an investigation to elucidate the relationship of the various viruses ascribed to the psorosis virus complex. The details of the investigation are reported here.

#### *Field Occurrence of Psorosis B in New South Wales*

Since the trees at Monak had typical psorosis B symptoms in leaves and fruit, the disease is provisionally referred to as psorosis B. Symptoms of

psorosis B develop on maturing leaves (principally shaded ones), are obvious on both surfaces, though paler on the lower surface, and persist throughout the life of the leaf. Small chlorotic lesions and ring spots, about 1 mm in diameter, and large translucent areas, sometimes in the form of an oak-leaf pattern, are characteristic (Fig. 1). Ring spots and a yellow etching pattern often develop along minor veins. Symptoms consisting of surface rings bordered by sunken grooves (Fig. 2) also occur on some fruit, rendering them unmarketable. Sometimes the flavedo cells in the area of the rings remain green after the fruit has ripened. Affected trees are smaller than unaffected ones, and less vigorous; they have a squat appearance rather than the rounded habit of normal trees.

Examination of the trees indicated that they had been topworked. Similar symptoms on foliage and fruit of a particular clone were observed in the Mildura district many years ago, and it is probable that the disease un-

der consideration was present in some of the original trees before they were topworked. This would explain the present occurrence of the disease in two well-known bud lines.

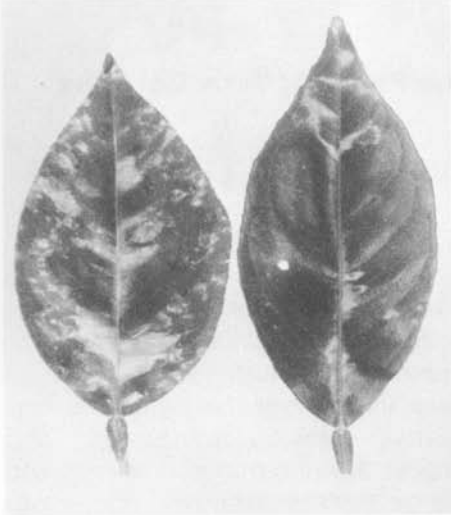


FIGURE 1. Yellow ring spots, etching, and large yellow translucent areas produced in sweet orange leaves by the Monak psorosis B virus.

No similar condition or symptom has been found in other blocks of old line Mildura Washington Navel and Leng Valencia sweet orange.

#### *Transmission Experiments*

Axillary buds or pieces of leaf tissue cut across a vein were taken from some of the diseased trees in the block at Monak and inserted into vertical slits in the stems of seedling indicator plants; the grafts were tied with budding tape. The symptoms induced on sweet orange, Orlando tangelo, and sweet lime seedlings were typical of those described for psorosis B. Few symptoms were

produced on rough lemon and Muscio mandarin seedlings and none on Troyer citrange. Eureka lemon seedlings, which did not exhibit tristeza yellows, developed psorosis B symptoms. West Indian lime and smooth seville seedlings developed tristeza symptoms but not those of psorosis B.

#### *Interference Studies*

The sources of inoculum used for cross protection tests were as follows.

*Crinkly leaf.*—A. Leaf pieces from a Eureka lemon mechanically inoculated with crinkly-leaf virus. The field source of the crinkly-leaf isolate was a 15-year-old Eureka lemon tree at Moulamein, New South Wales. B. Leaf pieces from a Harvard sweet orange seedling mechanically inoculated with crinkly-leaf virus. The field source of the crinkly-leaf isolate was a 20-year-old Eureka lemon tree at Lake Wyangan, New South Wales.

*Concave gum.*—Buds from 2 infected *Minneola* tangelo trees at Narromine. They showed trunk concavities and oak-leaf patterns. Tristeza virus present; exocortis virus absent.

*Concave gum plus psorosis A.*—Buds from a *Minneola* tangelo tree at Narromine. It had oak-leaf patterns, concavities in the trunk, and a psorosis A bark lesion. Tristeza virus present; exocortis virus absent.

*Psorosis A.*—A. Buds from a Washington Navel orange tree at Pitt Town with a twisted butt and bark scaling on the trunk. Tristeza virus present; exocortis virus absent. B. Buds from a Washington Navel orange tree at Pitt Town, with patches of scaling on

the butt and limbs that had callused at the margins of the lesions. Tristeza virus present; exocortis virus absent. C. Leaf pieces from a sweet orange seedling inoculated with buds from a Washington Navel orange tree at Barham, exhibiting psorosis A bark lesions on the trunk and branches. Tristeza virus present; exocortis virus status unknown. D. Leaf pieces from 10-year-old Bergamot trees held in quarantine at Rydalmere. These trees were intercepted during illegal introduction from Sicily. Oak-leaf patterns were seen in the growth flushes. Exocortis virus present; tristeza virus absent.

The isolates were grafted separately into Muscio mandarin and Ruby Blood sweet orange seedlings. After the development of crinkly-leaf or

psorosis symptoms—approximately 11 weeks later—the seedlings together with 20 noninoculated controls were inoculated with psorosis B virus. The psorosis B challenge inoculum consisted of buds from sweet orange seedlings showing yellow ring spot variegation and etching patterns, similar to the symptoms seen in the Valencia orange trees at Monak from which the original inoculum was taken; and leaf pieces from sweet orange seedlings with yellow ring spot, etching, and variegation patterns similar to the symptoms seen in Washington Navel trees at Monak from which the original inoculum was taken.

Of 100 seedlings inoculated, 32 developed ring spot, etching, and variegation patterns after 17 months. After 20 months, 29 more plants—making

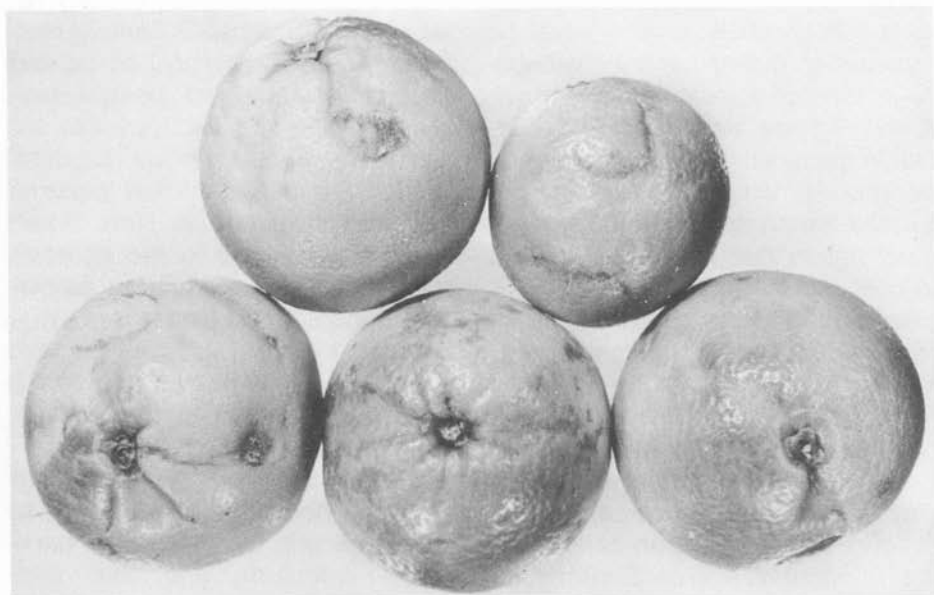


FIGURE 2. Surface rings, bordered by sunken grooves, produced on fruit of sweet orange by the Monak psorosis B virus.

a total of 61—had psorosis B leaf symptoms. Symptoms developed more rapidly and were more severe in sweet orange than in Muscio mandarin seedlings. Crinkly leaf, psorosis A, and psorosis A plus concave gum did not completely protect against psorosis B virus. Often psorosis B symptoms were produced in only 1 branch of sweet orange or on 1 or 2 leaves of Muscio mandarin. Plants inoculated with virus of psorosis B alone developed symptoms within 6 months of inoculation, whereas those previously inoculated with viruses of concave gum, crinkly leaf, or psorosis A took up to 2 years for ring spot and variegation patterns to develop.

#### *Conclusions and Discussion*

The retarded development of yellow ring spots and variegation patterns in seedlings previously inoculated with psorosis A, crinkly-leaf, or concave-gum virus, and the variability in time of appearance of symptoms suggest an interference between these viruses and the Monak psorosis B virus. The reaction of Eureka lemon and West Indian lime seedlings to inoculum of psorosis B implies that the tristeza components also influence the development of symptoms of the Monak psorosis B disease.

Roistacher and Calavan (8) reported the results of graft-inoculation experiments in which concave-gum virus failed to protect sweet orange seedlings against lesion-bark reaction of psorosis A virus. Similarly, by graft-inoculation experiments, Fraser (5) showed that crinkly-leaf virus provided no protection against psorosis

A virus. Wallace (10) found that mechanically transmitted infectious-variegation virus did not protect sweet orange against the psorosis A bark-lesion reaction. Corbett and Price (1) reported that psorosis virus did not protect citrus against local and systemic infection by citrus-variegation virus. These cross-protection tests suggest that concave-gum, crinkly-leaf, and infectious-variegation viruses are not related to psorosis A virus. The only presumed strains of psorosis virus to be transmitted mechanically are crinkly-leaf virus (2) and infectious variegation (6). The failure to date to transmit the other viruses in the psorosis complex mechanically seems to nullify evidence for their relationship to crinkly leaf and infectious variegation.

The identity of the virus disease in New South Wales, provisionally referred to as "psorosis B," and its relationship to psorosis and citrus ring spot virus, have not been established. There are two possible explanations. First, the virus responsible for the persistent leaf patterns and fruit markings in New South Wales is unrelated to the psorosis syndrome although usually associated with it. It may be related to citrus ring spot virus (11) or identical with it. Second, if the Monak psorosis B virus is related to psorosis virus, then Wallace's hypotheses (9)—that psorosis B type symptoms are the response of a healthy tree to initial infection by psorosis A bark-lesion inoculum, and that psorosis A consists of 2 virus components—may require modification. It is possible that there are 3 virus entities

involved in the psorosis A-B disease complex; one component is responsible for the immature leaf symptoms typical of psorosis A, another for bark scaling of the A type, and a third component—the B leaf pattern component—induces ring spots and leaf variegation in mature leaves and depressed rings or grooves in the fruit. The rapidly developing and extensive bark scaling is then the response of a healthy sweet orange seedling to a high titer of the bark scaling component of psorosis A.

There is no conclusive evidence that the viruses responsible for the immature leaf patterns of psorosis A, bark scaling, and mature leaf and fruit symptoms are related, as each of these symptoms can occur in the

field independently of the other symptoms.

If it is assumed that the Monak grove originated from one infected source, then it must be assumed that the budwood contained these 3 virus components and that they were transmitted irregularly. Some trees in the orchard received the B leaf pattern component either alone or in sufficient dominance to hinder the development of the immature leaf symptom and bark lesion components. Only 2 of 200 trees in the grove have shown psorosis A bark-lesion symptoms, and psorosis B symptoms did not develop in these trees. Bud inoculum from these 2 trees did not produce the psorosis A immature leaf patterns in sweet orange and mandarin seedlings.

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