Psorosis in New South Wales, Australia

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Psorosis was first diagnosed in New South Wales (N.S.W.) in 1928 on the basis of bark lesions, as described by Fawcett (1925), associated with deteriorating tree health and productivity. Consideration was given to eradication, but action was deferred pending surveys to determine the extent of the disease. A warning by the Biologist of the Department of Agriculture was issued against the distribution of wood for budding purposes from blocks of trees where the disease occurred.

Surveys revealed the presence of psorosis in all citrus-growing areas of N.S.W., generally in mature to aged trees of varieties of common orange, and in a few Washington navel and Valencia orange clones. The percentage of affected trees was low and eradication was not enforced. Variation in age of trees at onset of bark symptoms, from 12 to 20 years, was noted within blocks in most reports.

In 1949, the Chief Biologist of the N.S.W. Department of Agriculture, Dr. C. J. Magee, reported in a letter to his colleague Dr. Simmonds of the Queensland Department of Agriculture and Stock, that the level of the disease in N.S.W. was not significant, and credit was given to the N.S.W. Bud Selection Society for the maintenance of high standards for freedom from disease required in budwood source trees.

By 1953, all nurseries producing trees for commercial growers were using certified budwood and it was felt that the psorosis problem was diminishing and that the disease would gradually be eliminated without recourse to enforced eradication.

Comprehensive surveys were carried out in Queensland in 1949 and in Victoria in 1949-50. In Queensland, psorosis was noted in a high to very high proportion of Washington navel, Joppa, Valencia and seedling oranges and Beauty of Glen Retreat, Emperor and seedling mandarins. The numbers of seedling trees showing symptoms of bark scaling was high, but production was good. Some of the younger trees seemed to be more severely affected than trees of the previous generation (B. Oxenham, personal communication).

The Victorian survey revealed an overall percentage of infection of 0.6 to 1.5 per cent. Crinkly leaf was reported affecting 3.8 per cent of trees, mostly Eureka lemons. Transmission tests to indicator seedlings were used to confirm visual diagnosis.

In 1955, M. Spurling reported to the 2nd Australian Plant Pathology Conference that in South Australia it was not unusual for 40 per cent of trees in older orchards to show bark symptoms of psorosis.

Some confusion regarding symptoms was evident in the surveys. Several cases of Rio Grande (Florida) gummosis were included in early reports, with special mention of the profuse gumming typical of this disorder. A further cause of confusion was the apparent spread of psorosis-A type bark scaling from one originally affected variety to others in the vicinity. This was noted in a few early records of Coastal orchards in N.S.W. and more particularly in Queensland surveys, where spread of the disease in blocks of mature seedling trees was reported to have occurred in coastal situations. In these cases the scaling was described as atypical, without the pattern of lesion growth shown by psorosis A. Large areas of bark on limbs or butts were covered with small closely pressed scales, the lesions not having the well defined forward-growing edge, with lifting scale margins, typical of psorosis A. This symptom was subsequently found to be eliminated by

winter application of lime sulphur to trunks and limbs for the control of white louse scale, indicating that heavy infestations of this insect pest, and possibly other scales, could in some circumstances cause death and subsequent scaling of outer layers of bark which was sufficiently similar to symptoms of psorosis A to be confused with it.

Lemon crinkly leaf was noted in N.S.W. in 1954 and **concave gum** in 1952, both being restricted to a few old clones. Concave gum was reported to cause slow deterioration of tree health, most trees remained productive and of good general appearance for many years and fruit quality was particularly good. As with psorosis A, variation occurred in symptom intensity and age of tree at onset of symptoms.

Blind-pocket psorosis was not recorded until 1975 and has been seen only in trees of old budlines of Washington navel orange and grapefruit. Psorosis leaf symptoms had been recorded as early as 1955 in these blocks. The trees are still productive and of fair vigor. Death of individual limbs or whole trees, in coastal localities, may follow invasion of deep blind-pocket lesions by the wood-rotting fungus *Diaporthe citri* (Faw.) Wolf. As with the other psorosis strains, symptom intensity and age of tree at onset of pitting varies from tree to tree.

Citrus ringspot has been recorded in two inland blocks and **infectious variegation** has been detected only in indexing of one source of crinkly leaf of lemon.

STUDIES OF FIVE PSOROSIS-AFFECTED BLOCKS

In addition to routine indexing on citrus seedling indicators for confirmation of visual field diagnosis, a more extensive series of transmission studies was made of five orchards of special interest, in an attempt to identify component virus strains present in what appeared to be mixtures of several types.

1. **Stubbs, Pitt Town — A block of 205 trees of Valencia orange on rough lemon stock, aged over 40 years.** When first examined in 1955, all trees showed a pattern of flecks and some faint wavy lines in leaves of the spring growth flush. Two trees had large well developed bark lesions of psorosis-A type. No blind-pocket symptoms were recorded at that time.

In 1979, most trees showed blind-pocket lesions on the main trunk and larger branches. A high proportion also show small areas of psorosis-A type scaling, some around the margin of blind-pocket lesions, or in separate sections of the trunk, or at the cut ends of limbs. These small lesions are transient and frequently callous over within 1-2 years. Secondary wood-rotting fungi invading blind-pocket lesions have caused added damage to some trees. For their age the trees are reasonably productive. Tree-to-tree variation is shown in intensity of all symptoms. Lower limbs of some trees show flattening and distortion, which has been generally found to be present in trees affected with blind-pocket psorosis in N.S.W.

Viruses deemed to be present by visual field observations and indexing are psorosis A, blind-pocket psorosis, vein enation virus, and tristeza.

2. **Keen, Monak (Lower Murray) — A block of 200 trees of Washington navel (old Mildura budline) and Valencia orange (Leng budline) topworked on an unsatisfactory orange variety budded on rough lemon stock planted before 1950.** Symptoms typical of psorosis B were observed in leaves and fruit of 30-40 trees of Washington navel and Valencia Late in 1966. However, the conspicuous bark gumming and scaling described as features of psorosis B (Fawcett, 1932; Fawcett and Klotz, 1938) were not present. Both the old Mildura Washington navel and the Leng selection of Valencia orange were widely grown in the Mildura area and no similar symptoms have been seen in other blocks.

The trees had been topworked at an early age. Similar symptoms on foliage and fruit had been recorded in a particular clone in the Mildura district many years previously and it is probable that

the original trees in the Keen block were propagated from this source, which would explain the presence of the disease in two well-regarded budlines.

Symptoms develop on maturing leaves (principally shaded ones) and are obvious on both surfaces, though paler on the lower surface, and persist throughout the life of the leaf. Small chlorotic lesions and ringspots, about 1mm in diameter, and large translucent areas, sometimes in the form of an oak-leaf pattern, are characteristic. Ringspots and a yellow etching pattern often develop along minor veins. Symptoms consisting of surface rings bordered by sunken grooves also occur on some fruits, 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 normal trees and are less vigorous; they have a squat appearance rather than the rounded habit of normal trees. The lower limbs are flattened and distorted. Ringspot symptoms varied greatly in intensity from season to season and from tree to tree.

Ringspot symptoms developed on indicator seedlings of sweet orange, Orlando tangelo, and sweet lime following bud or leaf-piece inoculation. Ringspot symptoms were sometimes preceded by chlorotic lesions on the young leaves possibly indicating the presence of crinkly-leaf virus. Interveinal chlorosis and vein-banding symptoms were seen in only two sweet orange seedlings.

Few ringspot symptoms were produced on rough lemon and Muscio mandarin seedlings and none on Troyer citrange. Most Eureka lemon seedlings showed the seedling yellows reaction of tristeza, but some did not, and these developed ringspot symptoms. West Indian lime and smooth Seville seedlings developed tristeza symptoms but no ringspot symptoms.

Viruses deemed to be present are psorosis A, citrus ringspot, blind-pocket psorosis, crinkly leaf and tristeza.

3. **Harris, Dooralong — A block of 160 trees of Washington navel orange on trifoliate orange rootstock, planted in 1956.** Initially all trees grew well. At about 10 years, blind-pocket lesions, some of which approached in type those of cristacortis, developed in the crotch region or on the main trunk, and were frequently invaded by the fungus *Diaporthe citri*. Growth of these trees almost ceased, profuse blossoming occurred, new growth was weak and showed patterns similar to those of zinc and manganese deficiency, and leaf yellowing or yellow veins and leaf fall took place. Fruit size was reduced and the affected trees died within about 2 years from the onset of symptoms.

Most trees in the block showed vein banding and interveinal mottle symptoms of psorosis in the spring flush or in indicator plants. Tristeza was the only other virus detected by indexing. The limb abnormality elsewhere associated with blind pocket was present.

4. **Barlow, Narromine.** Old budline Minneola tangelo trees in N.S.W. carry concave gum virus. In this orchard one tree developed both concavities of the concave-gum type and a psorosis-A bark lesion, while the remaining trees exhibited only concave-gum lesions. Some deterioration of vigor was evident in all trees.

5. **Prothero, Wamoon — A block of 56-year-old Marsh grapefruit trees on rough lemon rootstock.** Four trees of 35 examined in 1979 showed bark lesions of psorosis-A type and a small number of trees showed blind-pocket symptoms in the trunk. Trees showing both mild and severe stem pitting from tristeza were present. Indexing onto Ruby Blood sweet orange seedlings produced symptoms of vein banding and interveinal chlorosis. Viruses deemed to be present were psorosis A, blind-pocket psorosis, and tristeza (stem pitting).

INTERFERENCE STUDIES WITH PSOROSIS VIRUS STRAINS

The presence of several types of psorosis virus in individual trees raises the possibility of interaction and suppres-

sion or modification of symptoms. A series of inoculations using combinations of isolates was therefore undertaken.

Nine sources of psorosis virus were used for these interference studies, two of crinkly leaf suspected of being mixtures with psorosis A, one of concave gum, one of concave gum plus psorosis A plus blind pocket and an unusual tiger-eye bark lesion psorosis with oak-leaf patterns from a Bergamot orange intercepted during illegal introduction from Sicily. With the exception of the Bergamot orange inoculum, all inocula used carried tristeza virus, but exocortis viroid was absent. The Bergamot orange carried exocortis viroid, but no tristeza virus.

The nine psorosis sources were inoculated separately into Muscio mandarin and Ruby Blood sweet orange seedlings. After the development of crinkly-leaf or psorosis leaf patterns (approximately 11 weeks), the seedlings, together with 20 uninoculated controls, were challenge-inoculated with ringspot virus (Keen isolate).

Seedling indicators inoculated with psorosis B alone developed ringspot and variegation leaf 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 ringspot and variegation patterns to develop. Of 100 seedlings inoculated, 32 developed ringspot, etching and variegation patterns after 17 months. After 20 months, 29 more plants, a total of 61, showed psorosis-B leaf symptoms. Symptoms developed more rapidly and were more severe in sweet orange than in Muscio mandarin seedlings. Often psorosis-B symptoms were produced in only one branch of sweet orange or one or two leaves of Muscio mandarin.

Prior inoculations, therefore, with crinkly leaf, psorosis A, or psorosis A plus concave gum delayed or partially masked but did not completely prevent development of symptoms of psorosis-B virus.

DISCUSSION

In blocks of trees where psorosis occurs in N.S.W., one or more of the following symptoms are present — immature-leaf patterns of flecking and vein banding, oak-leaf type leaf patterns, psorosis-A bark lesions, concave-gum trunk symptoms, blind-pocket trunk lesions, and very rarely leaf and fruit lesions of ringspot. The range of symptoms varied greatly in each block studied, pointing to mixtures of unevenly distributed components.

A much more marked oak-leaf pattern is characteristic of trees with concave gum.

The identity of the virus disease in N.S.W. provisionally referred to as psorosis B, and its relationship to other diseases which have been included in the psorosis complex, have not been established. The virus responsible for the persistent leaf patterns and fruit markings in N.S.W. appears to be identical with citrus ringspot virus (Wallace and Drake, 1968; Timmer, 1974) and with psorosis-B virus as described by Fawcett (1932), in part. Wallace's (1957) suggestion 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 two virus components, requires amplification. We suggest that there are three virus entities involved in the psorosis-A-psorosis-B complex: one component 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 or ringspot component — which induces ringspots and leaf variegations in mature leaves and depressed rings or grooves in the fruit. The rapidly developing and extensive bark scaling reported by Fawcett (1932) could then be accepted as the response of a healthy, vigorous sweet orange seedling to inoculation at high titre, with the bark-scaling component of psorosis A, as suggested by Wallace (1957).

There is no conclusive evidence that the viruses responsible for the immature

leaf patterns (flecking, oak-leaf) psorosis-A bark scaling, blind pocket, concave gum and ringspot causing mature leaf and fruit symptoms, are related.

If it is accepted that the Monak grove originated from one infected source, then it must be assumed that this budwood contained four virus components (psorosis-A leaf pattern, psorosis-A bark scaling, citrus ringspot and blind pocket), and that they were transmitted irregularly. Most trees showed blind pocket symptoms. Some trees in the orchard received the B-leaf-pattern (ringspot) component either alone or in sufficient dominance to hinder the development of the psorosis-A immature-leaf symptom and of the bark lesion components. The observation that two of the 200 trees in the grove have shown psorosis-A bark-lesion symptoms, and that psorosis-B leaf symptoms did not develop in these trees, is of particular significance as indicating segregation of components of a mixture in the parent tree.

The picture which emerges from the various experiments reported here and in overseas investigations is a rather fluid mixture of viruses or virus strains the individual components of which are variously distributed in the tree and, having various degrees of mutual interference, result in a range of field diseases in progeny trees. Time of onset of symptoms, and type, severity and persistence of those symptoms could be expected to vary from tree to tree according to the nature of the virus or viruses present in the original scion bud.

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