A HALF CENTURY OF RESEARCH ON PSOROSIS[1]

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INTRODUCTION

The name *psorosis* is now used to designate a related group of virus diseases of citrus that have certain characteristics in common, notably the symptoms they produce on young leaves. This name was used by Swingle and Webber (*14*) in 1896, when they first described the lesion-forming disease known at that time in Florida under the general name *gummosis* and in California as *scaly bark* or *scaly bark gum disease*. Later, in 1932, Fawcett (*2*) gave the name psorosis "A" to the commonly occurring form of the disease in order to distinguish it from a less common but more active type which he described as psorosis "B."

In addition to psorosis "A" and psorosis "B," Fawcett *et al.* (*2, 6, 9, 10, 11*) have described four other types or varieties of psorosis: these are *concave gum, blind pocket, crinkly leaf,* and *infectious variegation*. The bark-lesion-forming types are known to have been present in California as early as 1891 (*4*), and these and some of the other types are now distributed throughout the important citrus-growing sections of the world.

Psorosis "A" has been a particularly important factor in citrus production in the United States. The gradual reduction in yield of affected trees and the eventual need for replacing such trees have been costly to the citrus grower (*12, 15, 18*). However, losses from psorosis can be avoided in new plantings of citrus if intelligent use is made of the knowledge that has accumulated during the past half century, largely from the studies of the late Professor Howard S. Fawcett.

We know of no insects that transmit the psorosis viruses. The amount of seed transmission disclosed experimentally has been insignificant (*20*). There is only one chief source of infection, namely, the parent tree from which buds are taken. Bud perpetuation of the disease-causing agent has been known since long before the virus nature of these diseases was discovered. In fact, Fawcett (unpublished) actually produced the disease in healthy trees in 1916 by grafting pieces of lesion bark to them. However, it was not until he discovered the leaf symptoms in 1933 and established their relationship to the bark lesions that he proved psorosis to be a virus disease (*3*).

SYMPTOMATOLOGY

Leaf Symptoms

On citrus trees infected with any of the known strains of psorosis, young-leaf symptoms appear during the growth flushes and vary greatly in extent and degree on individual trees at a given time. Small elongated cleared places of lighter color than the rest of the leaf occur in the region of the veinlets (fig. 1, *A*). The clearing or flecking may be general over the entire leaf or may occur on only parts of the leaf. At times, most leaves of suitable growth show symptoms. At other times, relatively few leaves are affected.

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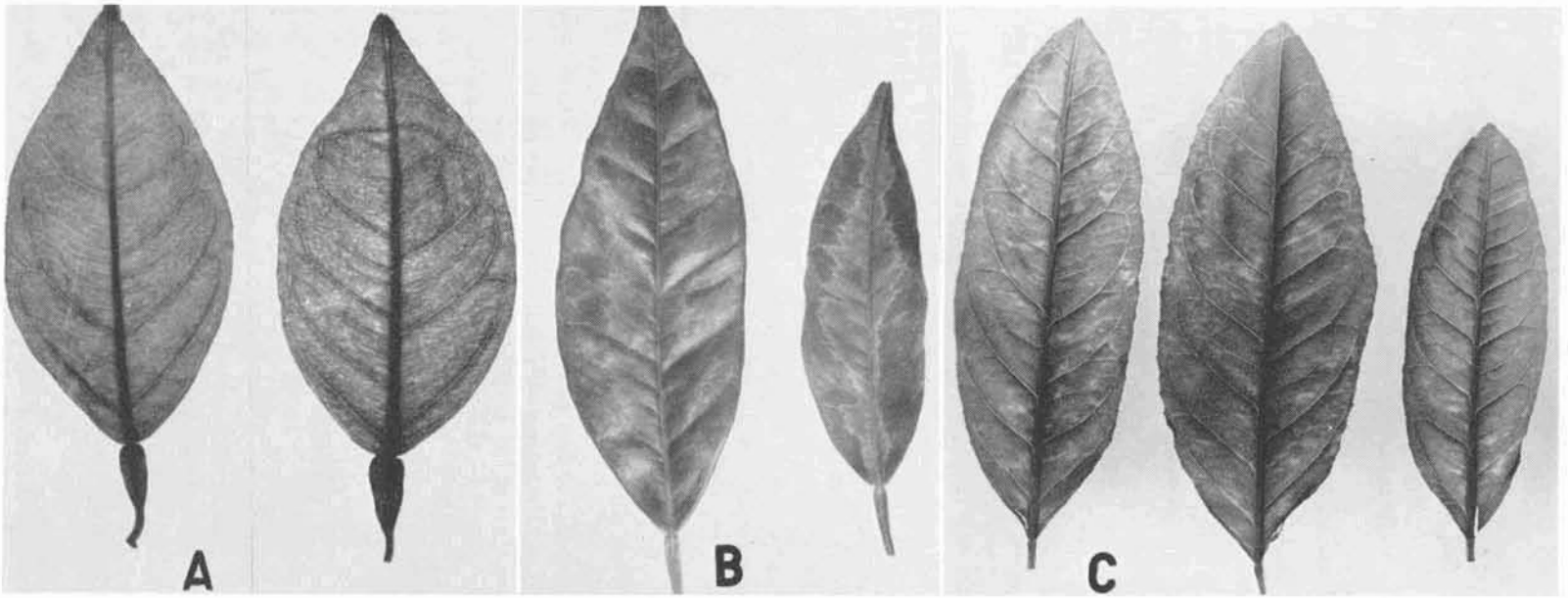

Fig. 1. Young-leaf symptoms of psorosis: A) typical veinal flecking on soft leaves of sweet orange; B) zonate or oak-leaf patterns of concave-gum psorosis on sweet orange leaves; C) young leaves of lemon with psorosis symptoms. Note the more spotted or blotchy effect on lemon leaves (C) than on sweet orange leaves (A).

Some of the small flecks are quite indistinct; others coalesce to form conspicuous blotches, especially on lemon leaves (fig. 1, *C*). The symptoms on the young leaves gradually disappear as the leaves mature. Symptoms are seen most readily when the leaf is shaded from the direct sun and viewed against the light of the sky.

With the exception of concave gum, the various types of psorosis cannot be identified on the basis of the symptoms produced on soft young leaves. In addition to the effects already described, young leaves of trees infected with the strain known as *concave gum* commonly display a zonate or oak-leaf pattern, particularly in the spring growth flush (fig. 1, *B*).

Other Symptoms

For the most part, the psorosis types are identified by other effects they induce. The psorosis-"A" and crinkly-leaf strains of the virus cause the bark lesions commonly known in California as *scaly bark*.

Sweet orange, grapefruit, and tangerine trees infected with either psorosis "A" or crinkly leaf commonly develop the typical bark lesions, but lemon and sour orange trees do not.

Psorosis "A" and psorosis "B" have been considered as being caused by closely related virus strains differing only in virulence (*2, 6, 9*). Psorosis-"B" type is not found often under natural conditions. Recent studies (*19*) indicate that the symptoms originally described for psorosis "B" are merely different manifestations of psorosis "A," resulting from infections from a particular kind of inoculum. Psorosis "B" is therefore no longer considered to be caused by a strain of virus distinct from psorosis "A."

Psorosis "A." Trees infected with psorosis "A" as a result of having originated from a bud from a diseased tree usually develop normally. They will periodically show transitory symptoms on young leaves, but bark lesions seldom appear until the trees are six years of age or older. The average age at time of the first lesion development is twelve to fifteen years. The virus is, of course, present in such trees at all times, and any progeny trees bud-propagated from them will likewise carry the virus and will eventually develop bark lesions. Prior to the discovery of the leaf symptoms (*3*), the absence of bark lesions on older trees was accepted as evidence that the trees were not infected. It is now known that many thousands of nursery trees were propagated from such carriers, and psorosis thus became widely distributed in California as well as in other citrus areas.

The histological aspects of psorosis "A" were studied in detail by Webber and Fawcett (*21*). Fawcett and Bitancourt (*6*) also studied the symptomatology of the bark-lesion-forming types of psorosis and described in detail the gum layers, accumulation of gum in the vessels, and the staining or discoloration in the wood beneath bark lesions. Bitancourt, Fawcett, and Wallace (*1*) studied the psorosis-induced wood alterations in relation to tree deterioration.

The bark lesions begin as pimples or small flakes of the outer bark, which loosen and break away from the live bark underneath (fig. 2, *A*). As the lesion advances, deeper layers of bark become disorganized and some of the tissues become impregnated with gum or gumlike materials. Scaling is more or less continuous, and the lesions increase in size. Portions of the bark die as a result of being cut off by phellogen layers which form below cells bearing the gumlike materials (*21*). As growth continues, the dead bark ruptures and eventually sloughs off in scales of various size and thickness (fig. 2, *B*), with the lesion area gradually increasing. Gum sometimes exudes from lesions, particularly from the margins of the affected area.

Soon after a bark lesion becomes visible, gum layers begin to form in the wood beneath. At intervals the embryonic layer of woody tissue immediately below the cambium is acted upon in some way so that the cells between the medullary rays are

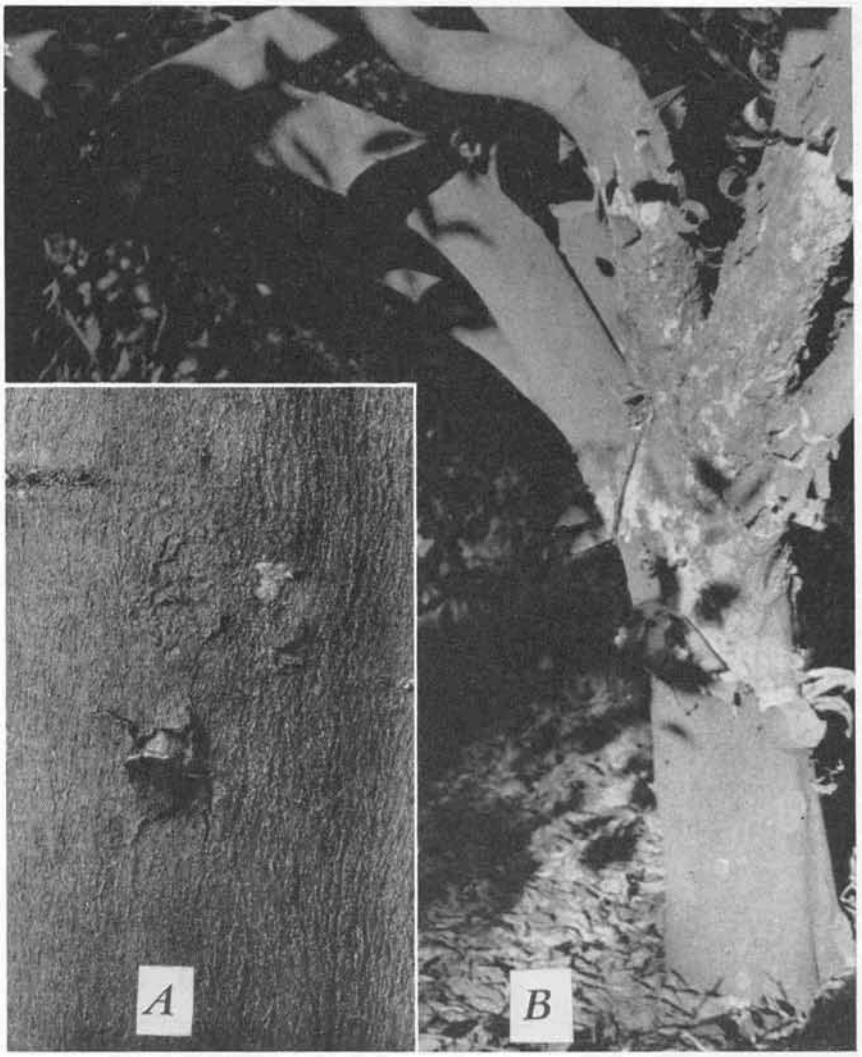

Fig. 2. Psorosis bark-lesion initiation and development on trees of sweet orange: A) initial stages of lesion; B) advanced stage of lesion after several years of activity.

forced apart and partially dissolved (fig. 3, *A*). The pockets become filled with a colorless gumlike substance. Normal wood is then laid down for some time before another gum layer is formed, but this does not follow a regular sequence. The process continues until there are many bands of normal wood alternating with thin layers of gum (fig. 3, *B*). The older layers of gum become buried deeper in the wood with each successive period of wood growth. The gum hardens as it ages and turns dark.

While the gum layers are being formed, gum appears in some of the xylem vessels and accumulates in the vicinity of the perforation plates of the vessels (fig. 3, *C*). In later stages, vessels, wood parenchyma, and medullary ray elements become impregnated with colored gum, and the wood-staining symptom of psorosis appears (fig. 3, *D*). At first, only small areas of stained wood are present, but in the wood under old and well-advanced lesions, staining becomes quite general. At this stage the number of

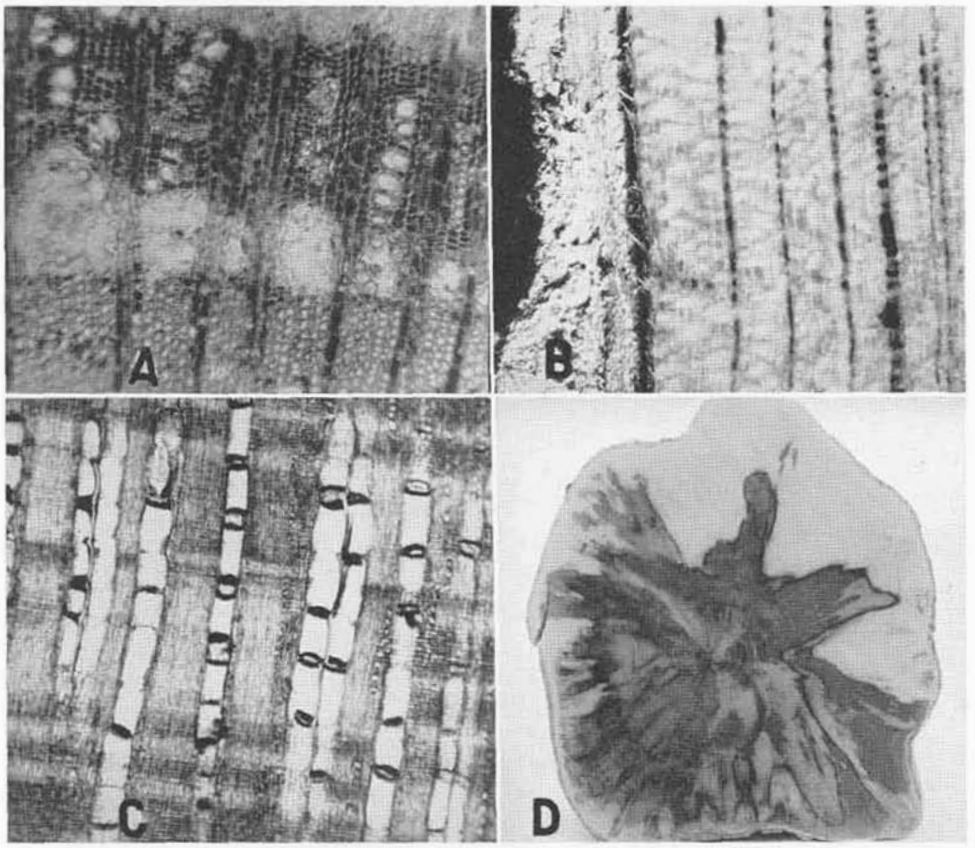

Fig. 3. Gum deposits and wood stain in xylem tissues of sweet orange beneath psorosis bark lesion: A) individual gum layer in transverse section, showing pockets of colorless gum between medullary rays; B) radial section showing series of dark-colored gum layers; C) accumulation of gum in vessels; D) transverse section through trunk of sweet orange tree (note extensive wood stain resulting from a bark lesion that had been active for many years).

functioning vessels is insufficient to supply water to the parts above, and the affected parts of the tree begin to deteriorate.

Concave Gum. In addition to the typical young-leaf symptoms and the oak-leaf pattern, the concave-gum strain of psorosis causes the formation of concavities of various sizes and numbers. The concavities remain open, and fairly normal bark covers the surface (fig. 4, *A*). Cracking of the bark often occurs in the central part or around the margins and gum appears on the surface. In the wood beneath the center of the concavities there is a somewhat limited region where growth has been almost entirely inhibited. Here thin bands of wood alternate with gum layers which can be traced from the center to the periphery of the concavity, where wood development has been normal (fig. 4, *B*).

Some of the xylem vessels in the vicinity of the concavities become plugged with gum, but not as extensively as in the case of psorosis "A." No general wood staining is present. Concavities may be few or many. Injurious effects usually appear slowly and are more pronounced when the concavities are numerous, but infected trees are sometimes stunted even though only a few concavities are present. This type of psorosis has been observed chiefly on sweet orange.

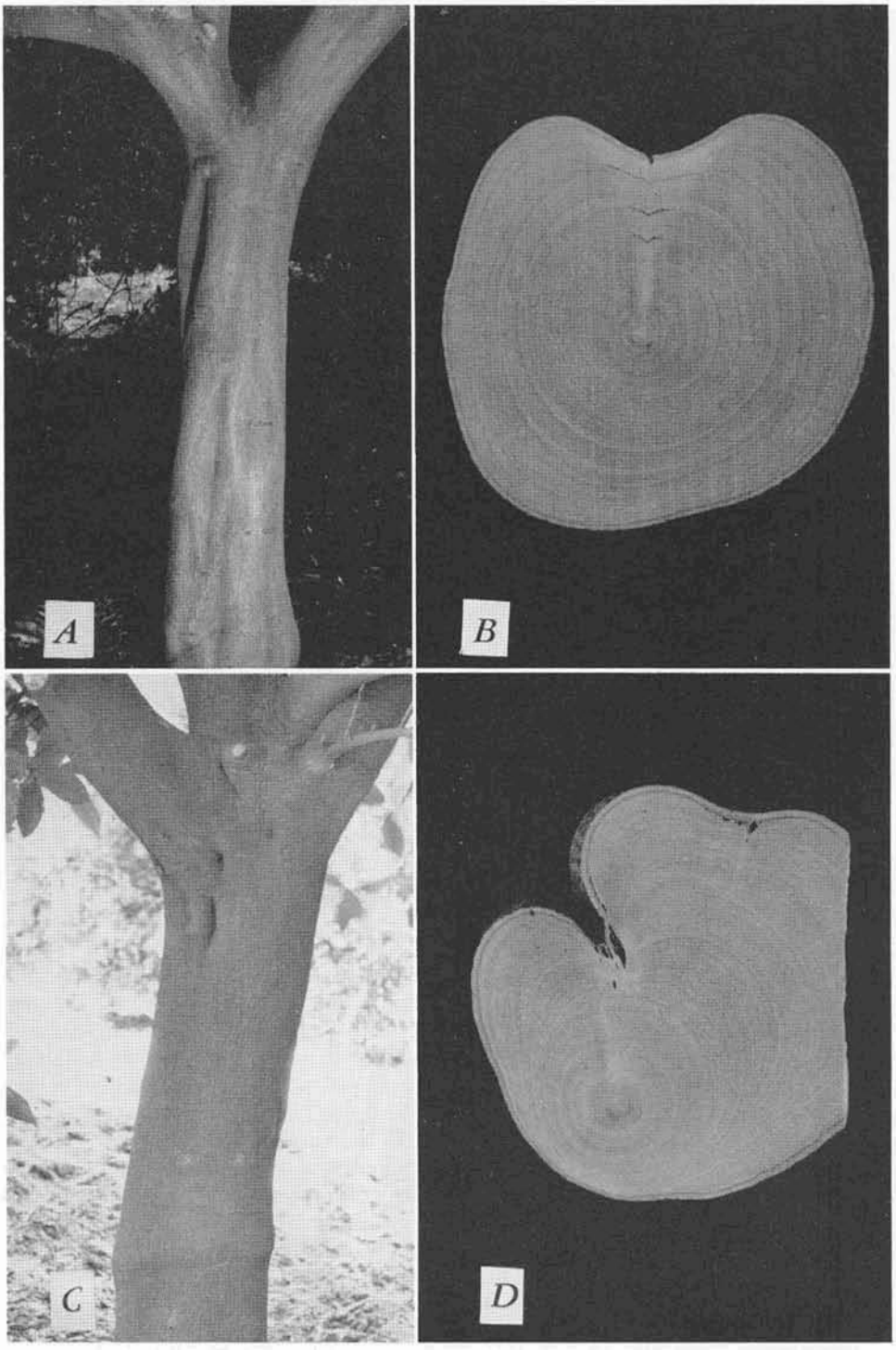

Fig. 4. Effects of concave-gum and blind-pocket strains of psorosis on bark and wood of sweet orange: A) tree trunk showing several large concave-gum concavities; B) wood section through concavity with three prominent gum layers; C) trunk of sweet orange tree with blind pockets; D) wood section showing two blind pockets in different stages of development.

Blind Pocket. Blind-pocket psorosis resembles concave-gum psorosis except that concavities of the former are more numerous and are deeper and narrower. A smaller area of wood is affected, so that as growth continues the depressions become very narrow (fig. 4, *C, D*). The point of origin is often found deep within the wood. There is usually more alteration of affected wood than is found in concave gum. Beneath the depressions one may find cores of tissue, ocherous salmon in color, and of a hard, gumlike consistency. It is not always possible to distinguish between the depresssions formed by concave gum and those formed by blind pocket, but they are considered to be caused by different virus strains because there are no oak-leaf-pattern symptoms associated with blind pocket. This strain of psorosis occurs chiefly on sweet orange but it also induces concavities on mandarin, grapefruit, and lemon.

Crinkly Leaf. Crinkly-leaf psorosis symptoms are seen chiefly on lemons, but this strain also infects other citrus. In addition to young-leaf symptoms, it causes a crinkling or blister effect, symptoms which are retained by the leaves after they become mature (fig. 5). Lemon fruits on diseased trees are often coarse and misshapen. There are no bark symptoms on lemon, but this strain of virus will cause lesions on sweet orange which are indistinguishable from those of psorosis "A."

Infectious Variegation. Infectious variegation, found principally on lemon, also induces symptoms on young leaves similar to those caused by the other psorosis strains. Symptoms resembling crinkly leaf are also found on old leaves of trees infected with infectious variegation (fig. 6). Some leaves on affected trees of lemon and sour orange are severely distorted; others are variegated. White to yellowish patterns of irregular distribution appear on some leaves. This type of psorosis, which may actually be

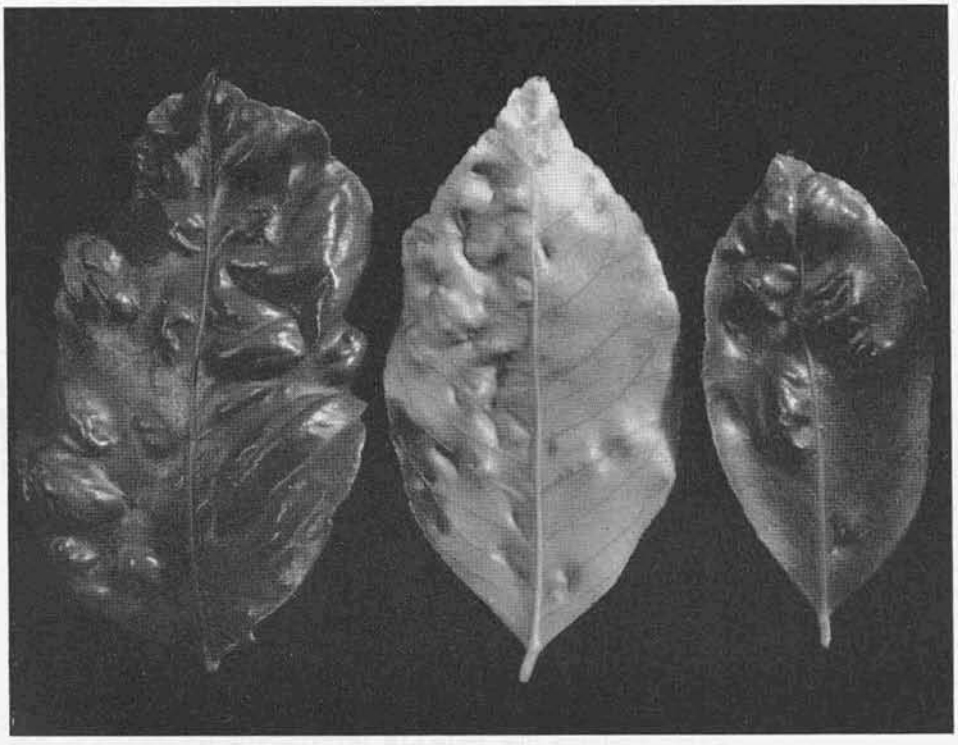

Fig. 5. Crinkly-leaf psorosis symptoms on mature leaves of lemon. The upper surfaces of the leaves have an uneven blistered appearance.

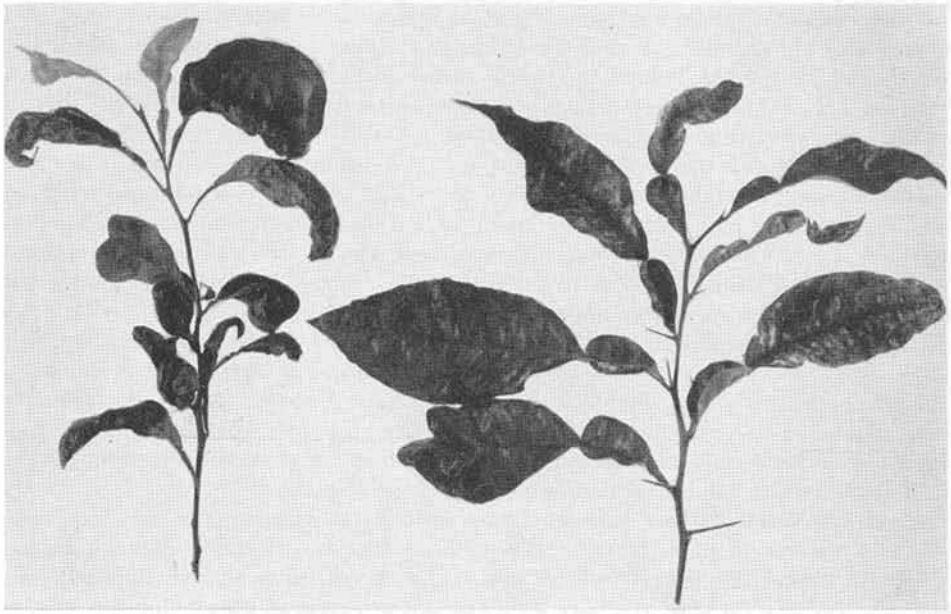

Fig. 6. Symptoms of infectious-variegation psorosis on leaves of sour orange. In addition to severe leaf distortion, portions of leaves display yellowish-white areas or mosaic-like patterns.

caused by severe strains of the crinkly-leaf virus, is not found commonly and is of no economic importance in California. The easily detected symptoms of infectious variegation have no doubt made it possible for propagators to avoid the use of diseased trees.

HOST REACTION IN RELATION TO KIND OF INOCULUM

Fawcett and Cochran (*7*) demonstrated that sweet orange seedlings inoculated by means of bark patches transplanted directly from a lesion of psorosis "A" developed young-leaf symptoms within 4 to 6 weeks and extensive bark lesions within 5 months. They found, further, that infections from normal bark of diseased trees also resulted in young-leaf symptoms soon after inoculation but that bark lesions did not develop on these trees for five years or longer. The writer has made additional studies of this kind with results that lead to the conclusion that psorosis "B," as originally described by Fawcett, is not caused by a strain of virus distinct from the strain or strain complex that causes psorosis "A" (*19*). Instead, it appears that the psorosis-"B" symptom type is the reaction of a healthy plant to infection from lesion inoculum of psorosis "A."

It has been demonstrated experimentally that sweet orange seedlings do not develop the psorosis-"B"-type bark lesions when inoculated from lesion bark if they have been infected previously from non-lesion inoculum (*19*). On such reinoculated trees, however, slowly enlarging localized bark lesions sometimes begin to develop within one and a half to two years around the sites of reinoculation. These lesions are identical with the naturally occurring lesions that develop on psorosis-"A"-infected trees but they result unquestionably from the lesion inoculum used for reinoculation. It has not been determined if additional lesions will originate later at other locations on the reinoculated trees.

The protection phenomenon mentioned above has been used to study psorosis strain relationships. Sweet orange seedlings that were first infected with the viruses of concave gum, blind pocket, crinkly leaf, infectious variegation, and psorosis "A" (non-lesion inoculum) developed no psorosis-"B"-type symptoms when reinoculated from

lesion sources. Thus this protective reaction demonstrates experimentally that these different forms of psorosis are caused by related virus strains, and corroborates the relationships suggested earlier by Fawcett and Klotz (*9*) and by Fawcett and Bitancourt (*6*). The cross-protection reaction has also been used to establish the fact that the causal viruses of psorosis and tristeza are unrelated (*19*).

The reaction of a 16-year-old healthy (psorosis-free) sweet orange orchard tree to inoculation from psorosis-"A" lesion bark is shown in figure 7. Extensive lesion development within the first year after infection is the characteristic response of healthy trees to infection from lesion inoculum. The tree shown in figure 8 was grown from a psorosis-infected bud and was thus infected throughout its life. The systemic presence of virus in this tree provided protection against lesion inoculum from the same source as that used on the tree pictured in figure 7. For a more detailed account of the protective reaction, the reader is referred to a recent publication by Wallace (*19*).

CONTROL OF PSOROSIS

Prevention. Because of the fact that the psorosis viruses are not spread from tree to tree in any significant amount by insects or other agencies such as pruning and cultural operations, and only rarely through seeds, these diseases can be controlled by

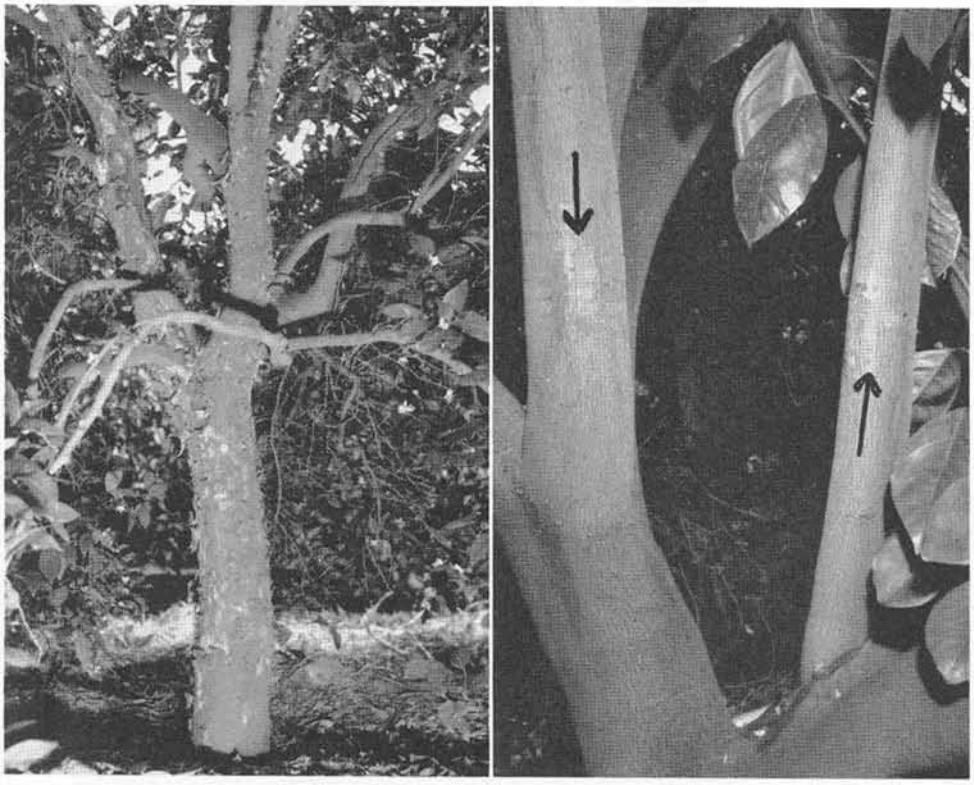

Fig. 7. (Left) Reaction of healthy (psorosis-free) sweet orange tree to graft-inoculation from psorosis-"A" bark-lesion tissue. This 16-year-old tree was inoculated in May 1953 by means of two lesion bark-patch grafts placed in separate primary limbs. Extensive lesion development started within one year. (Photographed May 1958.) Fig. 8. (Right) Reaction of psorosis-infected sweet orange tree to graft-inoculation from psorosis-"A" bark-lesion tissue. This tree was grown from a psorosis-infected bud and had displayed young-leaf symptoms at intervals. Psorosis-"A" bark-lesion patches (see arrows) caused no reaction within five years.

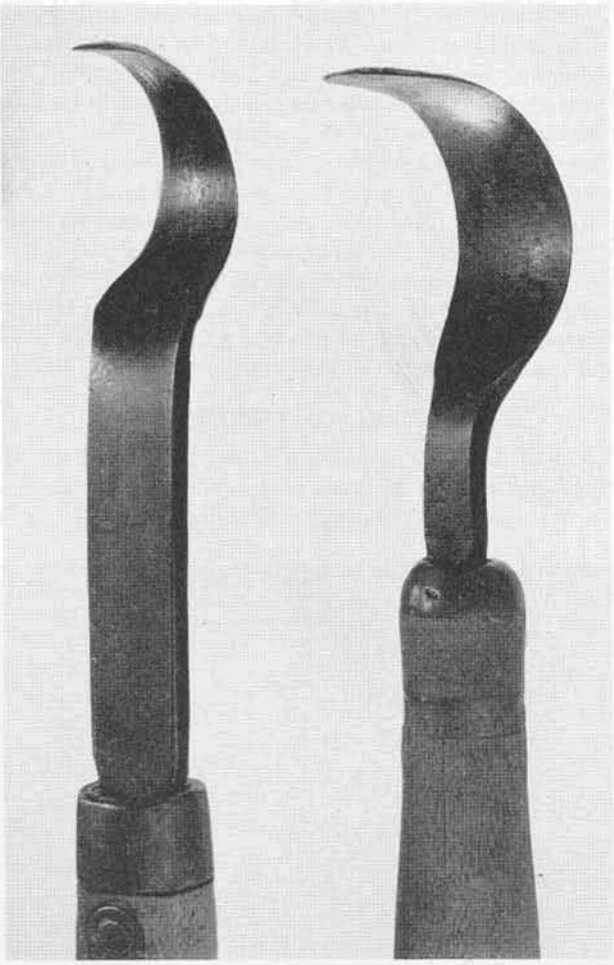

Fig. 9. Specially designed bark-scraping tools for removing psorosis lesions.

prevention. If trees are free of these viruses when planted, they will remain so unless by chance they form a natural root graft with an adjacent diseased tree.

In 1938 the Bureau of Nursery Service of the California State Department of Agriculture inaugurated a program to assist citrus nurserymen in the selection of psorosis-free parent trees from which budwood could be taken for the production of new trees (*5*, *17*). Under this program a selected parent tree is carefully examined by trained inspectors during the spring season, when young-leaf growth provides the most opportune conditions for symptom expression. If no psorosis symptoms are found at that time, additional inspections are made during the summer and the following spring. If it is desired to hasten the registration procedure, or if there is any suspicion of infection, transmission tests can be made on suitable citrus seedlings and final diagnosis can be made within 3 months. The technique described by Wallace (*16*) is used for this means of diagnosis.

Parent trees accepted as being free of psorosis can then be used for the propagation of nursery trees which can be represented as grown from buds from a source registered with the State Department of Agriculture as psorosis-free.

Since this service was established, more than 2,000 parent citrus trees have passed

inspection and have been registered in California. From these, hundreds of thousands of trees have been grown which will remain free of psorosis. During this same time, several hundred trees which would have been used as sources of budwood have been found to be diseased and their use has thus been avoided. In California, one needs only to compare the damage being caused by psorosis in some of the citrus plantings made before the inspection service was available, with the relative freedom from this disease in orchards planted later, to evaluate this prevention program.

Treatment of Bark Lesions. Removal of the bark lesions of the common psorosis-"A" form was at one time a regular practice with some California citrus growers. It has been well demonstrated that if this is done regularly and properly the productive life of most affected trees will be prolonged, but treatment should be made when the lesions are in early stages of development. Old, advanced lesions do not respond well to treatment, and if the disease has reached the stage where the wood beneath the lesions is visibly stained, treatment is of doubtful value.

There are two methods of removing psorosis bark lesions. The first and earliest treatment is known as *hand-scraping*. No records have been found to indicate when or by whom the scraping treatment was first used, but Smith and Butler (*13*), in 1908, recommended scraping of early-stage bark lesions. For some years prior to this date, "surgery" had been practiced on citrus trees with gumming diseases. It was thus natural that efforts should be made to remove psorosis bark lesions by cutting out or scraping away the affected bark.

In this type of treatment, the affected bark and a 15- to 20-cm margin of normal bark are removed by means of specially designed bark-scraping tools such as those shown in figure 9. The bark can be removed to within 1 mm of the cambium, if necessary, to eliminate the disorganized tissues. All tissues of abnormal color should be removed if possible. Where lesions are present on small limbs, the entire limb can be cut out. When possible, the cut should be made some distance below the lesion. Trees should be reinspected every second year. New lesions should then be treated, and if the previously treated lesions appear to be showing reactivity, they should be scraped lightly again.

The tree shown in figure 10 was scraped by hand, as needed, over a period of twelve years. The general condition and appearance of this treated tree is illustrated in figure 11. The benefits of treatment of this tree are apparent when it is compared with the untreated check tree shown in figure 12. The treated and untreated trees pictured were in early stages of psorosis bark-lesion development when selected for experimentation and are average representatives of trees in treated and untreated groups twelve years later. Beneficial response to treatment is dependent upon starting treatment when bark lesions first appear and re-treating when necessary. Lesion activity is so great on some trees, and new lesion development so extensive, that the disease cannot be checked. When this condition arises, treatment should be discontinued and the tree removed and replaced.

A different method of removing the bark lesions has been described by Fawcett and Cochran (*8*) and is known as the Dinitro treatment. The material used is a solution of 1 per cent dinitro-o-cyclohexyphenol in kerosene (prepared and distributed by The Dow Chemical Company under the name DN-75). After removal of the loose bark of the lesions by hand or by the use of a wire brush, the material is applied lightly to the affected parts and to a 15-cm margin of the normal bark around the lesions. A small paintbrush is suitable for applying the material. When applied carefully this material gives good results, particularly on lesions in early stages of development. The dinitro solution penetrates and kills the bark to within close proximity of the cambium. New bark then develops and causes the dead bark to be sloughed off. Figure 13 shows the results of treating with DN-75.

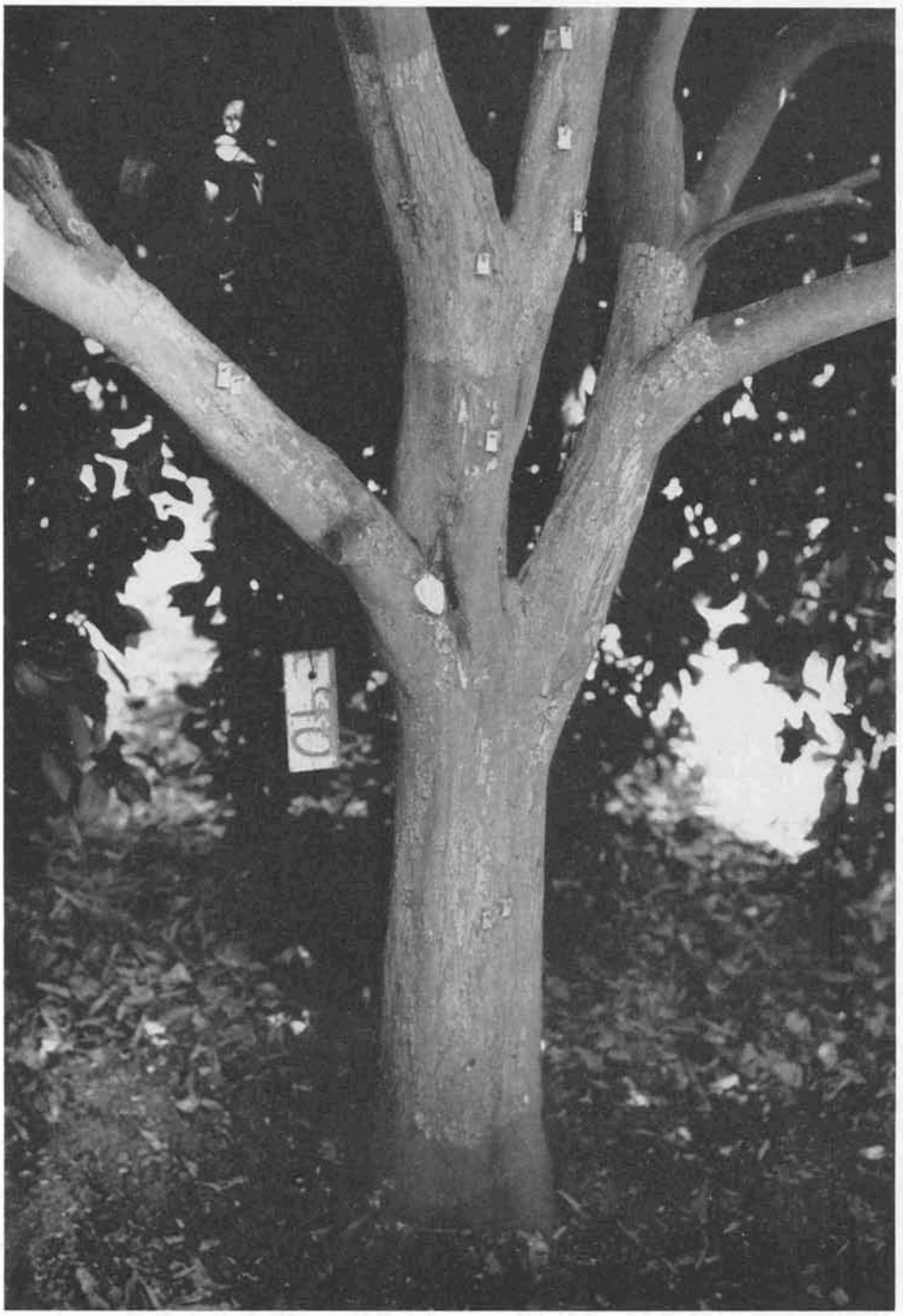

Fig. 10. Psorosis-affected Valencia orange tree which required scraping of the entire framework over a period of twelve years.

Although upon first thought one might not expect beneficial results from removal of virus-induced bark lesions, the explanation of the response to treatment is clear. In the case of psorosis "A," the scaly-bark form of psorosis, presence of the virus in citrus trees appears to have no appreciable effect on the host as long as no bark lesions are present. As bark lesions develop, new functional bark tissues are formed continually. Actually, it appears that the wound-healing and regenerative powers of the citrus tree are quite sufficient to offset the direct injury to the bark. Our studies (*1*) indicate that tree deterioration results largely from secondary effects—that is, from the impregnation of the wood and blockage of xylem vessels by the gum, which, for the most part at least, is produced in the affected bark and then moves into the xylem tissues.

It seems doubtful that treatment reduces the virus quantity sufficiently to account for the resulting lesion inactivity. Removal of diseased bark, either by scraping or by chemical treatment, eliminates some of the virus but not all. We know that virus is still present in the cambium and in the thin layer of bark tissue remaining after treatment. However, experimental studies not yet reported have given evidence that treatment re-

Fig. 11. Valencia orange tree after treatment by hand-scraping for removal of psorosis bark lesions. (This is the same tree as that shown in figure 10.) Photographed twelve years after beginning of treatment.

Fig. 12. Untreated (check) Valencia orange tree in severe decline from psorosis. When the experiments were begun, this tree was in the same stage of psorosis as the tree pictured in figure 11. It received no treatment and within twelve years had reached a stage of unproductiveness.

moves the bark tissues in which most of the gum is formed. Until such tissues are reestablished, and for varying periods of time thereafter, there is little if any lesion activity in the treated area, and the amount of gum moving from the bark into the xylem is greatly reduced.

The length of time a treated lesion will remain inactive varies with individual lesions and with different trees. Some lesions treated while in the initial stage have not become active again after twenty years. Most of them become reactive to some degree within two to five years. There is no good evidence that treatment of bark lesions reduces the incidence of new lesions on a treated tree.

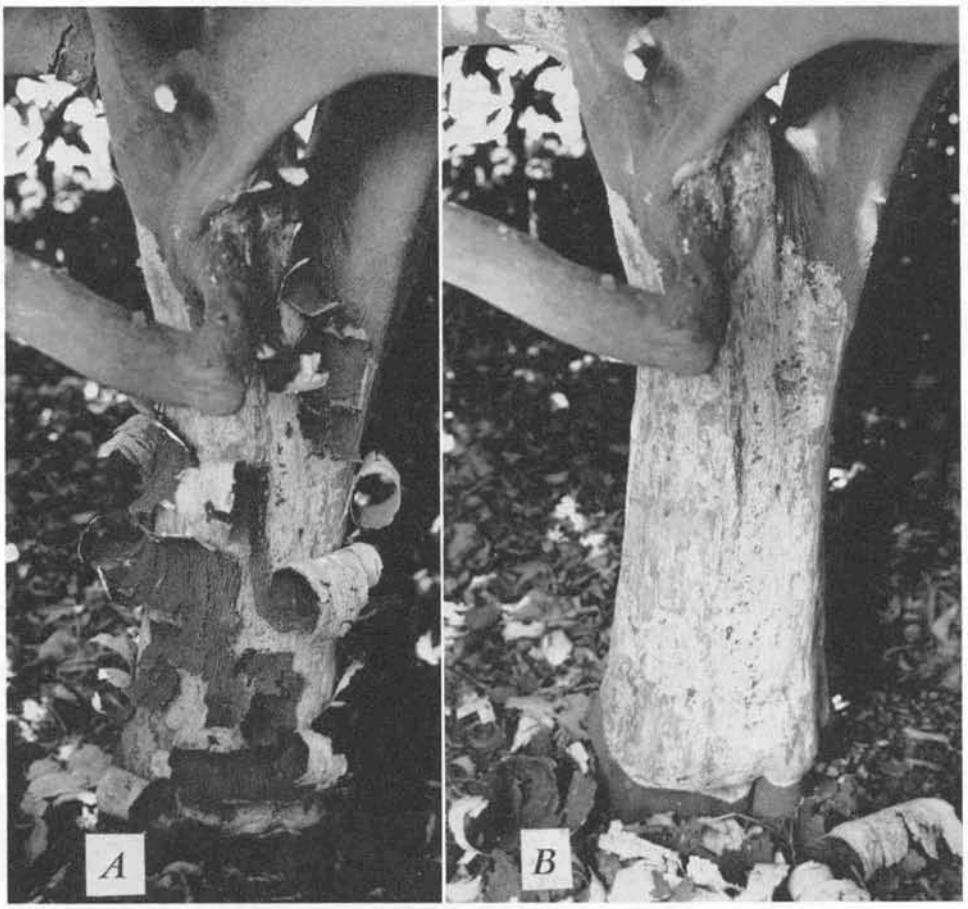

Fig. 13. Results of Dinitro chemical treatment of psorosis bark lesions: A) shelling of bark over and around lesion 15 months after application of DN-75; B) same tree with dead bark removed.

DISCUSSION AND CONCLUSIONS

In this paper it has not been possible to review all of the work that has led to our present understanding of the psorosis diseases of citrus. The pioneer work of Fawcett provided much accurate knowledge of these diseases. Long before his discovery of the virus nature of psorosis, Fawcett had shown that trees propagated from trees having psorosis bark lesions would likewise show bark lesions at some later stage of development. He had also demonstrated that a healthy tree could be infected with the causal agent of psorosis by means of tissue-grafts of buds, bark, or scions from diseased sources. Fawcett learned that lemon and sour orange trees were not subject to bark lesions but that sweet orange, grapefruit, and mandarin varieties commonly developed scaly bark.

Following his discovery of the young-leaf symptom of psorosis "A," Fawcett, on the basis of the common leaf symptom, host range, and identical means of transmission, concluded that the disorders known as concave gum, blind pocket, crinkly leaf, and infectious variegation, were likewise caused by viruses, all belonging in the psorosis group. He demonstrated that the amount of infection arising from transmission of the virus through seeds was negligible and he initiated the first experiments to prove that

virus-free nucellar citrus clones can be developed from parent sources carrying psorosis and possibly other viruses.

The early work of Fawcett and Cochran (*7*) on the symptomatology of psorosis "A" in relation to kind of inoculum led to other studies from which has come a cross-protection technique useful in determining virus-strain relationships. These latter studies have provided some interesting information in relation to initiation and subsequent development of bark lesions. The results of these investigations have been mentioned only briefly in this paper inasmuch as they have recently been published elsewhere (*19*).

Over a long period of years, Fawcett, in cooperation with others,[3] demonstrated experimentally the beneficial effects of the "scraping treatment" of psorosis bark lesions. In later years, he developed the simple, less expensive Dinitro (DN-75) chemical treatment for removal of bark lesions. Perhaps the most lasting contribution resulting from Fawcett's studies of psorosis came from his recognition that because of the absence of natural spread, the disease could be prevented in new plantings. This led to the program initiated in 1938 by the Bureau of Nursery Service of the California Department of Agriculture to assist California citrus nurserymen in the selection of psorosis-free budwood parent trees. Similar programs have since been adopted in Florida, Texas, and Israel. In other parts of the world, as the importance of preventing these diseases becomes evident, the same techniques are being used to some extent to avoid psorosis.

Wherever citrus is of any commercial importance, the consequence of ignoring psorosis should be recognized. In California it is well established that navel orange importations from Brazil were not infected with psorosis. This was also true of Valencia orange and Lisbon lemon. Eureka lemon, which originated in California as a seedling lemon tree, was free of psorosis in the beginning. But man, in his haste to increase the supply of these desirable citrus varieties, and in his ignorance of bud-perpetuated diseases, top-worked these varieties to old, established trees, many of which were diseased, and psorosis was introduced into these new selections. Fortunately, the above-mentioned discoveries by H. S. Fawcett came at a time when psorosis-free sources of these valuable citrus varieties were still available for the citrus industry of California and other parts of the world.

[3] Especially J. C. Perry, Marvin B. Rounds, and Paul Sloop.

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