lencia orange leaves was found in a recent survey (3) of 75 commercial orchards in the major citrus producing areas of the United States.

Summary

Young Valencia orange trees were grown for three years in large outdoor sand cultures on complete nutrient solutions that varied differentially only in the amount of boron. Three rates of boron were applied to single-tree plots. The plots were replicated four times.

No difference in tree size resulted from the differential treatments.

Rather large differences in the boron content of the leaves were induced. The low-boron plants showed mild foliage deficiency symptoms during the second year but not in the first or third years of growth. The high-boron plants showed slight leaf symptoms of toxicity throughout the three-year period.

Mature leaves showed virtually no differences in mineral composition other than the 10- to 24-fold difference in boron. Phosphorus tended to be present in slightly greater concentration when boron was low.

Young leaves showed this same relationship with phosphorus in a more pronounced manner. When the boron supply was low, potassium accumulation in the leaf was retarded and magnesium accumulation accentuated. The rate of calcium accumulation was depressed at the highest boron level. These differences appear to diminish as the leaf approaches maturity.

The only consistent difference in the quality of the fruit produced during the third year was a slight reduction in the ascorbic acid content in the low-boron cultures.

LITERATURE CITED


RIO GRANDE GUMMOSIS

Its Occurrence in Florida Citrus

J. F. L. Childs
Bureau of Plant Industry, Soils, and Agricultural Engineering, United States Department of Agriculture
Orlando

In 1945 G. H. Godfrey published an article entitled "A Gummosis Associated with Wood Necrosis" (4), in which he reported what was presumed to be a new disease attacking citrus trees, principally grapefruit, in the Rio Grande Valley of Texas. This disease is considered by the Valley growers to be their most serious citrus disease.

In November of 1949, in company with Dr. Godfrey and his former assistant Mr. Carl Waibel, I saw the Rio Grande Gummosis disease on the Experiment Station grounds at Weslaco. Several days later symptoms of the same disease were seen on grapefruit trees in the Coachella Valley area of California. Subsequently Mr. Waibel informed the writer that he had assisted Dr. Fawcett in identifying the disease.
in California and that Dr. Fawcett was satisfied that Rio Grande Gummosis is distinct from the virus disease, psorosis. This has an interesting bearing on the early history of gummosis in Florida.

Upon returning to Florida, many gummosis lesions were examined by the writer and were found to resemble closely the trouble seen in Texas and California. Later Mr. Waibel visited Florida and confirmed the suspicion that Rio Grande Gummosis is none other than the old Florida Gummosis disease under a new name. Without going into the complete history of this disease, it should be noted that the earliest detailed description of gummosis in Florida was published by Fawcett in the Agricultural Experiment Station Report of June 1907 (1). Later he published other reports of his work on gummosis, in one of which (2) he explained how to distinguish gummosis from foot-rot (*Phytophthora citrophthora*), and from leprosis (Florida scaly bark disease). Recognition of the importance of gummosis disease in Florida reached its high point when Rhoads and DeBusk published their bulletin in 1931 (5). After that date little was published, and gummosis eventually came to be regarded as merely a name to describe any disturbance giving rise to a little gum.

This situation is the result of a peculiar set of circumstances and events. In the first place some of the symptoms of gummosis are remarkably like certain symptoms of foot-rot on the one hand and like certain symptoms of psorosis on the other. As a result gummosis has been confused with these diseases. In addition gummosis has been known under other names such as "tears," and "gum disease," which led to confusion. Uncertainty as the identity of the causal organism has been detrimental to understanding gummosis. When Fawcett reported (3) that he had isolated *Diplodia natalensis* from gummosis lesions and that Diplodia caused more profuse gumming than any other isolate many were led to infer that Diplodia was the cause of the gumming when neither foot-rot nor psorosis seemed to fit the case. Although Fawcett reported that Diplodia inoculations did not form typical gummosis lesions (3), that fact was overlooked by many. It seems as though it was overlooked by Fawcett himself for when he later recognized the disease in California he did so under the name of Rio Grande Gummosis. However Diplodia infections cause the wood to become dark grey to black in color, which contrasts sharply with the buff and orange color typical of citrus wood infected with gummosis. Also, Diplodia readily attacks sour orange causing profuse gumming, but Stevens (6), Rhoads (5), and Godfrey (4) all agree that sour orange is highly resistant to if not immune from gummosis disease. As a result of these facts there is basis for considerable doubt that Diplodia is more than a secondary invader of gummosis lesions.

**SYMPTOMS OF GUMMOSIS IN TEXAS AND FLORIDA**

The symptoms of the gummosis disease as seen in Texas parallel closely the symptoms in Florida and are in close agreement with those described by Fawcett in 1907. On that basis, the disease as found in Florida, Texas, and California can safely be regarded as a single disease for which the name gummosis, as originally used in Florida, should take precedence.

Gummosis lesions may be active at any time of the year and on lemon trees they appear to be active almost continuously. On grapefruit the period of greatest activity seems to be early spring. This year (1949-1950) the disease was especially active from Decem-
ber through February, perhaps because of an unusually warm winter and an early spring. Since lemons ceased to be grown commercially in Florida (due in large part to gummosis, although foot-rot is usually blamed), gummosis is most frequently seen affecting mature grapefruit trees. Any point on the trunk and larger limbs may be attacked. The following table (Table 1) adapted from Rhoads and DeBusk (5) indicates the relative susceptibility of several citrus species to gummosis.

<table>
<thead>
<tr>
<th>SPECIES OF CITRUS</th>
<th>SUSCEPTIBILITY RATING</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lemon</td>
<td>Most susceptible</td>
</tr>
<tr>
<td>Grapefruit</td>
<td>Very susceptible</td>
</tr>
<tr>
<td>Sweet Orange</td>
<td>Moderately susceptible</td>
</tr>
<tr>
<td>Tangerine</td>
<td>Very resistant</td>
</tr>
<tr>
<td>Sour Orange</td>
<td>Most resistant</td>
</tr>
</tbody>
</table>

There are roughly speaking two types of lesions, depending on age and manner of infection. In appearance, young infections are very similar to young infections of foot-rot, i.e., a small quantity of light-colored gum oozing from a small spot where the bark appears slightly wet or water-soaked. However, the cambial surface of the wood beneath the gumming spot lacks the brownish-yellow stain characteristic of foot-rot infections. Frequently (at least on grapefruit trees) small woody galls or outgrowths from the wood under the bark are found associated with young gummosis infections. These outgrowths are usually green in color due to the presence of chlorophyll presumably stimulated by the disease. So far as is known such outgrowths are not found associated with foot-rot, with Diplodia infections, or with the virus disease, psorosis. Usually there is no bark scaling at the time of first gum production, although the bark may split slightly. Young lesions appear to heal by sloughing off a thin scale of dead outer bark, exposing a buff-colored scar. This occurs shortly after gumming ceases. The scar consists of callus tissue generated by the bark. Healing is only temporary, for later in the year, or perhaps the following year, gum exudes again, and additional scales of bark slough off, thus enlarging the lesion and repeating the cycle. In the course of repeated gumming and scaling, the lesions enlarge to cover a considerable area, and in time the wood becomes exposed. The direction of greatest enlargement is parallel to the axis of the trunk or limb and not around the circumference, as is the case with the psorosis. In addition, psorosis lesions always look ulcerated and give no appearance of healing, even temporarily. In foot-rot lesions the bark is killed down to the wood and is subsequently sloughed off as a single slab, and any healing that occurs takes place at the margins of the lesion.

In older infections of gummosis the disease usually has penetrated deep into the wood, and as a result it is often necessary to chisel through a half inch or more of healthy wood to expose the gummosis infection. When thus exposed the cut surface of the infected wood is seen to be a buff or buckskin color usually banded and bordered with a salmon-orange color that deepens in shade when exposed to the air. The banded appearance is due to the wood of certain growth rings having become impregnated with gum. Frequently gum collects in lens-shaped pockets that cause the outer layers of wood and the bark to become raised as though by large blisters. When these "gum pockets" break through to the surface large quantities of semi-liquid gum are released. The cavities vary in size, some being half an inch thick by an inch wide by two inches long, and the internal walls are usually covered with small gall-like protuberances that some-
times enlarge to the point of filling the cavity. The disease appears to penetrate long distances through the wood so that gum pockets may be formed at a considerable distance from the nearest bark lesion. The importance of the gum pockets in diagnosing gummosis disease was noted by Fawcett in 1907.

A summary of the more characteristic symptoms of gummosis is presented in Table 2, in comparison with the symptoms of foot-rot and psorosis, the two diseases with which it is most frequently confused.

Causal Organism

At present the cause of gummosis must be considered as unknown since there is no published record of typical symptoms of gummosis having been produced by inoculation with a pure culture of any organism or with a virus. The causative agents of foot-rot, psorosis, and Diplodia infection have been satisfactorily disposed of as possible causes of gummosis, and many years ago in Florida Fawcett (3) showed that uninfected mechanical injuries to citrus trees did not gum. It is true that certain chemicals stimulate gum formation, but the remainder of the symptom picture is lacking, i.e., cycles of gumming and healing, gum pockets, and certain other features have not been found associated with chemically induced gumming. The only other causal agent worth consideration at this time is the one reported from Texas. Godfrey found what he describes as an actinomycete-like fungus associated with the disease. Up to the time I talked with him in 1949 he had been unable to obtain this organism in pure culture, but he has been able to cause the disease on numerous occasions by inoculations with chips of diseased wood. Although this organism is suspected, its causal relationship has not been proved.

Control

From the citrus grower's point of view, emphasis on the identity of the causal organism is somewhat academic. What he wants to know is how the disease spreads and how it can be stopped. Old gummosis infections in Florida and in Texas indicate that pruning wounds are the most important point of entry of gummosis, with other bark injuries only slightly less important. In Texas the disease is sometimes referred to as "wet-back" disease because it is so often associated with bark injuries caused by Mexican fruit pickers, "wet-backs," who frequently climb the trees when picking fruit. Whether the organism can penetrate through uninjured bark is not known, though judging from some of the young lesions seen in Florida this year, it seems that it can. However, young infections that take place through the bark are easily cared for, and do not present the same hazard as infections arising in the wounds that

| Table 2. DIFFERENCES AND SIMILARITIES IN THE SYMPTOMS OF FOOT-ROT (PHYTOPHTHORA CITROPHthora), GUMMOSIS (CAUSE UNKNOWN), AND PSOROSIS (VIRUS). |
|---|---|---|---|
| **Disease** | **Foot-rot** | **Gummosis** | **Psorosis** |
| Gumming | Heavy | Very heavy | Practically none |
| Bark Sloughing | Entire bark thickness | Outer scales | Outer scales |
| Gum Pockets in Wood | None | Common | None |
| Color of Affected Wood | Yellow to Brown | Buff with Salmon Bands | Brown |
| **Causal Organism** | Fungus | Unknown | Virus |
result from cutting off large branches. The practice has been to remove large branches by sawing them off as close to the trunk as was convenient and to let the stump heal over as best it could. Even under the most favorable circumstances, it takes several years for a large pruning wound to heal over. In the meantime, the wound is open to infection by gummosis and other diseases.

All pruning wounds three-quarters of an inch in diameter or larger should have a wound disinfectant applied to them. For this purpose few materials are as satisfactory as Avenarius or Red Arrow carbolineum. In addition, any wound 1½ inches or larger should have a coating of water-emulsified asphalt applied to the carbolineum dressing one week afterwards. Such treatment will maintain the wound surface in a dry, fungus-repellant state until the bark has healed over it.

Painting the surface of an old wound will not eradicate gummosis from deep in the wood. Old infections will have to be excavated with a chisel or gouge. All the discolored diseased wood should be removed and, after several days of drying, the surface should be treated with carbolineum and asphalt emulsion as in the treatment of new wounds. When gummosis disease has been established a long time the grower will have to determine whether the tree is worth the expense of treatment. Young lesions are easily excavated and heal over in a short time if proper dressings are applied. However gummosis lesions that have apparently healed over without adequate treatment are still alive and will break out with renewed activity at a later date. The proper treatment of wounds is an excellent example of the adage that an ounce of prevention is worth a pound of cure.

BIBLIOGRAPHY

PRESENT STATUS OF SPREADING DECLINE

R. F. Suit and H. W. Ford
Citrus Experiment Station
Lake Alfred

The investigation of spreading decline of citrus in Florida has been in progress for the past five years. During that time information on the varieties of citrus and the rootstocks on which the decline was found has been reported (1). In addition, the effect of the disease on the tree (1) and the rate at which the decline spreads in the grove have been discussed (1,2). At one time it was considered that the citrus nematode (Tylenchulus semipenetrans Cobb) might be associated with spreading decline (1) but subsequent results showed that the citrus nematode was not the causative agent for typical spreading decline (2). In the experimental work on virus transmission, no evidence was found to indicate that the disease was caused by a virus (1,2). Although preliminary investigations did not indicate that a fungus was responsible (1), it appears that the trouble may be the result of a fungus infection of the fibrous roots that gradually spreads through the grove from root to root (2). Numerous experiments with various types of possible control measures were conducted but no