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THE OLIVE KNOT DISEASE: ITS INCEPTION, DEVELOPMENT, AND CONTROL

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THE OLIVE KNOT DISEASE: ITS INCEPTION, DEVELOPMENT, AND CONTROL[1, 2]

EDWARD E. WILSON[3]

INTRODUCTION

IN 1931 THE COLLEGE OF AGRICULTURE was asked to investigate the olive knot disease, caused by *Bacterium savastanoi* E. F. S., then becoming serious in various districts of the Sacramento Valley. Despite the excellent work of previous investigators, information on many cardinal points in the development of the disease was lacking; and no specific control measures, aside from the removal and destruction of knots, were known. A knowledge of the circumstances under which the knot passes from a rather innocuous, occasionally occurring disease into a widespread and destructive malady was considered important. Consequently, the several factors that might be instrumental in predisposing the host to attack and in favoring the inception and development of the disease were particularly studied. In addition, the possibility of control was considered. The control data, though admittedly not of sufficient extent or diversity to warrant detailed recommendations, are promising bases for trials in various localities.

Most of the work reported herein was done in orchards near Corning, California.

HISTORY OF THE DISEASE IN CALIFORNIA

In 1898 Bioletti[3, 4] reported finding the disease in Merced County and stated that it had been present since 1893. R. E. Smith[23] mentioned its prevalence in the Sacramento Valley in 1907. It did not, however, become serious until 1909, when Smith[24] stated that studies were being initiated. In 1912, Horne, Parker, and Daines[10] investigated a serious outbreak in Sacramento County. Then followed a period when no account of serious damage appears in the records except in isolated cases. One such outbreak developed in Butte County 10 or 15 years ago, although no published record shows how long this lasted or how severe it became. The disease is not known to have become common in Tehama

[1] Received for publication, December 13, 1934.

[2] The writer wishes to express his appreciation to Professors R. E. Smith and M. W. Gardner for advice during the investigations and for aid in preparation of the manuscript.

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County before 1929, the year when it reached severe proportions both there and in Butte County. It has subsequently become prevalent in all olive districts in the Sacramento Valley and in parts of the San Joaquin Valley, but has not been reported as serious south of the Tehachapi Mountains.

VARIETAL SUSCEPTIBILITY

Bioletti[4] reported that the Columbella olive appeared to be more susceptible to olive knot than the Mission. According to E. F. Smith[21] the Nevadillo Blanco and Manzanillo are more susceptible than the Mission. During the present work the Sevillano, Nevadillo Blanco, Manzanillo, and Ascolano have been more affected by the disease than the Mission. The trunks of Sevillano trees have been severely attacked while those of the Mission remained comparatively healthy, even though the disease occurred commonly in the tops. This difference in trunk susceptibility is due in part to the fact that large swellings, which are composed of numerous dormant buds, occur on the Sevillano, while the Mission trunks are generally smooth. These swellings, called "uovoli" by Bioletti[4], each year put forth many suckers. As the suckers emerge they split the outer bark, thereby enabling the bacteria to enter.

Although the Mission olive has proved the least susceptible in ordinary years, following the freeze of December 1932 it developed the disease severely in many cases. This, together with inoculation tests, shows that it is apparently no more resistant than other varieties once the bacteria enter the tissues.

The importance of olive knot in one locality may be influenced by the varieties grown. In one district, for instance, a large part of the acreage is planted to Sevillano trees, while in another district more Missions are grown. This may, in part, account for the fact that the disease did not become particularly serious in the latter district until after the freeze of December 1932, while it was widespread in the former as early as 1931.

IMPORTANCE OF INFECTION CENTERS

Since the bacteria persist in the knots from one year to the next, the disease may spread from any affected tree to others. In examining the reasons for a severe outbreak, one should consider the original source of infection—whether certain trees had harbored the disease for a long time, or whether it was introduced from some other locality. The evidence points to its existence in one district under observation for at least 15 years and not to its recent introduction from elsewhere, since little nursery stock has come into this district for several years, the local nurseries having supplied the demand.

In certain of the orchards under discussion diseased trees were recognized at least 10 years ago, and in such cases the bacteria spread from those earliest infected trees to adjacent trees. These centers are today distinguishable in many instances (fig. 1), there being only a few orchards (mostly young orchards) where the disease is uniformly distributed among all the trees. In other words, the severity of the disease is governed, to no small degree, by proximity to infection centers. Under the next heading this point will be brought out more clearly.

Before the next phase is discussed, however, the possible existence of the disease in other hosts will be mentioned. The information on this point falls into two categories: (1) hosts other than the olive susceptible to *Bacterium savastanoi,* and (2) similar maladies of hosts related to the olive. C. O. Smith[16], who gives the most extensive evidence concerning the first point, found that by inoculation, knots similar to those of olive were developed on Arizona ash (*Fraxinus velutina*), *F. floribunda,* and swamp privet (*Adelia acuminata*). Lesions, but no knots, developed on stems of *Osmanthus aquifolium* and fringe-tree (*Chionanthus virginica*); small galls did, however, appear on inoculated leaves of the latter. Doubtful results were obtained on privet (*Ligustrum ovalifolium*) and jasmine (*Jasminum primulinum*). Smith notes that symptoms resembling those on olive occurred only on those hosts most closely related to the olive. More recently[18] he has proved that *Olea chrysophylla* Lam. is susceptible to *B. savastanoi.* This host, closely related to *O. europea* L., has been introduced into this country from East Africa.

Two naturally occurring, tubercular diseases of hosts related to the olive are known, the oleander knot, and the ash knot. In 1908 E. F. Smith[19] reported failure to obtain infection of the oleander with *Bacterium savastanoi* and suggested that the oleander tubercle might be caused by *B. tumefaciens.* Later, Smith, Brown, and Townsend[22] stated that they believed *B. tumefaciens* bore no relation to the disease. In 1912, Tonelli[25] briefly described the oleander organism but did not name it. In 1926, Ferraris[8] named it *Bacterium tonellianum.* Later C. O. Smith[17], reporting pathogenicity and cultural studies with this organism, found that it was able to attack the olive but that *B. savastanoi* did not attack the oleander. Since the two organisms were very similar in culture, he regarded them as strains of the same species, and named the oleander organism *B. savastanoi* var. *nerii,* apparently not aware of Ferrari's earlier designation.

In England, Austria, Germany, France, and Italy, the European ash, (*Fraxinus excelsior* L.), is attacked by a bacterial disease that is manifested as a canker with some hypertrophy at the margins. In 1933 Brown[6] reported finding the disease on the European ash in Washing-

ton, D. C. Although the causal organism would also attack *Fraxinus americana* L., she was unable to infect either *F. excelsior* or *F. americana* with *Bacterium savastanoi*. In extensive cultural comparisons she found that *B. savastanoi* and the ash organism were similar. Certain consistent differences, however, led her to describe the ash organism as a variety of the olive organism, *B. savastanoi* var. *fraxini*, n. var.

Apparently, therefore, *Bacterium savastanoi* might be harbored in certain hosts closely related to the olive; but surveys around Corning have not shown its presence in these plants.

DISSEMINATION OF THE BACTERIA

Although many practical questions relating to the frequency of long-distance transmission of the bacteria remain unanswered, considerable observational and some experimental data have been collected on spread of the disease in the orchard. Before these data are reviewed, the literature will be cited.

Petri[13], in Italy, asserted that *Bacterium savastanoi* was constantly found in the intestinal tract of the olive fly larvae, *Dacus olea*. This insect does not occur in California. Horne, Parker, and Daines[10] made considerable advances in explaining how the disease was spread through the tree by showing that the bacteria were exuded as a slime to surfaces of knots during rains and were then washed downward, infecting other branches. These workers found no evidence that insects transmitted the disease, but they suggested that birds might carry the bacteria from tree to tree.

The present work has also failed to indicate insects as agents in spreading the bacteria. At the time the disease is most active, insects are hibernating and the few found are hiding in the crevices of the bark. No direct evidence has been obtained that birds carry the disease, although this is one possibility among many.

Pruners, on the other hand, may be instrumental in long-distance spread, since the pruning operations are usually carried out when pruning tools may easily become contaminated. Although E. F. Smith[21] found that the bacteria on agar plates were killed by 30-minute exposure to sunlight, two experiments in the present work showed that contaminated instruments may transmit the disease even after being exposed to direct sunlight for several hours. A number of teasing needles, having been dipped in a water suspension of the organism, were placed out-of-doors in direct sunlight, when the temperature ranged between 27° and 29° C. (81°–84° F). At 15-minute intervals inoculations were made with these needles. Infection occurred even after the needles had been exposed for 3 hours. The bacteria on the underside of the needle were

protected to some extent; but they would find even more protection on pruning tools. As shown by a further series of tests, the bacterial exudate, which had been placed on glass slides and allowed to dry in an incubator with a temperature between 17° and 18° C, contained viable bacteria at the end of a week. Conceivably, during winter pruning operations, contaminated tools might harbor the bacteria for an equally long time.

Probably a common method of transporting the disease for long distances has been the shipment of nursery stock. This can take place even in the face of rigid inspection, since the knots may not be visible at the time. Infections occurring in mid-winter will not develop knots until spring. Trees bearing such infection may therefore be dug, pass inspection, and be planted before the disease is visible. In one case that came to the notice of the writer, the disease developed in a lot of young trees shipped from a distance of 350 miles. One can easily understand, accordingly, how the disease might have been introduced into California.

During this work, considerable experimentation has shown that the bacteria may be spread downward by rains. The bacterial slime became visible in the fissures of the knots 20 minutes after the knots were wet. In one experiment healthy trees were wounded and were placed under diseased trees. A fine spray of water, allowed to fall over the trees for 7 minutes, resulted in heavy infection. This experiment showed that abundant, viable bacteria were present within a few minutes after the knots were moistened. Young potted trees placed under diseased orchard trees in wet weather developed numerous knots, further demonstrating the presence of bacteria during rains.

According to isolation studies, bacteria were present in knots containing live tissue; but they were markedly less abundant in knots that had died, presumably from the freeze, during the winter of 1932–33. Knots on greenhouse trees, never wetted by rain, exuded large amounts of bacteria upon being moistened. Under orchard conditions, therefore, viable inoculum will undoubtedly be present during the first autumnal rain following the hot, dry summer.

Sufficient evidence is at hand to prove the downward spread of bacteria during rains, but comparatively little to show the frequency and extent of lateral dissemination. Presumably the transmission by birds and pruners will be limited only by the activity of these agencies. On the other hand, in certain orchards where pruning has been done annually, the distribution of the disease by this means often appears to be limited. One end of an orchard, for example, may have been diseased for several years, while the other end remains comparatively healthy. Surveys in many orchards have revealed a rather consistent tendency for the disease

to confine itself to spots. This fact, mentioned earlier, is exemplified by the orchard represented in figure 1. The trees at the center of this area are more severely affected than those at the periphery, a situation resulting from the earlier diseased trees' furnishing inoculum for surrounding

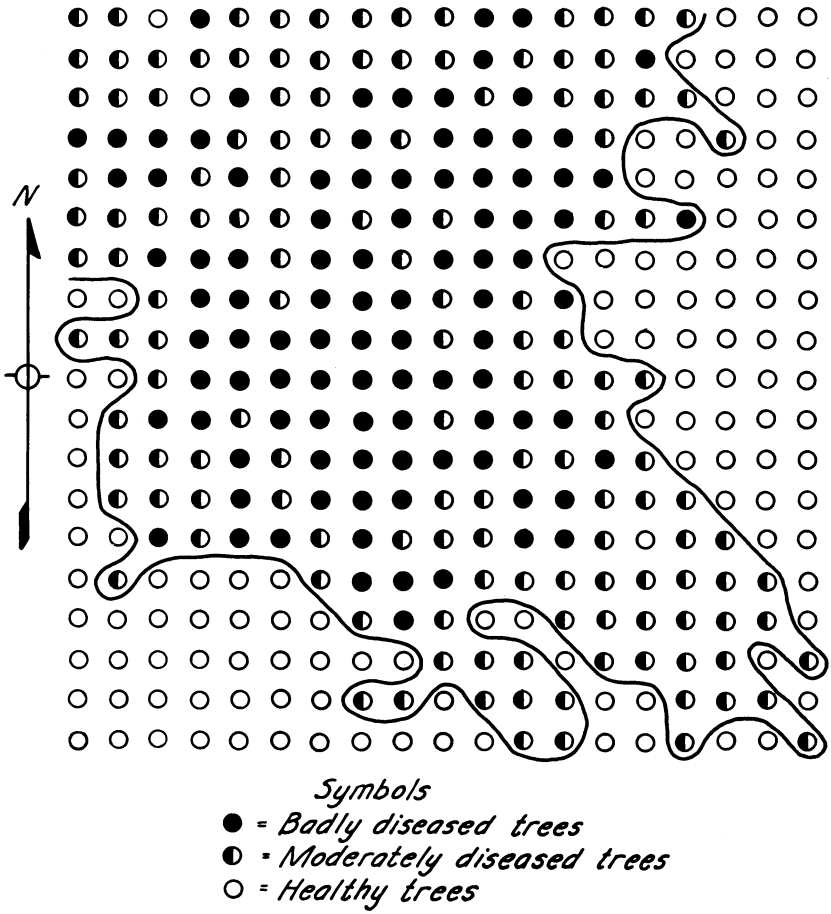

Fig. 1.—Distribution of infected trees in an orchard of Mission olives; mapped in 1932. The affected area is composed of a center of badly diseased trees surrounded by a zone of moderately diseased trees. This area is slightly longer in a north and south direction, the few remaining healthy trees being located on the east and west sides of the orchard.

trees. The extension of the diseased area during two years (1932 and 1933) (figs. 1 and 2) shows that the knot did not affect the entire orchard with uniform severity, even though 1932 was an epidemic year. This orchard, in common with others, contained affected areas somewhat longer in a north-south direction, the greatest number of healthy trees

being on the east and west sides. This phenomenon, often encountered, is considered strong evidence that the bacteria have been disseminated more freely in a northerly direction. Reasons for concluding that bacteria are carried farther to the north than to the south are given below.

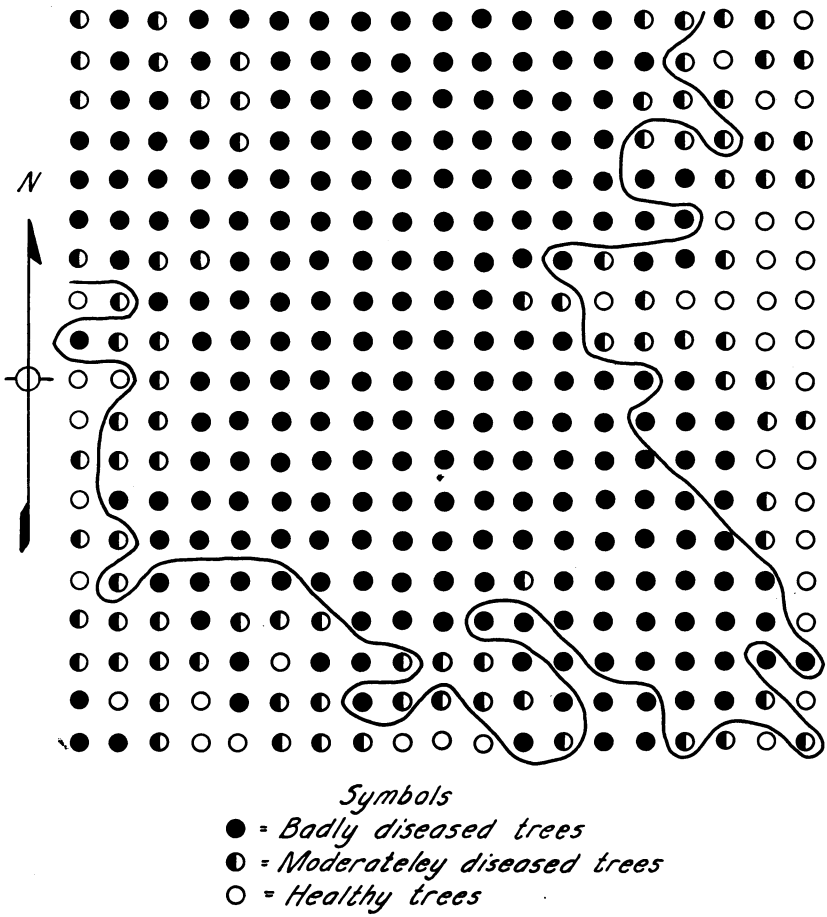

Fig. 2.—Same orchard as that in figure 1, mapped in 1934. The outline of the diseased area present in figure 1 is superimposed on this figure in order to compare the extent of spread between 1932 and 1934. The disease did not affect with uniform severity all of the orchard, even though 1932–33 was an epidemic year.

Figure 3 shows how the disease was distributed in an old grove of Mission olives. To the south of this orchard, separated by a road, is a block of badly diseased old trees; to the north, a younger block, also badly diseased. Several trees on the south end of this central group had become severely affected, while only a few scattered knots occurred in the

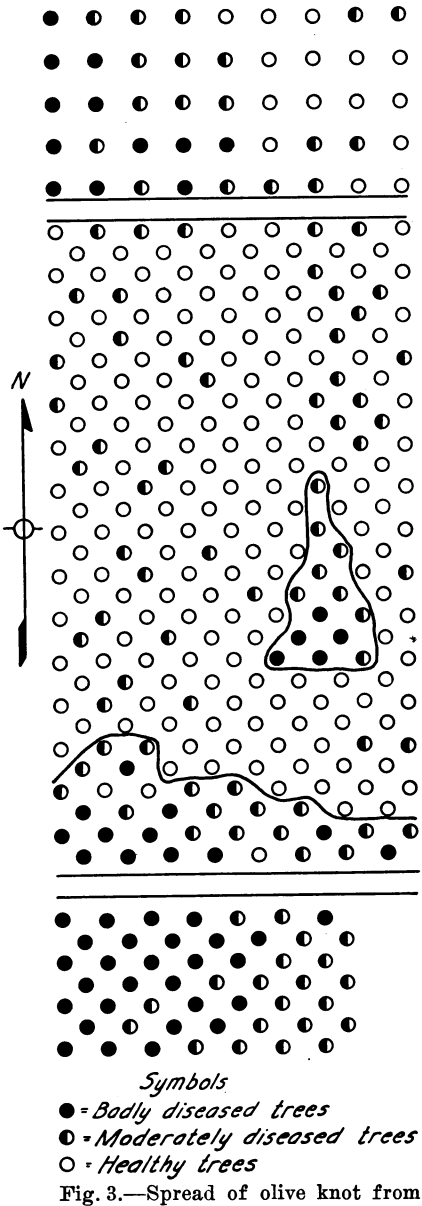

Fig. 3.—Spread of olive knot from one orchard to another. In the center orchard the disease had become more severe on the south end, 50 feet from badly diseased trees, than on the north end, 25 feet from a badly affected orchard. Note how the diseased trees are distributed in the small area near the center of the orchard.

trees along the north border, even though they were closer to badly diseased trees than those on the south. In the center of this orchard five trees had developed the knot severely. Although the disease had spread north of this area, adjacent trees to the south remained healthy.

In one grove, olive knot appeared for the first time in 1933. Only trees located in the northwest corner were affected. Knot had previously been present in the vicinity only on a few old trees, 100 feet to the southeast of this orchard. Apparently the bacteria had spread in a northwesterly direction but not far to the west.

All these observations indicate a disseminating agent that operated in a fairly constant manner. Wind or, more specifically, windborne rains appear to be the only factor that would do this. Since the bacteria are washed from the knots during rains, one may logically assume that wind might carry bacteria-laden particles of moisture for some distance. Two experiments have furnished some proof for this assumption. In one case potted healthy trees were wounded with a sterile knife and were placed 10 feet from the nearest diseased branches; in a second case, 40 feet to the north of diseased orchard trees. After a rainy period, accompanied by wind in each case, the trees were brought to Davis and placed in the greenhouse. The disease developed in several trees in

each instance. Although insects were not excluded from these trees, the experiments were conducted during mid-winter, when no insects were found active. Had they been responsible for carrying the bacteria, one would hardly expect the disease to develop only at wounds made with a sterile knife.

In rainy weather the prevailing winds are from the south or southeast. For example, during the two major infection periods of 1932–33 (December 17 to 23 and January 19 to 29), strong winds from the southeast occurred. The United States Weather Bureau (18 miles north of the experimental orchard) recorded a maximum wind velocity of 22 miles per hour on December 22, and 35 miles per hour on January 24. The potted trees, mentioned above as being located 40 feet to the north of the nearest diseased tree, were present in the orchard during the January infection period.

Although the possibility of dissemination of bacteria during dry weather is not entirely precluded, it does not seem likely to occur, inasmuch as a fresh supply of inoculum would not then be present on the surfaces of knots. Studies on this phase are planned for the near future.

INITIATION OF THE DISEASE

Time of Infection.—As shown by the foregoing discussion, *Bacterium savastanoi* is exuded when knots are wet. A supply of fresh inoculum will not be present on the surfaces of knots during the hot, dry summers. Infection, therefore, will probably not be common during the summer. To establish experimentally the time of infection during fall, winter, and spring, two methods have been employed. One consisted in making wounds at different times on healthy branches of diseased trees, thereby enabling the disease to start at these points. The second method consisted in placing wounded, young, healthy, potted trees under diseased orchard trees and at intervals of time replacing them with others. The potted trees, upon removal from the orchard, were brought to Davis, to avoid further exposure to infection. The first method merely indicated the occurrence or nonoccurrence of infection after the date of wounding and, to some extent, its abundance. The second method definitely established whether infection occurred during the period the trees were exposed in the orchard.

Since the distance to the experimental plots prevented a frequent change, the trees were left in the orchard over relatively long periods. The results, nevertheless, are useful when considered with those of greenhouse experiments. Figures 4 and 5 correlate the data collected by the second method with rainfall and temperature during 1932–33 and 1933–34. As the infection data shown in these figures represent the

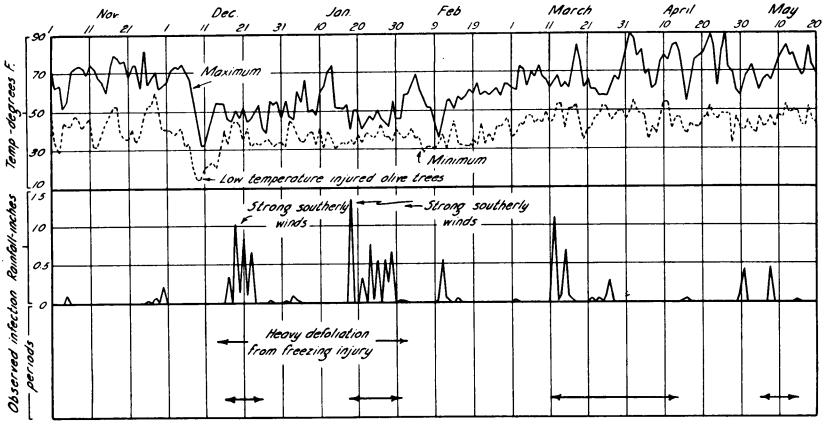

Fig. 4.—Temperature and rainfall during the winter of 1932–33 in relation to infection by *Bacterium savastanoi.*

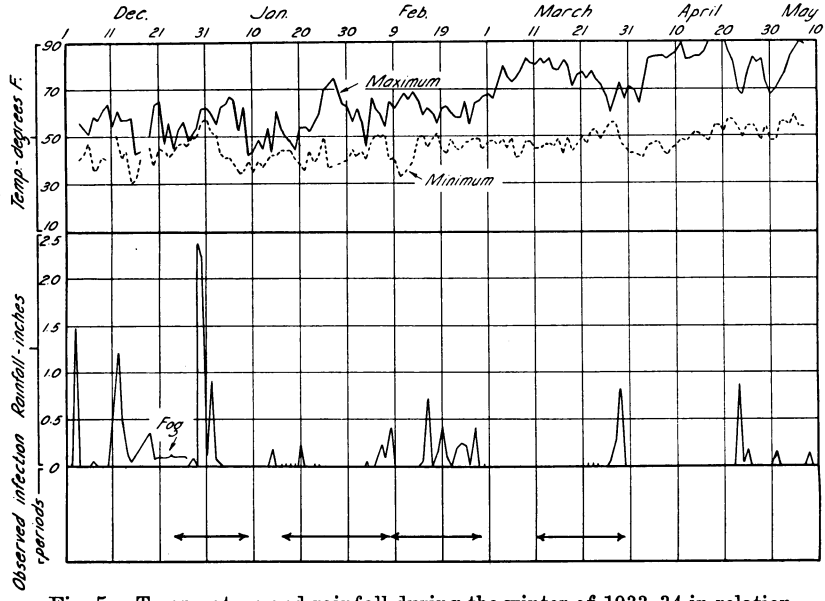

Fig. 5.—Temperature and rainfall during the winter of 1933–34 in relation to infection by *Bacterium savastanoi.*

periods during which trees were exposed and later became diseased, they do not necessarily mean that infection occurred throughout these periods. For example, the disease was probably initiated during the latter part of the last infection period shown in figure 5 and not during the early part.

According to these tests, infection occurred throughout the winter and spring. In 1933 the disease was initiated in blossom clusters as late

as May 16, during a rain that lasted only one hour. It was shown that the major part of infection during 1932–33 and 1933–34 occurred in December, January, and February, coincidentally with the longest rain periods of both winters. For instance, as mentioned earlier, during the winter of 1932–33 a greater part of the disease started in two periods—one from December 17 to 23, the other from January 19 to 29 (fig. 4).

Climatic Factors Favoring Infection.—In the dissemination of the disease, the importance of moisture in bringing the bacteria to the surface of the knots was apparent. Once the bacteria are spread over the surface of the tree, is any particular combination of temperature and moisture necessary for infection? If we study figures 4 and 5 carefully, we see that the disease was initiated under both winter and spring conditions. From December 16 to 26, 1932 (fig. 4), for instance, the mean daily temperature fluctuated between about 30° and 47° F. From May 6 to 16, on the other hand, the mean daily temperature was between 52° and 62° F. With trees in the greenhouse, it was shown that extremely high daytime temperatures will not preclude infection. On August 13, 1934, for instance, young olive trees were inoculated by placing the bacteria on cut ends of leaf peduncles. Within 14 days, definite symptoms had developed, although for 11 of these days the daytime tempertures went above 100° F (38° C), the average minimum temperature being 56° F. During fall, winter, or spring, therefore, the temperature is unlikely to be a limiting factor in infection.

Moisture, on the other hand, might conceivably be a limiting factor, for its absence would prevent movement and multiplication of the bacteria. Of necessity, only the moisture supplied from the outside can be considered, that supplied by the host tissue being an unknown variable. With a view to determining roughly the importance of moisture on the host surfaces to infection, two experiments were conducted. Young, potted trees were wounded in various ways with a sterile scalpel; *Bacterium savastanoi,* in water suspension, was atomized over the wounds; and the trees were placed in a chamber where the humidity was kept high enough to prevent drying and where the temperature varied between 15° and 20° C. At intervals of 1, 3, 5, 8, and 13 hours, three trees were removed from the chambers and were placed on greenhouse benches. All the trees developed knot, regardless of whether they were kept under humid conditions for 1 or for 13 hours. Infection of shallow surface wounds, however, made by lightly scraping the periderm or by cutting leaves from the twigs, appeared to require 3 or more hours of moisture. A later experiment did not entirely substantiate these results. In this experiment leaves were cut off close to the twig, and the bacteria were placed on the wounds. One series of twigs was enclosed in large test tubes

with moist, absorbent cotton for 5 days, and a second series was exposed to the greenhouse air. The air temperature did not go above 30° C for the first two days after inoculation. Enclosing the twigs in test tubes increased infection, since 93 per cent of such inoculations developed knots as against 68 per cent on twigs exposed to the greenhouse air. Apparently, therefore, rapid drying of the host tissue reduced infection but did not prevent it. Numerous inoculations by needle puncture have given uniformly high percentages of infection regardless of atmospheric humidity or moisture on the surface of the twigs. Bacteria that enter deep wounds would therefore be little affected by dryness of the atmosphere. Since conditions during winter and spring are not conducive to rapid drying of the trees, the bacteria would be able to infect during very short rains. Examples were afforded in 1933 when on May 8 and 16 *Bacterium savastanoi* was atomized over the surfaces of blossoms after the terminal blossom on each raceme had been removed. Light showers lasting only about one hour fell shortly after inoculation. About 15 per cent of the racemes developed the disease. An additional example of the ease with which infection occurs was cited earlier. A potted tree, which had been wounded by cutting pieces of bark from the trunk, was placed under a diseased tree; and water as a fine mist was sprayed over the two trees for 7 minutes, after which the inoculated tree was allowed to dry. Practically all the wounds became diseased.

INFECTION COURTS

E. F. Smith[21] states that wounds are necessary for infection. Although he had reference to stem infection, he failed in one experiment to obtain knots by spraying the bacteria on uninjured leaves. It has been commonly observed that wounds made by various agencies were infection avenues. Thus Bernès[2], Pagliano[11], Del Canizo[7], and Petri[14] have reported infection of wounds made by pruning tools, hail, frost, and wind-blown sand. In the present work, wounds made by pruning and cultivating tools were found to be commonly infected.

Probably the most important single factor in the severe development of the disease during 1933 was low temperatures of early December, 1932. As a result of this freeze numerous cracks developed in both large and small branches (fig. 6*A*) and the bark was loosened from the cambium for long distances above and below these cracks. In addition, the trees were badly defoliated; leaves were constantly falling throughout the latter half of December and throughout January. As noted earlier, two periods of rain, accompanied by strong winds, occurred from December 17 to 23 and January 19 to 29. The knots, in consequence, developed in such great numbers that entire tops of trees were killed (fig. 7).

Traveling in Italy in 1891, Pierce[15] noted that knots frequently developed at branch nodes. At first he thought that this came about through infection of the buds in the axils of leaves, but later he decided that the bacteria entered the terminal bud and that the knots developed as the

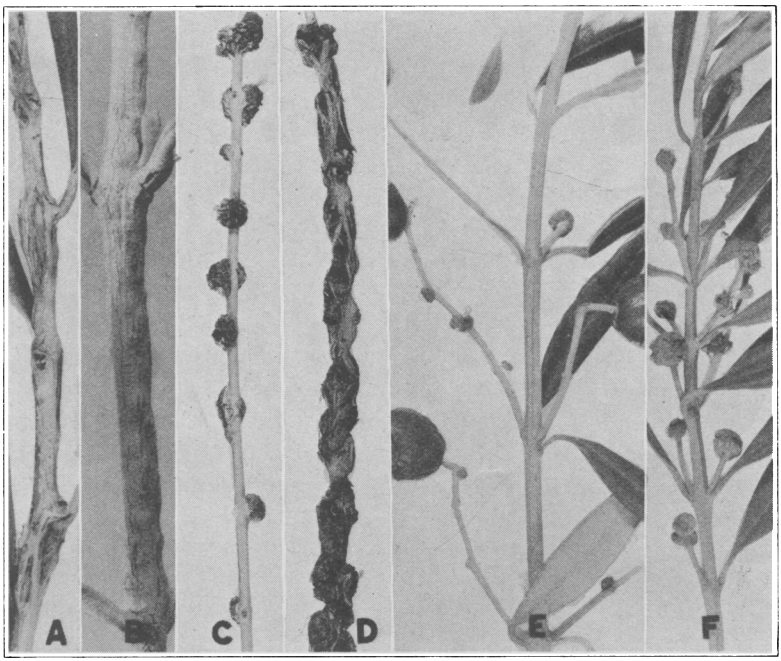

Fig. 6.—*A*, Knots beginning to develop in cracks caused by the freeze of December, 1932. *B*, Numerous knots not yet broken through the bark. The bacteria entered through minute cracks, caused by a freeze. The bark had separated from the wood in many places, enabling the bacteria to move freely along the cambium. *C*, Knots resulting from infection of leaf scars. *D*, Knots resulting from infection of frost cracks. The remains of the periderm covers some of the knots. Compare with *B*. *E*, Infection of blossom scars. Fruit had set in some cases. *F*, Infection of blossom scars. In cases where no fruit is set, the portion of the raceme distal to the knot withers, but the remainder persists for several months.

leaves appeared. Subsequently, however, Horne, Parker, and Daines[10] concluded that the knots at the branch nodes resulted from leaf-scar infection.

Bioletti[4] noticed knots appearing on the large swellings, or "uovoli," common on trunks of certain olive varieties. The knots were generally located at the bases of suckers arising from these areas. Horne, Parker, and Daines[10] noted infection of "growth cracks." The present writer has observed a similar situation. Suckers arising from the trunks break the outer bark as they emerge, leaving a crack through which the bacteria enter. Not infrequently, knots develop at the base of these suckers.

It is well known that the disease occurs also on the roots of the olive, although not so commonly as on aboveground parts.

Horne, Parker, and Daines[10] mention the common occurrence of knots at the scars produced by dropping of leaves. During the present

Fig. 7.—Severe disease in the top of a Sevillano olive tree. Note the distribution of knots at intervals along the branches. This is the result of leaf-scar infection. The tree has been greatly damaged by the disease.

investigation, these points were frequently infected (fig. 6*C*)—more commonly than any others, in fact, during the years when no freezes injured the twigs. For example, counts in one orchard during the spring of 1932 showed that as high as 90 per cent of the new knots on branches occurred at leaf scars.

An heretofore unreported infection of blossom racemes should be mentioned here (fig. 6*E, F*), showing that tissues exposed by the natural

abscission of organs other than leaves are avenues for invasion. Scars may be formed when individual blossoms drop from the raceme and when the entire raceme falls away. To distinguish them, the former are called blossom scars; the latter, raceme scars. Since raceme scars are, of course, formed in the axils of leaves, knots occurring at this point may appear to result from leaf-scar infection if the leaf has fallen. If a raceme sets fruit, it naturally persists until after the fruit is picked in the autumn, when it gradually withers and falls away, leaving a scar. Such natural breaks are designated fruit-stem scars to distinguish them from the raceme scars formed in the spring.

When knots were first found on racemes, blossom infection was considered possible. That it is unlikely is shown by the following experiment. Blossoms that had recently opened were sprayed with a suspension of *Bacterium savastanoi.* In one series the terminal blossom of the raceme was removed to provide an infection court; in a second series the racemes were left intact. Knots developed on about 15 per cent of the wounded racemes, whereas none occurred on the uninjured. According to figure 6 *E, F,* knots occurred regardless of whether the racemes had set fruit or not. In case no fruit is set, but a knot starts at one of the middle blossom nodes, the portion of the raceme distal to the tubercle withers and falls away, while the proximal portion persists. In other words, the developing knot prevents abscission of the raceme at the base, in this respect functioning as does a fruit. Assuming that direct blossom infection would affect the individual blossoms in the same way, some portion of this organ should be present in very young knots; but in no case has this condition been found, the first indication of infection being a slight swelling beneath the blossom scar. Apparently, therefore, raceme knots arise from entry of the bacteria into scars left by unset blossoms and not from entry into the blossom.

Infection of blossom and raceme scars will follow if rains occur while the blossoms are falling. Thus, in 1932, when the Sevillano olive was in full bloom on May 17, rains on May 21–22 resulted in infection of 30 per cent of racemes in certain trees. In 1933, on the other hand, no rain occurred after full bloom, and consequently no blossom or raceme-scar infection developed, even though rains did fall during the early part of bloom.

For some unknown reason, infection of fruit-stem scars has not been common. Two experiments showed that the fruit stems remain attached for 4 to 5 weeks after the fruit is picked. This fact may bear some relation to infrequency of infection.

FACTORS INFLUENCING THE FORMATION AND INFECTIBILITY OF ABSCISSION SCARS

According to observations presented under the preceding heading, every year a large part of infection in branches takes place through leaf scars, showing that infectible scars are present during the fall and winter. Consequently, the importance of susceptible tissue of this nature cannot be overemphasized. Defoliation during the winter of 1932–33 was responsible in no small measure for the fact that Mission trees, heretofore

TABLE 1

LEAF FALL IN SEVILLANO OLIVE TREES

Periods of leaf fall	Leaves off on branches produced in:		
	1929	1930	1931
	Per cent	*Per cent*	*Per cent*
Before January 7, 1932	57.0	17.6	0.0
From January 7 to May 17, 1932*	2.5	2.7	0.5
From May 17 to September 20, 1932	28.5	17.6	2.3

* These trees were in full bloom on May 17.

fairly free of the disease, became badly affected. Conversely, the comparative scarcity of infection courts during the winter of 1933–34 is the only known reason why new disease was rare in the spring of 1934, since the climatic conditions were shown to be extremely favorable. Any studies designed to determine the causes of epidemics must therefore consider the time and conditions under which the leaf scars are formed, together with the factors which determine the length of their infectibility. Blossom and raceme scars should also be considered, even though they are relatively less important than leaf scars.

Since little appeared to be known concerning time of leaf fall in olive trees, studies were initiated in 1932. A series of branches with three years' growth were tagged in January, and a record was kept of the defoliation from each year's growth. The data in table 1 show only a small amount of defoliation between January 7 and May 17, whereas many leaves fell between May 17 and September 20. The heaviest defoliation occurred during and immediately after the blossoming period. The 1931 growth had lost no leaves before January 7, 1932, and only a few up to September 20, 1932; but the 1930 and 1929 growth had lost considerable foliage before January 7 and continued to lose it up to September 20. A series of 80 branches on 8 trees tagged in July, 1934, have been observed at monthly intervals. Considerable defoliation occurred

during July, August, and September but decreased in October. At the present writing (November), occasional yellow leaves may be found in the trees, though they are distinctly fewer in numbers than in October.

Although the defoliation just described can be considered a normal course, some variations within this cycle have been observed when conditions were unfavorable. Trees that suffered from lack of water late in the season lost considerable foliage in September and October. At such a time, and to a considerable extent during the spring, greater defoliation may occur on the south and southwest sides of the trees. Considerable variation in leaf fall between trees in the same orchard appears to result from differences in soil conditions. The orchards in the district under observation are located to a large extent on Tehama loam soils[9], which are rather compact and easily puddled. The subsoil, from 12 to 30 inches below the surface, is usually heavier and more compact than the top soil. Because of the compactness of soil and undulating topography, water tends to penetrate very slowly in certain places and rapidly in others; the low-lying areas are poorly drained, while others dry rapidly. The trees reflect these differences in their manner of growth. Where drainage is poor or where penetration of irrigation water is slow, the trees may suffer during the summer. As a result they may lose more leaves than trees in better locations. Observations in one orchard showed that on one-year-old branches, from which defoliation is usually light, the leaves off varied from 2.3 to 18.1 per cent in the case of trees in a low, wet place, and from 1.9 to 6.4 per cent in the case of trees in a better drained area. Further counts indicated that defoliation is heavier from short terminal than from long terminal growth. On the same tree, for instance, one-year-old terminal growth that was 6 inches or more in length had lost but 1.8 per cent of its leaves, whereas that shorter than 6 inches had lost 10.2 per cent. In other words, if a tree is affected in such a way that it produces a preponderance of short terminal growth, somewhat heavier defoliation will occur from the newer wood than where the terminal growth is greater. The instances cited above were by no means extreme, since in certain other orchards the sparsity of foliage in some trees, as contrasted with the abundance in others, was obvious.

The disposition of certain trees to show greater defoliation may partially explain the following irregularities in disease distribution: (1) that trees sometimes developed the knot badly on the south and southwest side but sparsely on the north side, and that (2) in certain cases the worst-diseased trees in an orchard were known to be at a distance from originally infected areas. The tendency for trees to lose more foliage on the south and southwest than on opposite sides would explain why the trees might develop knots most severely on these sides. Likewise, trees

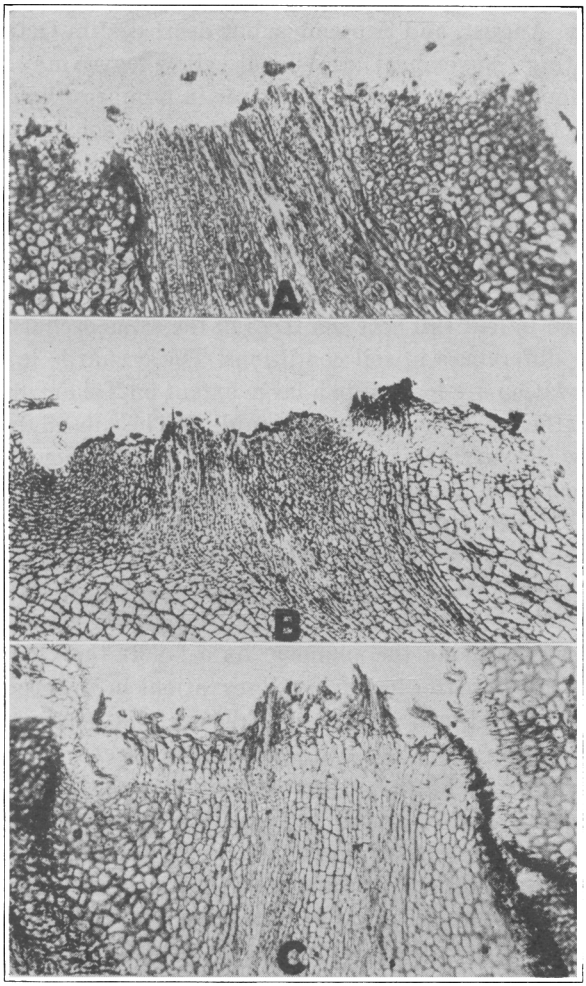

Fig. 8.—*A*, Leaf scar formed the day the fixation was made, showing the ragged ends of tissues. *B*, Blossom scar formed on May 5, fixation made on May 18. The xylem elements are still open at the scar. *C*, Raceme scar in the axil of a leaf. The blossoms on this raceme dropped about May 5; the raceme remained until the fixation was made on May 18, but fell upon being touched. A well-developed phelloderm is present. Compare with *B*.

that through unfavorable soil conditions are induced to drop more leaves during late autumn would be subjected to greater infection.

One point more might be mentioned regarding factors influencing leaf fall. In the spring of 1934 defoliation was greater on diseased branches than on healthy branches. Once a branch is diseased, it is weakened to such an extent that leaves are dropped, a condition which in turn in-

creases the avenues for entry of the bacteria. Under a later heading an apparent relation will be shown between the number of knots and the subsequent increase on uniform-sized limbs. As will be pointed out, several factors, including the effect of disease on leaf fall, might play equal parts in this phenomenon. That limbs might be predisposed to infection through the effects of the disease on defoliation seems to be a logical conclusion.

As shown by the preceding discussion, leaf fall may occur at almost any time of the year, but is most common in the spring and least common in the winter. This raises the question as to when the leaf scars are infectible and how long they remain so. Infection of scars of fruit stem, blossom, and raceme, being sporadic, is of less practical importance, but of great scientific interest. A detailed study is under way, beginning with the anatomical changes leading up to abscission of the leaf, and then following the development of phelloderm over the scar. Only a few preliminary observations on the latter phase are presented here.

Newly formed leaf scars, whether produced by artificial means, such as cutting off the leaf or allowing the soil to dry, or through natural defoliation, developed knots when inoculated. No information has been obtained on how long they remain infectible under a variety of conditions. In one experiment, leaf scars formed on potted greenhouse trees in November were inoculated the following March. No knots developed except at scars pricked by a needle before inoculation. Leaf scars formed during defoliation in the spring of 1934, together with blossom and raceme scars, were fixed and examined. Immediately after the leaf fell, the tissues at the scars were found to be torn, while the ends of the xylem elements were exposed and apparently open. Figure 8*A* shows a leaf scar the day the leaf fell; the ragged, exposed cells would seem to afford a foothold for the bacteria. Within a week, however, a well-developed phelloderm covered the scar. Figure 8 *B*, although picturing a raceme scar, adequately represents the situation in leaf scars. No phelloderm was present on the blossom scars, examined 13 days after blossom fall (fig. 8 *C*). If this condition is characteristic, these scars will probably remain infectible longer than leaf or raceme scars.

Although observations given above are preliminary to a more extensive study, certain tentative conclusions may be drawn. Judging from the rapid springtime development of phelloderm over leaf and raceme scars, these points remain infectible only a short while. Apparently, therefore, these scars are not avenues of entry the autumn following their formation. The one experiment where leaf scars did not become diseased when inoculated four months after formation would support this view. It seems likely that blossom scars might remain subject to

infection longer than either leaf or raceme scars. Assuming these conclusions to be essentially correct, those leaf scars which constitute the common infection courts, must be formed either during the winter when continued leaf fall would provide fresh, infectible tissues, or at an earlier period when phellogenesis is slow. The defoliation studies indicate only a slight amount of leaf fall during the rainy season. The exception, of course, was the winter of 1932–33 when heavy defoliation followed a severe freeze.

In conclusion it might be repeated that presence of infection courts, whether produced by artificial or by natural means, is one of the most important factors governing disease inception. The only apparent reason for sparsity of new knots during the season of 1933–34 was scarcity of infection courts. When these were produced in young potted trees, knot developed in abundance.

DEVELOPMENT OF SYMPTOMS

Although the first visible surface symptom of the disease is proliferation of tissue, removal of the branch periderm around a needle inoculation may reveal a water-soaking and apparent dissolution of tissue. These areas have at times become ¼ inch or more in length after midwinter inoculating of large limbs. Microscopic examination of infected young shoots show that considerable disorganization of the various tissues may occur before visible proliferation of cells begins. Such a disorganization is probably manifested as the water-soaking noticeable around inoculation points. A similar but more extensive symptom is present in the ash disease, caused by *Bacterium savastanoi* var. *fraxini*[6], where rather extensive canker formation is followed by proliferation of the tissues at the periphery.

The development of the knots depends upon growth of the host. Inoculations made in trees exposed to winter temperatures failed to produce knots until spring, although trees placed at temperatures favorable to tree growth produced visible knots within two weeks. No experiments have been performed at controlled temperatures, although inoculations at different times of the year have shown that knots will appear at temperatures much above the minimum for tree growth, provided the bacteria have become established in the tissue. As noted earlier, temperature probably does not limit infection during the ordinary season.

Under field conditions the development of knots has been followed rather carefully. In the winter of 1931–32, fifty branches on ten trees were examined at intervals. Aside from a few knots, probably overlooked during the first examination in the fall, no new development was recorded until April, after which they appeared in abundance. In a

similar series of branches observed during the winter of 1932–33, new knots failed to develop until late March, after which they appeared in such abundance as to prevent accurate counts. These observations agree with the experimental results, which showed that inoculations made in midwinter developed no symptoms until spring.

Certain investigators have described a metastasis, or development of secondary knots from migration of bacteria through the host tissue. According to E. F. Smith[21] (p. 389), "Deep tumors may also arise at a distance from the first tumor and these are due to bacteria which have mi-

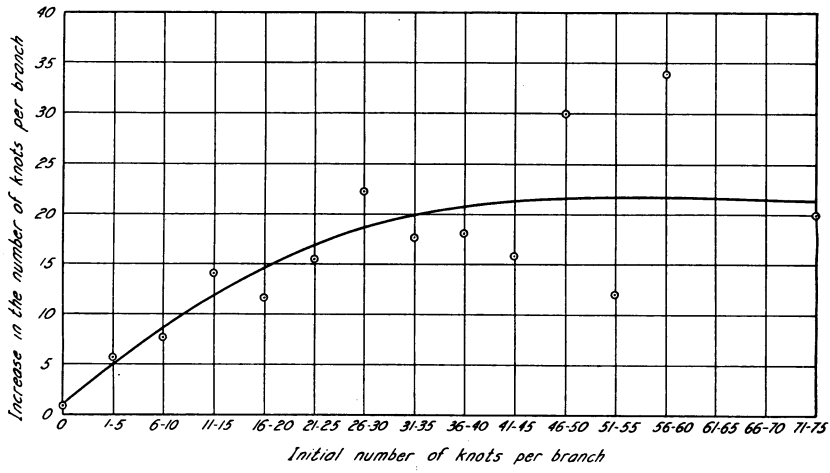

Fig. 9.—Relation of the initial number of knots to their subsequent increase on uniform-sized branches of olives.

grated from the primary tumor by way of the spiral vessels of the inner wood which in such cases are browned, more or less disorganized, and occupied by the gray-white slime of the bacteria." He[20] (Vol. 2, p. 71) further "observed numerous deep tubercles develop at a distance of 1, 2, and 3 feet from the point of inoculation within a period of 7 months in actively growing plants, both down and up the shoot."

According to Bonanni[5] the bacteria spread from the primary infection in the cortex to the wood, and finally, in the case of young twigs, to the pith. Upon reaching the wood an extensive diffusion of the organism through the vessels occurs, resulting in development of a knot at a distance from the primary infection. Should this phenomenon be common under field conditions, it would obviously complicate a control program. Once the branch is infected, there would be little hope of preventing subsequent disease development, either by excision of the primary knot or by spraying. Though the present work has not resulted in final proof one way or the other, the preponderance of evidence thus far has indi-

cated that new knots arising at a distance from primary infection can be traced in many cases, to entry of bacteria from the surface. In the field new knots may arise at or within a few inches of the base of old ones. Some of these may be metastatic tumors, although there is an equal likelihood that they result from surface infection. Considering the data in figure 9, we see an apparent relation up to a certain point between the initial number of tubercles on branches selected for uniformity in size, and the subsequent increase. There are three possible reasons: (1) the proximity of an inoculum source increases surface infection; (2) branches with large numbers of knots drop more leaves than those with fewer numbers, thereby increasing infection courts; and (3) the greater the number of knots the greater the frequency of "deep tubercle" development. The first two factors might conceivably operate in a complementary manner.

Although migration of bacteria through the vessels may result in secondary knots at or within a few inches of old infections, both field observations and some greenhouse experiments failed to show that they develop very far from the primary infection. In the first place, current or one-year-old branches are seldom attacked, even though they arise from badly diseased limbs. When the disease appears on one-year-old growth, it is usually located at leaf scars or at blossom and raceme scars. The current year's growth has not been observed to develop symptoms the year it is produced.

The greenhouse experiments have been even more convincing. Young, vigorously growing trees were on many occasions inoculated beneath the growing tip or at other places. Although some have been allowed to grow for two years, no disease appeared except at the point of inoculation. In one case a tree was inoculated at several places on the main stem about 6 inches below the growing tip. The knots at the inoculation points are now two years old and from 1 to 1½ inches in diameter, but no secondary knots have appeared. Meanwhile the tree has produced lateral branches 2 feet or more in length from points near the diseased area. In two experiments in the spring of 1934, young, vigorously growing branches were inoculated within an inch of the growing tip. These branches have since (four months later) grown from 18 to 20 inches without developing secondary knots. In all these experiments ordinary care has been taken to prevent water from being splashed over the trees, since, as was shown earlier, there is danger of spreading the bacteria to healthy parts even though the water comes in contact with the knots for only a few minutes.

EXPERIMENTS ON CONTROL

In the foregoing discussion an attempt has been made to bring into proper relation those factors which determine the feasibility of available control measures. Spread of the bacteria and initiation of the disease during the rainy season indicate the time during which certain preventive measures might be employed. Breaks in the natural protective layer of the host are infection courts; this fact and the occurrence of such infectible breaks in the form of abscission scars, pruning wounds, and growth cracks indicate the points at which preventive measures should be applied. The large number of quickly available, viable bacteria furnished by the knots already in the tree suggests removal of knots as a desirable method of control. Just how far each of these methods can be followed with beneficial results will be considered here.

Three possible approaches to control have been mentioned in the literature. The first, removal and destruction of knots, has been advised by most investigators interested in the problem; the second, increasing resistance of the trees to infection through fertilization of the soil, was suggested by Paoletti[12], who recommended the use of 2 to 4 kilograms of mineral superphosphates per tree and discontinuance of pruning operations; the third, applications of spray materials to prevent infection, was advocated by Bellini[1], who saw the necessity of protecting injuries produced by hail and who suggested bordeaux mixture as the preventive material. The present writer[26] obtained promising results with three applications of bordeaux. Paoletti[12] has also reported satisfactory control with 1 or 2 per cent bordeaux in four applications as follows: (1) after autumn rains begin, from September 1 to 15; (2) after picking the fruit, at the end of December; (3) at the end of February, to protect hail injuries; and (4) at the onset of the spring rains, from April 1 to 10.

A combination of the first and third method would seem advisable, inasmuch as the removal of knot alone would not insure against its return. Without entering into a discussion that could not be supported by much direct evidence, the writer would regard the second method of control—use of fertilizers to harden the tree tissues, thus making them less liable to injury and consequently to infection—as the least likely to succeed.

Removal and Destruction of Knots.—From a sanitation standpoint removal of all knots from a tree is a logical way of reducing infection; yet this method is limited in its application by the practical impossibility of removing all knots from badly diseased trees, and impaired in its usefulness because severe pruning opens new avenues for infection and

exposes large limbs to injury from sunburn. Actual attempts at a careful cleanup of diseased parts by a number of orchardists, furthermore, have demonstrated the inadequacy of this method; the disease reappeared the following year, in some cases with greater severity. There are two reasons for this: the opening of numerous new wounds by the pruning operations, and the impossibility of removing large numbers of knots from

TABLE 2

RESULTS OF SPRAYING SEVILLANO OLIVE TREES FOR THE CONTROL OF OLIVE KNOT, 1931–32

Treatments*	Increase of knots in three branches in each of 16 trees	
	Number	Per cent
Unsprayed	33	100
Homemade bordeaux, applications 1, 2, 3, 4	6	18
Homemade bordeaux, applications 2, 3, 4	6	18
Commercial bordeaux plus Volck, applications 2, 3, 4	10	30
Lime-sulfur, applications 2, 3, 4	19	57
Zinc-lime, applications 1, 2, 3, 4	15	45
Zinc-lime, applications 2, 3, 4	13	39
Sodium fluosilicate, applications 2, 3, 4	33	100
Unsprayed A†	27	100
Homemade bordeaux, application 3A	17	63

* Homemade bordeaux mixture, 4-4-50 (stone lime); commercial one-package bordeaux, 4-4-50, and 1 pint Volck per 9 gallons of spray; liquid lime-sulfur 1-40; zinc sulfate, stone lime, and water in the ratio of 4-4-50, respectively; sodium fluosilicate, a proprietary product used at the rate of 4 pounds per 50 gallons of water. Dates of spray applications: *1*, September 29; *2*, November 11; *3*, January 6; *3A*, February 25; *4*, March 29.

†The initial number of knots on these trees were counted at the time the application of homemade bordeaux *3A* was made, February 25.

limbs in such a way as to insure against their return and at the same time leave the limb undamaged.

In the face of these limitations the writer feels that, though destruction of all knots through pruning is to be desired, such a radical procedure must be subordinated to one less severe. Where drastic pruning is necessary to remove branches killed by the disease, or those so weakened by large numbers of infections that they are of no further use, a few large cuts to remove entire limbs would be preferable to numerous small cuts. The large cuts could then be covered with a bordeaux paste or some other good protective material.

Prevention of Infection by Sprays.—In the experiments of 1931–32[26] homemade bordeaux mixture, commercial bordeaux plus an oil emulsion (Volck), liquid lime-sulfur, zinc sulfate plus lime (zinc hydroxide) and sodium fluosilicate were tested comparatively. Table 2 gives the results of these tests; and figure 10 shows graphically the increase of disease in individual trees receiving bordeaux, zinc-lime, sodium fluosilicate, and

no spray. Homemade bordeaux appeared to be most efficient in preventing infection. Oil-bordeaux, lime-sulfur, and zinc-lime appeared to be distinctly inferior to homemade bordeaux, while sodium fluosilicate gave no evidence whatsoever of control.

Since bordeaux gave promising evidence of preventing infection, the work was expanded in 1932–33. A series of duplicate plots, receiving

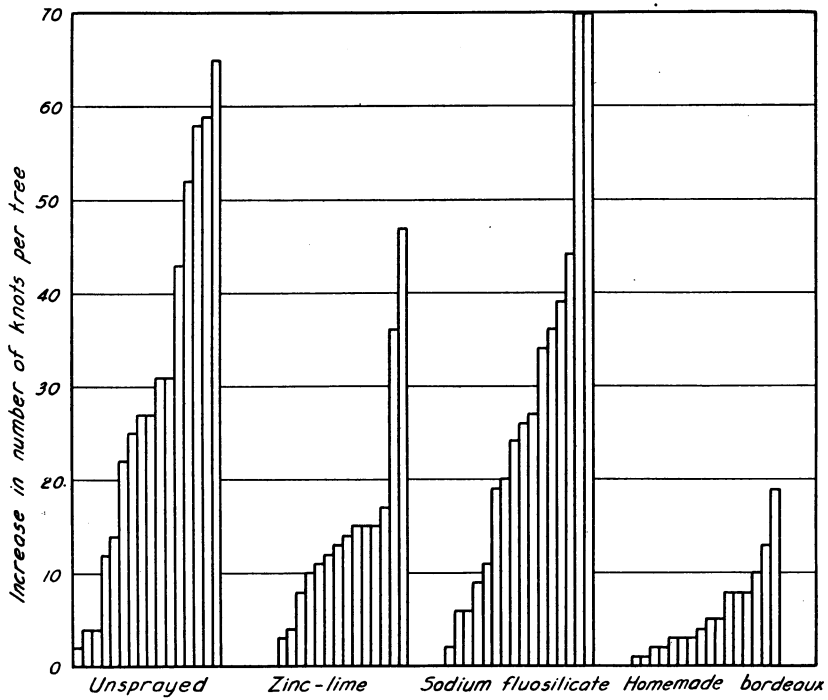

Fig. 10.—Comparison of the control obtained in individual trees receiving different spray materials.

different numbers of applications, was laid out in two districts. The freeze of December, 1932, however, rendered these tests useless, for some of the trees were killed outright, and most of them were severely injured.

In the spring of 1933 two experiments were performed to test the effectiveness of bordeaux in preventing infection of blossom racemes. At the time the blossoms were beginning to open, bordeaux mixture, 4–4–50, was applied to racemes on which the end blossom had been removed to insure an infection court. On a similar series no spray was applied. *Bacterium savastanoi* was then atomized over all of the blossoms in both series. Observations (table 3) made on June 13, before the unset racemes had fallen, showed that there was rather sparse infection of the unsprayed blossoms (15 and 5 per cent in the first and second experi-

ments, respectively) but that on August 15, 68 per cent of the unsprayed racemes in the first experiment, and 24 per cent of those in the second were infected, while the sprayed racemes developed only a few knots. Although the difference in percentage of infection between the June 13 and August 15 readings may have resulted in part from appearance of additional disease after the former date, the greater part of it was caused by the dropping of unset racemes, the total number of which had been used as a basis of computing the June 13 results.

Disease development in 1933–34 was very light. As mentioned earlier, this appeared to result from scarcity of infection courts and not from

TABLE 3

CONTROL OF BLOSSOM-SCAR INFECTION BY BORDEAUX SPRAY IN MAY, 1933

Experiment		Total number blossom clusters sprayed	Percentage of blossom clusters that set fruit	Percentage of blossom clusters infected	
				June 13*	August 15†
First experiment	Unsprayed	326	44	15.0	68
	Sprayed	364	34	0.6	3
Second experiment	Unsprayed	396	48	5.0	24
	Sprayed	473	42	0.4	2

* Total racemes included in these counts.

† Only racemes that had remained were included; those that had failed to set fruit had, in most cases, dropped off. The differences between the June and August observations arise, therefore, from the dropping of unset blossom clusters, although a small difference may have resulted from development of new knots after the June observations.

unfavorable conditions for infection. An orchard of two-year-old trees sprayed with 4–4–50 and 8–4–50 bordeaux failed to develop sufficient disease for test purposes. In other orchards of older trees, where individual branches were sprayed, some data were obtained. In one experiment 30 branches on each of 3 trees were sprayed after 10 leaves had been removed from each branch. Bordeaux 4–4–50 and 8–4–50 were used in one orchard, and 6–6–50 was used in a second orchard. Table 4 shows the uniformly high degree of control obtained with all strengths of bordeaux used. In another experiment several materials were tested comparatively in two applications. The three new materials were used in concentrations that would give the copper equivalent of 4–4–50 bordeaux. The results (table 5) show that bordeaux mixture afforded somewhat better control than copper ammonium silicate and copper resinate and decidedly better control than basic copper sulfate. Application *3* was of very little benefit, since a large part of the infection occurred before it was made. A decided increase in control followed an application of bordeaux on February 7 (application *2*), a fact that emphasizes the need for renewing the spray material at intervals during the winter.

TABLE 4

RESULTS OF SPRAYING FOR THE CONTROL OF LEAF-SCAR INFECTION, 1934

Variety, orchard, and treatment*		Tree number	Percentage leaf-scars infected
Sevillano (Sloan) orchard	Unsprayed	1	27
		2	18
		3	10
	Homemade bordeaux, 4-4-50	1	0
		2	4
		3	1
	Homemade bordeaux, 8-4-50	1	5
		2	0
		3	3
Mission (Heinz) orchard	Unsprayed	1	29
		2	12
		3	14
	Homemade bordeaux, 6-6-50	1	3
		2	3
		3	0

* Spray applied February 7.

TABLE 5

RESULTS OF SPRAYING MISSION OLIVES WITH DIFFERENT MATERIALS FOR THE CONTROL OF OLIVE KNOT, 1933–34

Treatment*	Average increase in number of knots per ten branches	Percentage increase
Unsprayed	13.7	100
Homemade bordeaux (4-4-50), applications 1, 2, 3	2.0	14
Homemade bordeaux (4-4-50), applications 1, 3	4.3	31
Copper ammonium silicate†, applications 1, 3	6.3	45
Copper resinate†, applications 1, 3	6.3	45
Basic copper sulfate†, applications 1, 3	8.7	63

* Applications: *1*, November 24; *2*, February 7; *3*, February 27.

† Copper ammonium silicate (Copocil), manufactured by the California Spray Chemical Co.; copper resinate in an emulsifiable pine oil (Palustrex sulfonate), manufactured by the Wood Chemical Co., Jacksonville, Florida; basic copper sulfate, a so-called basic copper sulfate, manufactured by Marsh Brothers, Oakland, California.

Throughout the work a careful watch has been kept for signs of injury from the spray materials. No injury occurred until the winter of 1933–34, when applications of bordeaux on November 24 caused some defoliation of young trees. This injury is apparently produced only under certain weather conditions, since no injury followed an application on January 30. Rains, beginning on December 10, were followed by fogs that kept the foliage wet almost continuously throughout the rest

of December and most of January. By January 23, after two or three days of sunshine and somewhat higher temperatures, a few trees had lost some foliage. By January 30 increased defoliation was evident. There was no noticeable difference in defoliation between trees receiving 8–4–50 bordeaux and those receiving a 4–4–50 strength. The loss of foliage, though not great enough to injure the trees, was important in that it opened new avenues for infection. Further observations are necessary before the frequency and extent of injury can be ascertained. Judging from past experience, however, defoliation will be of only occasional consequence.

As shown in the third column of table 3, bordeaux mixture 4–4–50 apparently reduced the set of fruit when applied just prior to full bloom. This is of no practical importance since, if sprays are ever used to control blossom-scar infection, they will be applied when the unset blossoms are falling.

SUMMARY AND CONCLUSIONS

Olive knot, caused by *Bacterium savastanoi* E. F. S., has developed in California within the past five years from a level of little importance to one of great destructiveness. Although the disease has been present in the state since 1893, some of the olive districts have not heretofore experienced a serious outbreak.

Starting as scattered infections in a few trees, the disease spread to adjacent trees, thus gradually enlarging the affected areas.

The possibility that hosts other than the olive might be harboring the disease is considered unlikely, since none of these hosts were found affected near the localities under observation.

No commercial olive variety has proved immune to olive knot. The Mission, heretofore the least affected, became badly diseased following a freeze in December, 1932. The Sevillano, Nevadillo Blanco, and Manzanillo varieties are highly susceptible.

Dissemination of the bacteria for long distances may be accomplished by shipment of nursery stock and by pruners moving from orchard to orchard. Some experiments have shown that at moderately low temperatures and under dry conditions the bacteria may survive in the exudate for several days.

No evidence has been found that insects commonly transmit the disease.

Confirming earlier work, experiments showed that the bacteria are exuded to the surface of knots and are spread downward by rains. The exudate may become visible within a few minutes after the knot is wet. Further experiments showed that bacteria escaped from the knots and

infected healthy limbs below when a fine mist of water was directed over experimental trees for 7 minutes. Bacteria were abundant in knots with live tissue but were markedly less abundant in knots which had died recently.

In a number of orchards the lateral distribution of the disease is apparently more rapid in a northerly direction. The observational and experimental evidence suggests that wind-borne rain may be the responsible agent, since the prevailing winds during rainy weather are from the south and southeast.

A detailed field study showed that the disease is initiated during almost any rainy period but that the greatest amount of infection occurred during the longer rains of midwinter. Greenhouse studies indicated that temperature, within the range encountered during most winter and spring seasons, probably will not limit infection. Further tests indicate that, although it is not necessary for the surface of trees to be wet after inoculation of deep wounds, a rapid drying reduced, though it did not prevent, infection of shallow wounds.

Wounds of some sort are apparently necessary for infection of branches. During this work, freezing injuries, pruning wounds, and bark cracks produced by emergence of suckers proved to be common avenues for entry of the bacteria. In addition, scars produced by the dropping of leaves, individual blossoms, and racemes were attacked. Since, during ordinary years, the greatest amount of new knot on branches develops at leaf scars, the time and conditions governing formation of these scars were studied. These studies indicate that drought and the disease itself may increase defoliation and consequently infection.

According to preliminary microscopic studies of phellogenesis at leaf scars, those formed during the spring are not infectible the following autumn. Apparently, therefore, such leaf scars as are attacked by the bacteria during winter must be formed either during the winter, when continued leaf fall would provide fresh, infectible tissue, or at an earlier period when phellogenesis is slow.

Under field conditions, external symptoms in the form of knots did not develop during the winter. Even when infection occurred in December, the knots did not appear until spring. Trees placed under temperatures favorable to growth, however, developed visible symptoms in two weeks, merely demonstrating the fact that growth of tubercles depends on growth of the host, which, in turn, depends upon temperature.

Consideration has been given to the importance of metastasis, or formation of secondary knots resulting from migration through the host tissue of bacteria from a primary infection. Both observations and ex-

periments tended to show that the development of secondary symptoms at a distance from primary infections is not of common occurrence.

The important reasons for the recent widespread outbreak in the Sacramento Valley appear to be: (1) the presence of the disease in most localities for a number of years; (2) the presence of highly susceptible olive varieties, such as Manzanillo and Sevillano; (3) the dissemination of the bacteria by pruners, by wind-borne rain, and, to some extent a few years ago, by diseased nursery stock; (4) and a freeze during December, 1932, resulting in splitting of the bark and defoliation during the rainy season.

Studies on control have dealt largely with prevention of infection by spray applications during fall, winter, and spring. The following spray materials have been tested: homemade bordeaux mixture, commercial bordeaux plus a spray oil, zinc sulfate plus lime, lime-sulfur, sodium fluosilicate, copper ammonium silicate, copper resinate in an emulsifiable pine oil, and a so-called "basic copper sulfate." Homemade bordeaux mixture, in strengths of 4–4–50, 6–6–50 and 8–4–50, prevented infection to a considerable degree. All of the other materials, with the exception of sodium fluosilicate, reduced infection somewhat, but none appeared to be so effective as bordeaux.

Although further work is necessary to determine the number of applications necessary for the most efficient control, such data as are available indicate that the spray must be renewed at different times during the winter and spring, the first application being made in the fall before the rains begin.

Defoliation occurred to a moderate extent following an application of bordeaux 4–4–50 and 8–4–50, prior to a prolonged period of rains and fog. A second spray, however, applied after this wet period, produced no injury. The damage from this cause is not considered great enough to warrant objections to the use of bordeaux.

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