PECAN ROSETTE

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HISTORY AND DISTRIBUTION

Rosette has been rather generally recognized by growers as a serious disease almost from the inception of commercial pecan orcharding. As early as 1902 requests came to the United States Department of Agriculture for an investigation into the causes of the disease and possible methods of control. The work was at once undertaken by the senior author and carried on for about four years in connection with other work in the Southern States, but between 1906 and 1910 little attention was paid to the disease. Since 1910, and more particularly during the seasons of 1912 and 1913, the experimentation has been continued by the junior author.

The disease is well distributed over the pecan-growing territory from Texas to the Atlantic coast and from Florida to Virginia. (See fig. 1.) It has been definitely seen by one or the other of the authors at Whittier, Cal.; San Antonio, Boerne, Waring, Kerrville, San Saba, Waco, Austin, McKinney, Tex.; New Orleans, La.; Ocean Springs, Miss.; Atlanta,  

1 The work of the junior author was carried out while he was employed as scientific assistant in the Office of Fruit-Disease Investigations, Bureau of Plant Industry.
Statesboro, Albany, De Witt, Baconton, Thomasville, Cairo, Valdosta, and Blackshear, Ga.; Bellevue, Palatka, Sisco, Gainesville, St. Augustine, Jacksonville, Mcclenny, Glen St. Mary, Alachua, Lake City, Monticello, Newport, and Tallahassee, Fla.; Mt. Pleasant, Denmark, Bamberg, Greenwood, Blackshear, Orangeburg, St. Matthews, Fort Motte, Cameron, Sumter, Summerton, and James Island, S. C.; Durham, N. C.; and at Eastville, Va. Besides personal observations at the places above enumerated, specimens of pecans (Carya illinoensis) showing undoubted symptoms of rosette have been received from a much wider territory including Arizona, Tennessee, and other States. Similar symptoms have been observed by the authors upon other species of hickory, notably the mockernut (Carya alba (L.) K. Koch.), and the pignut (C. glabra (Mill.) Spach.), also upon the butternut (Juglans cinerea L.), the rock walnut of Texas (Juglans rufesbris Engelm.), the hackberry (Celtis occidentalis L.), and the common locust (Robinia pseudacacia L.)

Furthermore, pecan rosette does not appear to be limited to any particular soil type, topography, or season. We have noted many distinct and undoubted cases in the deep sand of the Florida Coastal Plain with the water table at 3 to 3½ feet from the surface, farther inland in deep sand or sandy loam with the water table varying from 2 to 10 feet, in sand or sandy loam underlain by yellow, red, or white clay at depths varying from a few inches to several feet and with a varying water table, in the clay or sandy clay of washed-out hillsides, in the river bottom and alluvial soils of Louisiana and Texas, in the black upland soils of Texas, in cultivated and uncultivated land, with and without fertilization, in extremely rich and extremely poor soils, and in wet and dry seasons. In fact, for the localities personally investigated, swamp land has presented the only location so far entirely exempt. It is true that wherever the soil tends to be water-soaked through a considerable portion of the growing season the pecan presents an unhealthy appearance through its failure to make proper growth and through the sickly yellow appearance of the leaves. Under such conditions the tree usually dies sooner or later. The symptoms, however, bear so little resemblance to those of rosette that even the most casual observer will not confuse the two diseases.

SYMPTOMS AND VIRULENCE OF PECAN ROSETTE

Pecan rosette first makes itself evident through the putting out of undersized, more or less crinkled, and yellow-mottled leaves (Pl. XXIV, figs. 1 and 2), particularly at the ends of the branches. The veins tend to stand out prominently, giving a roughened appearance to the leaf blade, and the light-green or yellowish areas which give the leaf its mottled appearance occur between the veins. In these light-colored parts the tissues are thinner and less fully developed than in the normal leaf, and later in the season they frequently become dark reddish brown and dead.
In many cases the intervascular tissue here and there fails to develop at all, so that the lamina is dotted with smooth-margined holes suggesting insect perforations which have subsequently healed over (PI. XXV). These first symptoms may occur over the whole tree at once, but often one or more branches may be affected for several months before the whole tree appears involved. At this stage the foliage as a whole often presents a rusty appearance: The diseased branches usually fail to reach their normal length, so that the leaves are clustered together on a shortened axis, giving a bunched appearance to the group which led the senior author, about 1902, to apply the term "rosette" as an appropriate name for the disease (Pl. XXVI; cf. fig. 1, rosetted shoot, with fig. 2, normal shoot). Nuts are frequently borne and carried to maturity on these branches.

In some cases the disease goes no farther. The trees may continue in this way for several seasons, or they may recover completely after showing the early symptoms for one or more years. However, in a well-defined case where the symptoms are general over the greater part of the tree, the affected branches begin to die back from the tip during the latter part of the first season or later (Pls. XXVII and XXVIII). At first brownish spots and streaks appear in the green bark, and these dead areas increase in size until the whole end of the twig or branch dies. While death appears to start in the green bark, the cambium soon becomes affected and the wood and pith are usually discolored. This dying back or "staghorn" stage is followed during the same or the following season by the development of numerous lateral shoots from dormant or adventitious buds. In young vigorous trees these first shoots of the season are usually large and succulent, and the leaves are dark green and above the normal in size. In all probability this effect is physiologically equivalent to the effect of severe pruning. Toward the middle of the season, however, the typical yellow-mottled color appears and the later-developed leaves are more or less cramped and roughened, as well as below the normal in size. Dormant axial buds of one or two series may develop into abortive shoots, and toward the end of the season clusters of short or spindling branches usually put out from adventitious or dormant buds farther back on the branches or on the main trunk. The leaves in these cases are much reduced in size and may appear as a mere skeleton with ragged edges.

This process goes on from year to year. The growth of the tree is checked, and these abnormal clusters of branches are formed only to die back each season and be followed by others. Thus a well-marked case of several years' standing presents a characteristically gnarled and forlorn appearance (Pl. XXVIII, fig. 3). Rosette in all its forms occurs in trees from seedling and budded or grafted nursery stock to trees of long-established maturity, a hundred or more feet in height, and it is one of the worst diseases known to affect pecans.
PRUNING EXPERIMENTS

If the rosette were of a parasitic nature, it seemed entirely possible that a severe pruning out of the diseased parts or at least a cutting back to the stump might entirely eliminate the disease. To test out this proposition, 10 distinctly rosetted trees in the orchard of Mr. J. B. Wight, Cairo, Ga., were severely pruned and 5 similarly diseased trees were cut off at the ground and allowed to send up sprouts from the stump. This work was done in the winter of 1902–3, and observations the following midsummer showed the new growth in all the trees to be distinctly rosetted (Pl. XXVIII, fig. 2).

In like manner three 7-year-old trees were severely cut back, or "dehorned," and five other badly diseased trees were cut back to a stub 18 inches high. This work was carried out in February, 1912, in the orchard of Mr. G. W. Saxon, Tallahassee, Fla. The following spring most of the new growth was vigorous and the leaves were dark green and normal in appearance. Toward midseason, however, the leaves began to appear yellow mottled and those most recently developed were undersized; before the end of the season every tree and nearly every shoot was badly affected with rosette.

In the summer of 1911 three badly diseased trees belonging to the Standard Pecan Co., Monticello, Fla., were cut back to the trunk. The following midsummer all the new growth was rosetted as badly as before cutting back.

Further observations have been made upon the effect of severe pruning and cutting back in orchards at Belleview, St. Augustine, Monticello, and Tallahassee, Fla.; Thomasville, Baconton, and Albany, Ga.; and at Orangeburg, S. C. In all cases the same negative results have occurred. Usually in vigorous trees the new growth appears healthy, as in the case of rosetted trees severely cut back by the disease itself; but before the end of the summer or at least by the next season the rosette again appears. The disease was in no case eliminated by pruning.

TRANSPLANTING EXPERIMENTS

In order to determine whether the cause of the disease was to be sought in the tree itself or in the soil, several transplanting and germination tests were carried out.

In December, 1902, 8 badly rosetted trees were dug up from the J. B. Wight orchard at Cairo, Ga., and healthy seedling nursery trees were immediately set in the holes. At the same time 41 nursery trees were set in vacant places where no trees of any kind had been growing for one or more years. The following August, 1 tree out of the first group and 8 out of the second were dead, probably from effects of transplanting. All the remaining trees were apparently in a normal condition.
Of the remaining 7 trees in the first group, 4 continued healthy for two seasons and were dug up. One was badly rosetted the following season, 1 showed a slight trace of the disease at two years and was dug up, and the last tree, which was normal at the time of observation in 1903, 1904, and 1912, showed a distinct case of rosette in 1913. Of the 33 remaining trees of the second group, 16 remained normal through two years, and were cut out, 5 were normal at the time of all observations, while 12 at one time or another showed distinct symptoms of rosette. It will be noted that the percentage of trees contracting the disease did not differ greatly in the two cases.

In the same orchard, during the fall and early winter of 1904–5, 35 healthy trees, comprising 11 varieties, were set in holes occupied by rosetted trees within one year. Likewise, 11 trees, comprising 7 varieties, were set after healthy trees had been removed or in places previously unoccupied. Observations in 1912 and 1913 showed 33 trees of the first group with pecan rosette and only 2 normal. Of the second group, 6 contracted rosette, while 5 were normal in appearance.

Similarly in February, 1908, 27 trees of 7 varieties were set after rosetted trees, and 4 trees of 3 varieties in vacant places or after healthy trees. Observations in 1912 and 1913 showed 24 trees of the first group to be rosetted, and 3 healthy. In the second group 2 were rosetted, while 2 were healthy.

In the winter of 1904–5, 10 rosetted nursery seedlings at Cairo, Ga., were transplanted to a part of the same field previously unoccupied by pecan or other hickory trees. Observations during October, 1905, showed 6 trees apparently normal, 3 with symptoms of rosette on the older leaves, but with the later growth normal in appearance, and 1 with traces of rosette. The following August, 9 trees appeared healthy, and 1 presented a doubtful case of rosette. In August, 1907, all 10 trees were apparently normal. In February, 1908, 2 trees which had died from unknown causes were replaced with healthy trees of the Stuart variety. The next observation, made in September, 1912, showed 6 distinct cases of rosette (including the 2 Stuart pecan trees above mentioned), 2 trees with doubtful symptoms of rosette, and 2 normal. The last note, made in August, 1913, showed 5 distinct cases of rosette, the same 2 doubtful cases, and 3 normal trees. Four of the rosetted trees were much improved in appearance over that of the preceding season.

Forty-three rosetted nursery trees at Glen St. Mary, Fla., were transplanted from the nursery row (March, 1907), where the water table was about 18 inches below the surface, to another part of the place where the soil was a loamy sand underlain by clay, with the water table at a considerable distance below the surface. Owing probably to the late spring transplanting, 18 of the trees died without putting out leaves. Of the remaining trees (October, 1907) 18 showed distinct symptoms of
rosette, 3 had doubtful traces, while 4 were normal. During the following winter part of the vacant places were filled from the nursery, making 38 trees in all. No further observations were made until the summer of 1910, at which time no traces of the disease were apparent. In August, 1912, 18 trees were normal, 12 were distinctly rosetted, while 6 showed traces of the disease. The following August, 31 trees were normal, 2 were distinctly rosetted, and 3 showed traces of the disease. Throughout the experiment the trees received no pruning, and little attention of any kind save an occasional cultivation and moderate applications of a complete commercial fertilizer.

In December, 1903, 6 nursery trees showing symptoms of rosette were taken up at DeWitt, Ga., and sent by Mr. Herbert C. White to Washington, D. C. During the latter part of November, 1904, a like number of rosetted nursery trees were sent by Mr. J. B. Wight from Cairo, Ga. Upon receipt these trees were potted in garden soil and placed in one of the greenhouses of the Department of Agriculture. All the trees lived, but observations up to January, 1907, gave no evidence of rosette in any of them. At this time 4 were set out at Takoma Park, D. C., 4 at Glen St. Mary, Fla., along with the trees described in the preceding experiment, and the remaining 4 were left in the greenhouse. The Takoma Park trees died from other causes after the second winter, but showed no more rosette. The 4 trees set at Glen St. Mary, Fla., were healthy during 1910 and 1911. In August, 1912, 1 tree showed a trace of rosette, but the following season all 4 were healthy. The greenhouse trees remained healthy until destroyed the next year to close the experiment.

In the winter of 1912, 5 Stuart nursery trees which had reached the staghorn stage of rosette were sent by Mr. H. K. Miller from Monticello, Fla., to Washington, D. C., where they were potted in garden soil and placed in one of the Department greenhouses. The trees were rather large for potting, and therefore both roots and tops were severely pruned. Probably as a result of this severe treatment, together with the almost entire absence of lateral roots, the tops of all 5 trees died, but the following season 3 sent up sprouts from the crown. These shoots have made a perfectly normal growth for three seasons and have at no time shown the faintest traces of rosette.

In 1910 a badly rosetted Stuart pecan tree in the orchard of Dr. R. B. Garnett, at St. Augustine, Fla., was taken up by the owner and reset in another part of the place. The following winter 5 badly diseased young orchard trees were taken up and reset about a quarter of a mile distant. Observation by the junior author in August, 1912, showed the Stuart tree to be entirely recovered. Three out of the second group were entirely normal in appearance, while two still had symptoms of the disease.
It will be noted from the results of these experiments in transplanting that of the healthy trees set after rosetted trees nearly all subsequently contracted the disease, while of those set after healthy trees only about half subsequently showed symptoms of the disease. This would point toward the conclusion that some relation exists between pecan rosette and the soil, either directly through the soil itself or through its previous infection by rosetted trees.

Furthermore, it has been shown that of rosetted trees set after healthy trees in the same locality or replanted in entirely different situations, a very high percentage of the trees and often all recovered. This would tend to indicate that the soil relation is the direct cause of rosette rather than infection of the soil with parasitic organisms from previously diseased trees.

Top soil and subsoil were taken separately in March, 1913, from the immediate vicinity of trees in the last stages of rosette at Belleview and at Tallahassee, Fla. This soil was shipped to Washington D.C., and in early June, 17 normal, recently germinated pecan seedlings were set in each of the four soil types. At the same time a like number of seedlings were set in the garden soil ordinarily used in the Department greenhouses. In both cases the top soil was a sandy loam. The Belleview subsoil was almost clear sand, while the Tallahassee subsoil was a pasty red clay. Observations were frequently made throughout two seasons, but no symptoms of rosette appeared in any case. Of course, a test of this kind with a small quantity of soil in a porous 8-inch pot must be rather inconclusive with reference to any effect of the chemical ingredients of the soil, but it was thought that if the rosettte were caused by any organisms living in the soil surely there would be a chance of at least some of the trees contracting the disease. Even from this point of view two seasons under observation are not sufficient, but taken in connection with the other rosette work the evidence at this stage of the experiment is perhaps worthy of record as tending to indicate the non-parasitic nature of the disease.

GERMINATION OF NUTS

In order to determine the communicability of the rosette, pecan nuts matured in the fall of 1912 on rosetted trees were planted in moist sand in one of the Department greenhouses in Washington, D.C. As they came up they were potted in garden soil and kept under observation during the spring, summer, and fall of 1913, and the summer of 1914. The nuts were obtained under the following conditions:

Of 12 nuts obtained directly from a rosetted branch, 9 germinated. Of 10 from a tree, most of which showed rosette, 9 germinated. Both lots were from the seedling orchard of Dr. W. P. Williams, Blackshear, Ga.
Of 93 nuts from a Frotscher pecan tree, showing rosette over the whole top, 52 germinated. This lot was sent by Mr. C. A. Reed, of the Bureau of Plant Industry, from the Parker orchard, Thomasville, Ga.

Of 25 nuts obtained from rosetted branches of Teche, Alley, Stuart, and Van Deman pecans from the orchard of Mr. W. P. Bullard at Albany, Ga., 18 germinated.

Of 4 nuts from a rosetted branch on an old seedling tree at Marion Farms, near Ocala, Fla., 3 germinated.

Of a second lot of nuts from Blackshear, Ga., number unknown, taken from a rosetted tree but not from a distinctly diseased branch, 28 germinated.

Out of all the nuts which germinated not a single seedling showed any symptoms of rosette, so that whatever the cause of the disease, it is apparently not transmissible through the seed.

**ISOLATION OF MICROORGANISMS AND INOCULATIONS**

To further test the communicability of the disease, several healthy nursery trees at Cairo, Ga., were inoculated with pieces of tissue from a badly diseased branch in August, 1902. The bark was removed from the latter, and bits of the wood scraped up with a sterile scalpel were placed in sterile water. Incisions were then made near the terminal buds of vigorous, healthy branches and bits of the diseased material inserted. This experiment was duplicated in August, 1906, when slices of diseased buds were inserted into the terminal branches of 11 nursery trees. The inoculated trees in both cases remained healthy.

During the fall of 1911 a series of attempts was made to isolate any organisms that might be present in various parts of diseased trees. Numerous Petri-dish cultures were made from the inner bark, cambium, wood, and pith of living rosetted twigs and from the pith and inner bark of the living roots ½ inch to 3 inches in diameter. Pieces of the tissue in each case were transferred to beef agar and corn-meal agar. All these cultures remained sterile. Material for these and the following tests was obtained by the junior author from three orchards at Tallahassee, Fla., and from specimens received from Sacaton, Ariz.

With the partly dying tissues, however, many of the cultures gave bacteria and fungi. This was to be expected, since, as is well known, large numbers of saprophytic forms soon obtain entrance to tissues which have died from almost any cause. This is particularly true of tissues which have died from physiological causes, since they are not already infested with fungous or bacterial growth.

Out of 55 pieces of pith tissue from partly dying twigs, 39 remained sterile, 12 gave colonies of fungi, including an Aspergillus, a Penicillum, and a nonfruiting whitish fungus, and 4 gave as many different types of bacteria.
Out of 22 pieces of tissue taken at the juncture of dead and living wood on badly rosetted twigs, all developed fungous colonies.

Of 40 bits of inner bark taken from dying twigs, 27 were sterile, 6 developed fungous colonies, and 7 grew bacterial colonies.

Of 32 pieces of the inner bark taken from partly dying roots, 25 remained sterile, while 7 developed colonies of bacteria.

Since no constant form appeared in the cases where organisms did develop, it was thought highly improbable that the disease could be attributed to any of them, especially since a majority of the cultures remained sterile. Nevertheless, for the sake of completeness, greenhouse inoculations were made with all the different strains isolated, including 15 types of fungus and 17 types of bacteria. In each case needle-puncture inoculations were made in the tender growing tip and in the older bark of one or more pecan seedlings, and the latter were left under bell jars for several days. Check trees were similarly punctured with a sterile needle. Daily observations were made, and at the end of a week the needle punctures in the checks, and with two exceptions in all the inoculated trees, were healing over. The tips of these two inoculated trees were beginning to wither, but since their tissues were much broken up in the process of inoculation, this was thought to be due to mechanical injury. However, for further certainty several other trees were inoculated with these two bacterial strains, care being taken to injure the succulent tissues as little as possible. These all healed over without signs of infection.

Several examinations of healthy and rosetted roots showed a whitish, fungous weft on the young roots of healthy trees which was at first not found on those with rosette. It was thought possible that the pecan might be dependent on some mycorrhizal relation for its well-being and that the absence of the fungous symbiont gave rise to the diseased condition. However, diseased and healthy trees have been dug up in Texas, Georgia, Florida, and South Carolina, and in some cases the sterile, fungous weft has been found on healthy roots, sometimes on rosetted roots, but more frequently, so far as could be detected, it was absent. Moreover, of 300 healthy trees grown from seed in the Department greenhouses at Washington, D. C., those with and without fungous weft on the roots were about equal in number. Twelve isolations were made from specimens with this fungous growth, and all, when grown on corn-meal agar, developed typical Fusarium spores.

It is by no means demonstrated that rosette has no mycorrhizal relations, but the preponderance of evidence lies strongly on the positive side of the question. The apparent absence of fungi and bacteria from still living rosetted material, as shown by cultural tests, and the negative results of inoculations with organisms obtained from partly dead rosetted material strongly support the view that the disease is not of parasitic nature.
BUDDING AND GRAFTING EXPERIMENTS

NORMAL BUDS AND CIONS ON ROSETTED STOCKS

Four badly rosetted trees in the J. B. Wight orchard at Cairo, Ga., were budded from healthy Frotscher pecan trees in April, 1903. All lived and showed rosette the following season.

Eighteen rosetted trees in the orchard of Mr. G. W. Saxon, at Tallahassee, Fla., were cleft-grafted with normal cions from an old seedling tree in February, 1912. Most of the cions began to put out leaves in the spring, but all except three were destroyed by bud worms. These three put on a vigorous and apparently healthy growth the first part of the season, but toward fall and during the following summer symptoms of rosette were distinct in all three cases.

In an orchard of the Standard Pecan Co., at Monticello, Fla., two badly rosetted trees of each variety—Schley, Stuart, and Pabst—were budded with several buds from a healthy tree. Observations the following August showed two buds on each of the Schley pecans living, four and six buds on the two Stuart trees, and one bud on one of the Pabst trees. All shoots from these buds were badly rosetted.

ROSETTED BUDS AND CIONS ON NORMAL STOCK

In the spring of 1903, 24 buds subtended by distinctly rosetted leaves were put into healthy nursery trees, and 24 similar buds into healthy orchard trees of Mr. J. B. Wight, Cairo, Ga. Observations in midsummer, 1904, showed 13 living buds in the nursery, and of these 1 had a distinct case of rosette, 1 a trace, while the others were normal. Of the buds put into the orchard trees, 20 were living and only 1 had rosette. In the latter case the tree had developed rosette over the whole top subsequent to the budding operation. This being the only one behaving in this manner it can hardly be considered probable that the rosette in this case was transmitted through the bud.

One hundred buds from rosetted branches were worked on nursery seedlings at the same place in August, 1906. A large percentage lived, and in the following midsummer no traces of rosette could be found on any of them, though the trees from which the buds were taken still showed the disease.

In August, 1907, 82 more buds were inserted on healthy seedling stocks in the same nursery. Observations the following season (October, 1908) showed the same results as in the last experiment.

Twenty to thirty rosetted buds of each of the Schley, Pabst, and Stuart varieties were worked on nursery seedlings belonging to the Standard Pecan Co., Monticello, Fla., in August, 1912. In the following August, out of the 12 living Schley pecan buds 1 showed a doubtful trace of rosette; of the 10 living Pabst buds 1 showed a distinct and 1 a doubtful trace; and of the 10 living Stuart buds 1 showed a distinct
and a doubtful trace of rosette. Counts in the two adjacent nursery rows on either side showed at least as high a percentage of rosette as those worked with diseased buds.

Buds from badly rosetted branches were taken from Tallahassee, Fla., and were worked on 8 healthy orchard trees at Glen St. Mary, Fla., in August, 1912. At the same time healthy buds were inserted in branches of 2 trees with distinct symptoms of rosette. Out of 30 to 35 diseased buds inserted, 14 developed, and the following August no rosette could be found on any of them. Of the 12 healthy buds on rosetted stock, 4 had lived. In the case of 1 bud the shoot was perfectly normal, but the tree as a whole had meanwhile recovered from rosette. The 3 others showed only traces of rosette, but the tree on which they were worked also had nearly recovered from the disease.

Grafting was also attempted in this connection at Washington, D. C. Sixty 1-year-old seedlings were grafted by the veneer method with rosetted cions from Cairo, Ga., but none of the cions developed.

In February, 1912, 105 badly rosetted cions from two orchards in Tallahassee were whip-grafted into a part of the general nursery of Mr. H. K. Miller, at Monticello, Fla. Nursery trees on all sides were grafted to healthy cions. The following August 45 cions were living. Of these, 7 showed traces and 2 had developed distinct symptoms of rosette. Counts in the adjacent general nursery showed about the same percentage of rosette.

It will be noted that normal buds and cions on rosetted stocks invariably gave rosetted shoots. Rosetted buds and cions on apparently healthy stocks, with but few exceptions, gave healthy shoots, and wherever exceptions occurred the percentage of rosetted shoots was no greater than in adjacent stocks worked with normal buds. The results here tend to show that pecan rosette is not caused by a perennial mycelium, or by bacteria, or by any infecting virus within the tissues of the host.

FERTILIZER EXPERIMENTS

A fertilizer test was started in March, 1902, in a badly rosetted orchard belonging to Mr. J. B. Wight, Cairo, Ga. Alternate rows were used for the five plots, and the intervening rows in each case were left untreated. Plot 1 received nitrate of soda; plot 2, lime; plot 3, cottonseed meal, acid phosphate, and kainit; plot 4, a liberal application of stable manure; plot 5, ground bone meal. Observations in the summer of 1904 in plot 1 showed 5 trees with the same amount of rosette as at the beginning of the experiment; 1 tree, better; and 5, worse. In plot 2 there was no change in 3 trees, but 10 others were worse. In plot 3, 7 trees were in the same condition as at the beginning, 1 was better, and 3 were worse. In plot 4, 9 trees were the same, 1 was better, and 3 were worse. In plot 5, 6 trees were the same, 2 were better, and 4 worse. Two check rows in the same orchard showed, respectively, 6 trees in the same condition, 1
better, and 6 worse; and 3 the same, 2 better, and 7 worse, showing that during the years in question a slight increase in the disease had taken place, and any effect of the fertilizers applied was scarcely discernible. The plot treated with lime stands out somewhat from the rest, since there was distinctly more increase in the disease here and no tree showed signs of recovery.

In April, 1911, a 16-plot fertilizer test was started among nonrosetted 8-year-old Georgia Giant and Nelson pecans in an orchard at Baconton, Ga., at that time the property of Mr. Chas. M. Barnwell. The soil is a sandy loam underlain at 1½ to 2 feet by red clay. Two rows of each variety were taken through the block, giving 4 trees of each variety to a plot. The same general scheme of fertilizer combinations was used as that employed by Mr. M. B. Waite in his apple-nutrition experiments, and annual applications were made, except in the case of lime, which was used only at the beginning of the experiment. The land was cropped to winter oats, followed by cowpeas, the first two seasons, and the third season velvet beans only were grown in the centers. At the last observation there was but little apparent difference among the fertilized trees in general vigor and length of growth, color of foliage, and quantity of nuts, but the two check plots bore foliage of a conspicuously paler color. The condition of the various plots in August, 1913, is shown in Table I.

Table I.—Summary of results from a fertilizer test with pecan trees at Baconton, Ga.

<table>
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<th>Plot No.</th>
<th>Fertilizer.a</th>
<th>Number of trees with rosette</th>
<th>Number of doubtful cases</th>
<th>Number of healthy trees</th>
<th>Average increase in diameter</th>
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<tr>
<td>12</td>
<td>Muriate of potash, nitrate of soda, and acid phosphate; no lime</td>
<td>2</td>
<td>4</td>
<td>1.48</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>Muriate of potash and acid phosphate; no lime</td>
<td>8</td>
<td>8</td>
<td>1.08</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>Stable manure; no lime</td>
<td>8</td>
<td>8</td>
<td>1.94</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>Stable manure and ground bone; no lime</td>
<td>8</td>
<td>8</td>
<td>1.05</td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>Control; no lime</td>
<td>7</td>
<td>7</td>
<td>1.03</td>
<td></td>
</tr>
</tbody>
</table>

a Fertilizers were applied at the following rates: Lime (CaO, acted on jointly by air and water), 1 bushel per tree; nitrate of soda, 8 pounds; cottonseed meal, 32 pounds; muriate and sulphate of potash, 8 pounds; acid phosphate, Thomas phosphate, and ground bone, 24 pounds; stable manure, a liberal application.
It will be noted from Table I that rosette occurred on all but two of the plots receiving an application of lime. In the five unlimed plots there were only two doubtful cases of rosette and these two trees directly bordered a limed plot. Care was taken in spreading the fertilizers on the side bordering a contiguous plot not to distribute to the middle, but the lime was spread to the dividing line, and, hence, there was a chance of its affecting the adjoining unlimed plot. The largest number of cases and those showing the most advanced stages occurred on the two limed plots, Nos. 9 and 10, treated, respectively, with acid phosphate and muriate of potash and with acid phosphate and nitrate of soda. Thus, while the results of this test have so far been partly negative, they have at least tended to show that some relation exists between pecan rosette and the constituents of the soil.

A fertilizer test was started in April, 1912, in the badly rosetted part of a 7-year-old orchard belonging to Mr. G. W. Saxon, at Tallahassee, Fla. The soil here is a sandy loam underlain by a stiff red clay. Thinking that possibly the lack of proper drainage might be a factor predisposing to rosette, the subsoil around six badly diseased trees was dynamited. Three 6-foot holes were bored at 10 to 12 feet from the trees and $\frac{1}{2}$ to $\frac{3}{4}$ pound of dynamite was used to each hole. Applications of sulphur flour and of copper sulphate to the soil were also tried, each at the rate of 6 pounds to the tree. With the exception of lime, which was applied at the rate of one-third of a bushel to the tree, the fertilizers were used at the rate noted for the preceding experiment. The results of this experiment are given in Table II.

**Table II.—Summary of results from a fertilizer test with pecan trees at Tallahassee, Fla.**

<table>
<thead>
<tr>
<th>Plot No.</th>
<th>Fertilizer</th>
<th>August, 1912</th>
<th>August, 1913</th>
<th>Number of normal trees</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Lime, muriate of potash, nitrate of soda, and acid phosphate</td>
<td>9</td>
<td>9</td>
<td>3</td>
</tr>
<tr>
<td>2</td>
<td>Muriate of potash</td>
<td>3</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>3</td>
<td>Acid phosphate</td>
<td>4</td>
<td>4</td>
<td>6</td>
</tr>
<tr>
<td>4</td>
<td>Control, subsoil dynamited</td>
<td>6</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>5</td>
<td>Control, untreated</td>
<td>7</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>6</td>
<td>Nitrate of soda</td>
<td>5</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>7</td>
<td>Muriate of potash</td>
<td>3</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>8</td>
<td>Acid phosphate and nitrate of soda</td>
<td>3</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>9</td>
<td>Muriate of potash, nitrate of soda, acid phosphate</td>
<td>6</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>10</td>
<td>Stable manure</td>
<td>1</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>11</td>
<td>Thomas phosphate</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>12</td>
<td>Lime</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>13</td>
<td>Cottonseed meal</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>14</td>
<td>Copper sulphate</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>15</td>
<td>Sulphur flour</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>

a As these trees were badly affected with rosette at the beginning of the experiments, it is probable that their death was at least in large measure due to this disease.

b Dead.
As will be observed from the data given, the results were largely negative. There were no signs of recovery, and in the case of stable manure (plot 10), lime (plot 12), and cottonseed meal (plot 13), there was an increase in the number of rosetted trees. It should be noted that most of the trees having rosette at the start were well-advanced cases.

At the same time, the subsoil was dynamited around three 15-year-old rosetted trees in the orchard of Mr. G. G. Gibbs. The results in this case were likewise negative as shown by observations after one and two years. The results of dynamiting the subsoil in these two orchards, together with observations showing the absence of pecan rosette in swampy land, seem to indicate that the disease is not due directly to lack of proper subsoil drainage.

The soil around two rosetted trees in the J. B. Wight orchard, Cairo, Ga., in the spring of 1907, was treated with copper sulphate and magnesium sulphate at the rate of 1 pound of each for every inch in diameter of the trunk. The trees were decidedly injured by this treatment, and the diseased condition continued as before. A negative result followed the use of 8 pounds of copper sulphate around a single 9-year-old rosetted tree at Baconton, Ga.

The soil around each of 22 rosetted trees in the 4-year-old Davenport orchard at Belleview, Fla., was treated in June, 1912, with 2 to 4 pounds of copper sulphate, according to the size of the tree. By the following midsummer 7 trees had recovered, 7 were somewhat improved in appearance, and 8 were either in the same or in a worse condition than at the beginning. In the same 40-acre block, 117 out of 389 rosetted trees had recovered in the same period. In other words 31 per cent of the trees treated with copper sulphate recovered and also 30 per cent of the untreated trees in the same block. A considerable number of the untreated trees were also improved in appearance. Both groups of trees were fertilized by the owner with a complete commercial fertilizer at the rate of 8 pounds to the tree.

The copper-sulphate treatment has from time to time been recommended by a number of orchardists, and in a few cases observed by the junior author some apparently beneficial results have occurred. But the usual failure of the grower to run proper checks with an experiment, together with the fluctuation of the disease without any treatment, lends a rather doubtful character to the results. At any rate, this treatment is not to be recommended, except in an experimental way, until further tested out.

SPRAYING EXPERIMENT

Five rosetted trees in the J. B. Wight orchard at Cairo, Ga., were given three applications of Bordeaux mixture—in March, April, and May, 1903. No positive results from this experiment were discernible.
ORCHARD RECORDS

Rosette records for periods varying from 2 to 12 years have been kept in several orchards in Georgia, Florida, and South Carolina.

Of 159 trees observed in the J. B. Wight orchard for three successive seasons (1902 to 1904), 24 were healthy for all three seasons, 17 were healthy at the beginning, but later contracted the disease, 13 had rosette, but recovered, 108 were rosetted throughout the period, and 7 fluctuated each season.

Eighty trees in the same orchard were observed in 1902, 1903, 1904, 1912, and 1913. So far as these observations go, 30 trees remained normal throughout the period, 7 were normal, but contracted the rosette, 12 were rosetted, but recovered, 8 were rosetted throughout, and 21 fluctuated back and forth between the normal and rosetted condition.

Observations on 274 trees in the same orchard during 1912 showed 136 trees with the rosette, and during the following season 33 trees more had contracted the disease. The cultivation and fertilization had not been varied.

In a 40-acre block of the Davenport orchard at Belleview, Fla., containing 1,069 trees, 389 had the rosette in 1912 and 256 in 1913. All had received the same treatment, except for the few trees used in the copper-sulphate test, and as previously noted this treatment gave negative results. Of the three varieties present, 19 per cent of the Van Deman, 25 per cent of the Stuart, and 28 per cent of the Teche pecans had rosette in 1912.

Sixty-two soil borings were made to 6 and 9 feet from the surface and in the vicinity of both healthy and rosetted trees. Of 31 trees in a clay to sandy-clay subsoil, 13 had the rosette and 18 were normal. Of 23 trees in a sandy subsoil, 9 were rosetted and 14 were normal. Of 8 trees in a subsoil containing considerable quantities of a soft lime rock, all were healthy. In the first two groups the difference in the number of diseased and healthy trees was not conspicuous. On account of the entire absence of rosette in the third group, partial analyses of the subsoil around three trees were made. In sample No. 1 the percentages of lime, magnesium, and phosphorus, computed as the oxids, were 9.68, 0.82, and 8.32, respectively; in sample No. 2, 5.42, 1.09, and 6.03; and in sample No. 3, 0.58, 0.99, and 3.44. More or less clay was present in all the samples, and in No. 2 there was a considerable admixture of creolin.

In a small orchard of 96 trees belonging to Mr. G. G. Gibbs, at Tallahassee, Fla., 38 trees were rosetted in 1912, and during the following season 4 of these recovered. In another orchard of 116 trees on the same farm, with similar topography and apparently similar soil, 6 were rosetted in 1912 and 12 the following season.

In the G. W. Saxon orchard of 231 trees at Tallahassee, Fla., 125 trees had rosette in 1912 and 173 in 1913. Most of the orchard had received
no fertilizer for several seasons, and where fertilizer had been applied no distinct difference in the rosette could be detected.

In the W. P. Bullard orchard near Albany, Ga., 291 out of 646 trees were rosetted in 1912. By the following season 101 more trees had contracted the disease. Fertilization and cultivation were uniform for the two seasons.

In a block of 233 trees belonging to Mr. H. K. Miller, at Monticello, Fla., 81 trees had the rosette during 1912 and 1913. Fertilization was uniform for both years, and the soil was a sandy loam underlain by a rather stiff, red, sandy clay.

Observations were taken during 1912 and 1913 in three blocks belonging to the Standard Pecan Co., at Monticello, Fla. Out of 406 trees observed in the first block 231 had rosette in 1912 and 254 in 1913. In the second block of 450 trees 119 were rosetted at the first observation and 121 at the second. In the third block of 570 trees, 42 were rosetted in 1912, and 34 in 1913. A part of all three blocks were in swamp land that had not been fully drained, and the absence of rosette here was conspicuous. The soil in the remainder of the three blocks was a sandy loam underlain by a red clay or sandy clay. For three seasons preceding 1912 a complete commercial fertilizer was used. In 1912 a small application of stable manure was made around each tree.

In the small orchard of 100 trees belonging to Dr. R. B. Garnett, at St. Augustine, Fla., 3 were rosetted, both in 1912 and 1913, 7 were rosetted but recovered during the second year, and 6 new cases developed during the second year. The soil is a sandy loam underlain by a clear-sand subsoil, with the water table 3 to 3½ feet below the surface. Fertilization was uniform over the orchard, except that the soil around the 10 trees showing rosette in 1912 was treated with lime and copper sulphate. Seven of these trees recovered, but no checks were run to verify the result.

In a block of 371 trees belonging to Mr. M. O. Dantzler, at Orangeburg, S. C., 146 trees had rosette in 1912 and 137 in 1913. Fertilization and cultivation were uniform during the two seasons.

It is evident from these records that rosette fluctuates from year to year without any variation in the treatment given by the grower and that diseased trees may apparently make a complete recovery and remain healthy for an indefinite period, or after a season or two they may again contract the disease. It should be stated here that in the majority of cases the trees recovering from rosette had not reached the staghorn stage. However, a considerable number of trees with terminals dying back from the disease have been seen to recover and remain normal through the one or more seasons they have been subsequently under observation by the authors. From the variations in rosette recorded from year to year under uniform cultivation and fertilization it seems
highly probable that seasonal climatic changes, such as variation in the water content of the soil, may have at least an indirect relation to the prevalence of the disease.

**ASH ANALYSES**

Complete ash analyses were made of rosetted and healthy leaves and of rosetted and healthy twigs from Cairo and Dewitt, Ga., and from Belleview, Fla. In each case the diseased and healthy material was from trees of the same age and variety and had received similar cultivation and fertilization in the same orchard. The pure-ash analyses are given in Tables III to V.

**TABLE III.**—Ash analyses of leaves and twigs of the Stuart pecan from Cairo, Ga., September, 1912

<table>
<thead>
<tr>
<th>Constituent</th>
<th>Leaves</th>
<th>Twigs</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rosetted</td>
<td>Normal</td>
</tr>
<tr>
<td>Total percentage of pure ash</td>
<td>5.59</td>
<td>3.69</td>
</tr>
<tr>
<td>SO(_3)</td>
<td>15.47</td>
<td>18.35</td>
</tr>
<tr>
<td>Cl</td>
<td>.33</td>
<td>.33</td>
</tr>
<tr>
<td>K(_2)O</td>
<td>23.36</td>
<td>23.01</td>
</tr>
<tr>
<td>Na(_2)O</td>
<td>31.48</td>
<td>34.09</td>
</tr>
<tr>
<td>CaO</td>
<td>21.50</td>
<td>14.09</td>
</tr>
<tr>
<td>MgO</td>
<td>3.25</td>
<td>8.33</td>
</tr>
<tr>
<td>SiO(_2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>100.04</td>
<td>100.09</td>
</tr>
</tbody>
</table>

**TABLE IV.**—Ash analyses of leaves and twigs of the Van Demon pecan from Belleview, Fla., September, 1913

<table>
<thead>
<tr>
<th>Constituent</th>
<th>Leaves</th>
<th>Stems</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rosetted</td>
<td>Normal</td>
</tr>
<tr>
<td>Total percentage of pure ash</td>
<td>4.68</td>
<td>3.68</td>
</tr>
<tr>
<td>SO(_3)</td>
<td>7.57</td>
<td>8.24</td>
</tr>
<tr>
<td>P(_2)O(_5)</td>
<td>.38</td>
<td>.33</td>
</tr>
<tr>
<td>Cl</td>
<td>18.46</td>
<td>16.03</td>
</tr>
<tr>
<td>K(_2)O</td>
<td>37.15</td>
<td>32.45</td>
</tr>
<tr>
<td>Na(_2)O</td>
<td>20.05</td>
<td>20.30</td>
</tr>
<tr>
<td>CaO</td>
<td>1.90</td>
<td>.65</td>
</tr>
<tr>
<td>MgO</td>
<td>.40</td>
<td>.23</td>
</tr>
<tr>
<td>SiO(_2)</td>
<td>4.83</td>
<td>14.30</td>
</tr>
<tr>
<td>Total</td>
<td>99.94</td>
<td>98.36</td>
</tr>
</tbody>
</table>

\(^1\) The ash analyses here given were made by the Bureau of Chemistry, Department of Agriculture.
TABLE V.—Ash analyses of leaves and twigs of the Schley pecan from Putney, Ga., September, 1913

<table>
<thead>
<tr>
<th>Constituent</th>
<th>Leaves</th>
<th>Stems</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Per cent.</td>
<td>Per cent.</td>
</tr>
<tr>
<td>Total percentage of pure ash</td>
<td>4.72</td>
<td>4.78</td>
</tr>
<tr>
<td>SO₃</td>
<td>6.76</td>
<td>5.63</td>
</tr>
<tr>
<td>P₂O₅</td>
<td>6.23</td>
<td>6.52</td>
</tr>
<tr>
<td>Cl</td>
<td>2.09</td>
<td>2.66</td>
</tr>
<tr>
<td>K₂O</td>
<td>23.03</td>
<td>14.12</td>
</tr>
<tr>
<td>Na₂O</td>
<td>4.49</td>
<td>3.34</td>
</tr>
<tr>
<td>CaO</td>
<td>35.12</td>
<td>42.04</td>
</tr>
<tr>
<td>MgO</td>
<td>10.74</td>
<td>16.42</td>
</tr>
<tr>
<td>Fe₂O₃</td>
<td>3.03</td>
<td>4.84</td>
</tr>
<tr>
<td>Al₂O₃</td>
<td>1.19</td>
<td>2.44</td>
</tr>
<tr>
<td>Mn₃O₄</td>
<td>6.45</td>
<td>7.23</td>
</tr>
<tr>
<td>SiO₂</td>
<td>104.45</td>
<td>104.82</td>
</tr>
</tbody>
</table>

Most of the differences between the pure ash of healthy and of rosetted material were not constant in the three sets analyzed. In both leaves and twigs from Cairo, Ga., the magnesium content was much higher in the diseased material, but in the two other sets the percentage was nearly the same in both diseased and healthy material.

The percentage of phosphorus was greater in the normal leaves of two sets and in the normal twigs of one set. In the other cases the percentage was about the same in both healthy and diseased material.

The calcium content was greater in the normal leaves and twigs of two sets and considerably less in the remaining set of material.

The percentage of potassium was greater in all the rosetted material, with the exception of one set of rosetted and normal twigs, where there was slightly more potassium in the normal.

Other differences shown by the analyses are either slight or greatly variable.

DISCUSSION OF RESULTS

PARASITISM VERSUS NONPARASITISM

The following experimental data have a bearing on the question of possible parasitism or upon nonparasitism.

Out of 144 nuts collected in different localities from badly rosetted branches, 91 germinated and none of the seedlings gave symptoms of the disease during the two seasons under observation. These nuts were placed in the greenhouses of the Department of Agriculture at Washington, D. C., which is far removed, both as to locality and environment, from the orchards where the nuts were obtained, but the conclusion from
this experiment nevertheless seems justifiable that whatever the cause of the disease it is not transmissible through the seed (see p. 155).

Inoculation of tender growing tips with bits of diseased tissue gave negative results. Attempts at isolation of micro-organisms from living rosetted material gave negative results in all cases. Various fungi and bacteria were isolated from partially dead material obtained in four different orchards, but no constant form appeared and inoculations gave negative results (see p. 156). These results strongly support the view that pecan rosette is not of a parasitic nature.

Mycorrhiza have been found promiscuously on both normal and rosetted trees, or perhaps more often the roots of both normal and rosetted trees have appeared to be free from any such fungous growth. It is probable that the disease is not due to the presence or absence of mycorrhiza (see p. 157).

Where normal buds or cions were worked upon rosetted stocks they have invariably developed the disease during the same or the following season, except in a few cases where the trees used as stock had in the meantime themselves recovered. With few exceptions rosetted buds and cions worked upon apparently normal nursery and orchard trees developed into normal shoots. Where rosetted shoots developed their percentage was no greater than in contiguous nursery rows or orchard trees worked to normal buds or cions (see p. 158). The results here tend to show that rosette is not caused by a perennial fungus mycelium, by bacteria, or by any infecting virus within the tissues of the pecan.

Healthy nursery trees set in large pots of top soil and of subsoil taken around badly rosetted trees remained normal during the two seasons under observation (see p. 155). This series of tests was carried out in the Department greenhouses at Washington, D. C., small quantities of the soil being used under entirely different environment than present in the location from which obtained. The results are therefore not taken as conclusive, but merely as tending to indicate the nonparasitic nature of the disease.

Transplanting of healthy nursery trees to holes from which rosetted trees had been removed gave a very high percentage of rosette in the replants, while transplanting badly rosetted trees to situations where less rosette or none at all had been observed gave a very high percentage of recovery (see p. 152). By comparison of the results of these two tests it appears that a soil relation is the important factor in causing rosette rather than any transmission of the disease from one tree to another.

Thus, from the nontransmission by seed, the negative results of isolation and inoculation tests, the varying presence and nonpresence of mycorrhiza, the budding and grafting tests, and the transplanting work, the nonparasitism of pecan rosette is considered a reasonable assumption.
RELATION TO THE SOIL

The relation of pecan rosette to the soil is partially elucidated by the following experimental data.

Severe pruning of rosetted trees or cutting back to a stump gave negative results (see p. 152). The new growth was often healthy in appearance, but toward autumn or during the following season symptoms of rosette invariably reappeared (Pl. XXVIII, fig. 2). The fact that in no case was the disease eliminated by this treatment is at least not unfavorable to the view that it is contracted directly or indirectly through the soil.

Transplanting of healthy nursery trees to holes from which rosetted trees had been removed gave a very high percentage of rosette in the replants (see p. 152). These results point toward the conclusion that some relation exists between rosette and the soil, either directly through the soil itself or through previous infection of the soil by rosetted trees.

The transplanting of badly rosetted trees to situations where less rosette or none at all has been observed gave a very high percentage of recovery (see p. 153). All rosetted trees replanted at Washington, D. C., recovered, though many of them were dying back with the disease when first taken up (see p. 154). In the latter cases the entire change of both soil and climate is held accountable for the uniform recovery of all rosetted trees. All these transplanting tests with rosetted trees tend to indicate the soil relation as the direct cause rather than infection of the soil with a parasitic organism or virus from previously diseased trees.

The results of the fertilizer experiments were partially negative (see p. 159). However, in a 16-plot test upon normal trees at Baconton, Ga., 9 out of 11 limed plots developed cases of rosette, while 1 out of 5 unlimed plots showed doubtful traces on two trees bordering a limed plot. Careful observation at the beginning of the experiment did not reveal a single case of rosette, and at the last observation no rosette was found in contiguous parts of the orchard. The plots receiving lime, acid phosphate, and nitrate of soda and those receiving lime, acid phosphate, and muriate of potash developed by far the most rosette. In fertilizer experiments at two other points considerably more rosette developed on limed than on unlimed plots. These results, while not in all cases very definite, have at least tended to show that some relation exists between rosette and the constituents of the soil and that the lime content alone or in combination with other substances may have a varying causative effect.

Dynamiting the subsoil in two different orchards gave negative results (see pp. 161–162). This fact, together with observations showing the absence of rosette in swampy land (see p. 150), seems to indicate that the disease is not directly due to lack of proper subsoil drainage.

Orchard records of individual trees over periods of 2 to 12 years show considerable fluctuations in the disease, irrespective of treatment and without any special treatment (see p. 163). Trees with a mild or moderate attack frequently recovered, and even when the staghorn stage
had been reached, trees occasionally regained their normal appearance without any artificial treatment. From the seasonal variations in rosette recorded from year to year under conditions of uniform cultivation and fertilization it seems highly probable that seasonal climatic changes, such as variation in precipitation, may have at least an indirect relation to the prevalence of the disease. In large orchards the more or less simultaneous appearance of rosette in patches, together with its usual limitation to these areas, clearly suggests some definite connection with the soil conditions.

In general, the ash analyses of rosetted and healthy material showed slight to highly variable differences, so that very little positive light was thrown upon the problem by this part of the work (see p. 165). In both leaves and twigs from one orchard the magnesium content was much higher in the rosetted than in the normal material, but in two other sets of analyses the percentage was nearly the same in both diseased and healthy material. The percentage of phosphorus was greater in the normal leaves of two sets and in the normal twigs of one set. In the other cases the percentage was about the same in both healthy and diseased material. The calcium content was greater in the normal leaves and twigs of two sets and considerably less in the remaining set. The percentage of potassium was greater in all the rosetted material, with the exception of one set of rosetted and normal twigs, where there was slightly more potassium in the normal. Other differences shown by the analyses are either slight or greatly variable.

It thus appears from the results of experiments in pruning and cutting back, transplanting tests, fertilizer experiments, results of subsoil dynamiting, and orchard records that the disease is directly or indirectly caused by some soil relation. On account of their variable character, the ash analyses shed little light on the problem.

**COMPARISONS WITH OTHER DISEASES**

Leaf-hopper injury suggested itself at one time as a possible cause of rosette of pecans. The rather far-reaching effects of the work of this insect were known, and the demonstration of their causal relation to curly-top of beets lent further plausibility to this theory. However, extensive observations have failed to disclose any connection between this insect injury and rosette. Leaf-hopper injury has occasionally been seen on pecans, but its symptoms are distinct and it has occurred both in the presence and absence of rosette. The leaves are often yellowed around the margin, somewhat curled, and if attacked while young, their growth is considerably interfered with. But there are not the distinct yellow mottling over the whole blade between the veins, the raised appearance of the latter, and the tendency to reduction of the leaf blade, followed by the dying back of the shoot from the tip.

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Neither is rosette to be confused with sun scald, or "winterkill," which affects young trees especially and manifests itself in the death of the cambium at the base of the trunk. The roots are usually uninjured and healthy sprouts soon put out from the base. Again, frost injury may kill back the tips in a manner somewhat analogous to the staghorn stage of rosette, but here the foliage symptoms are lacking, and in rosette the death of the branches is usually most prominent in late summer, a season far removed from frost.

Both the yellows and rosette of peach in a general way suggest pecan rosette. However, though some symptoms may be common to all three diseases, the complete clinical picture is distinct in each case. Some of the most prominent symptoms of peach yellows consist in the production of abnormally long, spindling branches in dense groups due to the putting out of normally dormant and possibly adventitious buds. Yellowing of the leaves always occurs at some time during the course of the disease; but in spite of the name "yellows," the leaves are often abnormally dark green in a case of recent attack. The pushing into growth of normally dormant buds and also possibly of adventitious buds is likewise characteristic of pecan rosette, but the axes are shortened rather than elongated as in peach yellows. In this shortening of the axes of growth the resemblance to peach rosette is seen. In the last-named disease the twigs are so much shortened that the leaves as they develop become clustered into a compact rosette. Peach trees affected with rosette usually die during the first and never survive the second season, and those suffering from yellows rarely survive more than five seasons, but rosetted pecan trees have been known to live for 15 years. Moreover, no case is on record of a recovery from peach yellows or peach rosette, while recovery from a moderate attack of pecan rosette is frequent, and not rare, even for the later stages. Furthermore, from the experiments outlined in this paper it appears that pecan rosette is not transmissible either through the seed or by budding or grafting, while the infectious nature of peach yellows and rosette is well established through experimental bud transmission to healthy trees.

A striking resemblance is to be observed between pecan rosette and ordinary chlorosis of various trees. In moderate cases of the latter disease yellowing of the leaves occurs without notable change in form or size, and the conspicuous dying back of the branches from the tip is lacking, but all gradations occur between such cases and those where the symptoms closely simulate rosette of pecans.

The spike disease of pineapples bears some general resemblance to rosette of pecans both as to effect and apparent cause. The leaves of affected pineapples become contracted at the base so as to form a spike-like blade, and the color of the leaf also becomes modified. It is claimed that cottonseed meal, sulphate of ammonia, kainit, muriate of potash,

and acid phosphate aggravate the disease, while nitrate of soda and sulphate of potash seem to have a beneficial effect upon diseased plants. The fertilizer tests with pecans point toward the conclusion that rosette is also a nutrition trouble.

PROBABLE NATURE OF PECAN ROSETTE

Rosette of pecans evidently belongs among the chlorotic diseases of plants grouped by Sorauer \(^1\) into two main classes: (1) Noninheritable and noninfectious diseases due mostly to improper nutritive supply or to injurious physical conditions, and (2) inheritable and infectious diseases due probably to enzymatic disturbances. From the results of the experiments and observations outlined in this paper it seems legitimate to conclude that pecan rosette should be placed in the first class of chlorotic diseases—viz, those nontransmissible diseases caused by improper nutritive supply or injurious physical conditions.

From the definite sequence of a considerable number of symptoms in pecan rosette it would seem more probable that the disease is directly caused by one set of conditions which, however, may be indirectly influenced by other conditions such as amount of rainfall, etc., rather than that this same set of symptoms may in different localities be caused by entirely different sets of conditions. Such a statement can not be laid down as a demonstrated fact, but the general knowledge of both plant and animal pathology renders it extremely probable.

The spike disease of pineapple, to which reference has previously been made, has been rather clearly demonstrated to be caused by improper nutritive supply, and it seems rather probable that pecan rosette is of a similar nature. From its wide distribution pecan rosette is clearly not confined to any one general soil type, but it is entirely possible that in those soils subject to the disease the proper balance between two or more soil ingredients may not be maintained. For example, the effects of a lack of proper balance between lime and magnesium are fairly well known, and it is possible that some such condition as this may be responsible for rosette. Indeed, the results of our preliminary fertilizer experiments point in this direction. The lime used was a high-grade stone lime purchased in barrels, but its content of magnesium was not known. The percentage of rosette was distinctly higher in the plots treated with lime, but in the absence of an exact analysis of the lime used it can not be determined from these tests whether the injury came from the lime alone or whether magnesium played a part in causing the disease. However, the chemical analysis of subsoil from the Davenport orchard, at Belleview, Fla. (see p. 163), would seem to show that lime of itself is not injurious. In this orchard the only type of subsoil entirely free from pecan rosette gave by analysis 0.58 to 9.68 per cent of lime, 3.44 to 8.32 per cent of phosphorus, and 0.82 to 1.09 per cent of magnesium, all com-

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puted as oxids. Lime was present as both carbonate and phosphate. It will be noted that the magnesium content was low. Considerable clay was present in all these cases and frequently with a considerable admixture of creolin.

The possibility of some relation to soil organisms is not entirely precluded, but it is thought that the direct cause will ultimately be found in some lack of balance in the nutritive supply, or possibly in some toxic organic substance or substances in the soil. The large group of physiological diseases to which pecan rosette seems to belong constitutes one of the most baffling series of problems now before the pathologist; and although a large number of workers have been investigating these diseases, none of them has as yet been fully worked out, either as to cause or control.

CONTROL

No great or constant difference in varietal resistance has been observed among the common orchard varieties. In one orchard a certain variety may have a much higher percentage of rosette than some other variety, but in another place the relative amount on the same two varieties is just as likely to be reversed. This has been shown clearly by orchard records in widely separated localities. Evidently the difference in apparent resistance in such cases is due either to a difference in soil conditions in the two parts of the orchard or to a difference in the resistance of the stocks to the inciting cause. That there is sometimes a difference in the true resistance of the stocks seems evident from the fact that of two trees of the same variety growing side by side (1 foot to several rods apart) one may have rosette and the other appear perfectly normal. If the cause of the disease lies in the soil, as appears to be the case, such an influence of the stock would naturally be expected. There appears to be little doubt then as to the existence of a difference in the resisting power toward rosette, but orchard records and observations tend to show that this difference is usually manifested through the stock rather than through the variety worked upon it.

It should be added that until more is definitely known as to the direct cause of pecan rosette little can be said of its control by use of resistant stocks or by other methods. Good care and fertilization are to be recommended, but until more is known of the lime-magnesium balance in relation to rosette, orchardists should test the effects of lime upon a few trees before using it on a commercial scale. The use of copper sulphate upon the soil, though favored by many growers, can hardly be recommended as a remedy for rosette without more conclusive data as to its efficacy than have yet been forthcoming (see p. 162). The results of our experiments have shown that pruning as a remedial measure is of no avail. Since there is no evidence that pecan rosette is transmissible from tree to tree, the cutting out of orchard trees showing only traces of the disease is hardly to be recommended, because they
very often completely recover. However, with well-advanced cases, especially where dying back of the branches is prominent, there is so little chance for recovery that it would seem better to replant with sound, healthy, new trees, notwithstanding the fact that the unfavorable soil conditions may persist and cause a second failure.

As to the advisability of using rosetted nursery stock, no absolute statements can be made with the present state of knowledge concerning the cause of the disease and varying resistance of the stock to that cause. However, orchard and nursery records show rather clearly that a difference in resistance of stock does exist. This being granted, it is reasonable to suppose that among nonrosetted and rosetted stocks in the same nursery the latter, if ultimately set out in soils tending to cause rosette, will be far more likely to give rosetted orchard trees than the former. This theory is borne out by the fact that in one large orchard where records of the condition of the nursery stock used in planting were kept, observations after several years showed a much higher percentage of rosetted trees from the rosetted stock than from the nonrosetted stock. It is true that the rosetted stocks were set together in one part of the orchard and that some difference in soil constituents not revealed by soil borings may have caused the difference in prevalence of the disease, but this seems hardly probable.

Hitherto, pecan nurserymen have paid little attention to the presence of rosette in the nursery stock used in budding and grafting except in extreme cases of the disease, and it is thought by the authors entirely possible that part of the wide prevalence of the disease may be due to the dissemination of susceptible stock from the nurseries. The disease is a serious one on account of the fact that crop production and recovery from well-advanced stages are seldom seen, and for the good of the pecan industry in general the number of cases of rosette should be kept at a minimum. In view of these facts, the discarding of all rosetted nursery stock is to be strongly recommended.

**SUMMARY**

Pecan rosette has been rather generally recognized by growers as a serious disease almost from the inception of pecan orcharding. It does not appear to be limited to any particular soil type, topography, or season. The disease first makes itself evident through the putting out of undersized, more or less crinkled, and yellow-mottled leaves. The veins tend to stand out prominently, giving a roughened appearance to the leaf blade, and the lighter areas between the veins are usually not fully developed. The axes of growth are usually shortened, so that the leaves are clustered together into a sort of rosette. In well-marked cases the branches usually die back from the tip, and other shoots are developed from normal or adventitious buds, only in their turn to pass through the same series of symptoms.

The nonparasitism of the disease seems rather definitely established experimentally from the nontransmission by seed, the negative results
of isolation cultures and inoculation tests, the varying presence and non-
presence of mycorrhiza on both healthy and rosetted trees, the budding
and grafting tests, and the transplanting experiments.

It appears from the results of experiments in pruning and cutting
back, transplanting tests, fertilizer experiments, results of subsoil
dynamiting, and orchard records that the disease is directly or indirectly
caused by some soil relation. On account of their variable character
the ash analyses have shed but little light on the problem.

Leaf-hopper injury has been observed on pecans, but is distinct from
rosette and has occurred both in the presence and the absence of the latter
disease. Sun scald, or "winterkill," manifests itself in the death of
the cambium at the base of the trunk and is not likely to be confused
with rosette. Frost injury may simulate rosette in the killing back of
the terminals, but the other rosette symptoms are lacking. Rosette and
yellows of peach in a general way suggest pecan rosette, but though some
symptoms may be common to all three diseases the complete clinical
picture is distinct in each case. A striking resemblance is to be observed
between pecan rosette and ordinary chlorosis of various trees, where all
gradations occur from mere yellowing of the leaves to cases where the
symptoms closely simulate rosette of pecans. The spike disease of pine-
apples also bears some general resemblance to the rosette of pecans,
both as to effect and apparent cause.

Observations and experimental evidence point to the conclusion that
pecan rosette belongs among the chlorotic diseases of plants grouped
by Sorauer into two main classes: (1) Noninheritable and noninfectious
diseases, due mostly to improper nutritive supply or to injurious physical
conditions, and (2) inheritable and infectious diseases, due probably to
enzymatic disturbances. It seems legitimate to conclude from the data
outlined in this paper that pecan rosette belongs in the first group. The
evidence strongly points in the direction that the disease is caused by
improper nutritive supply, and it seems probable that it is directly re-
lated to a lack of balance between two or more soil ingredients. The
possibility of some relation to soil organisms is not entirely precluded,
but it is thought that the direct cause will ultimately be found in some
lack of balance in the nutritive supply, or possibly in some toxic organic
substances in the soil.

There appears to be little doubt as to a difference in resisting power
toward rosette, but orchard records and observations tend to show that
this difference is usually manifested through the stock rather than
through the variety worked upon it. Good care and fertilization are
to be recommended, but the effects of lime should be tested upon a few
trees before using it on a commercial scale. Pruning is of no avail as a
remedial measure. Trees showing only traces of rosette may be left in
the orchard, but all advanced cases should be cut out and replanted. On
account of resistance versus susceptibility of stock, the discarding of all
rosetted nursery trees is to be strongly advised.
Fig. 1.—One normal pecan leaf and two leaves with rosette from Dewitt, Ga. Note the partial reduction of the leaf blade to the midrib.

Fig. 2.—Pecan shoot with early symptoms of rosette.
PLATE XXV

Rosette pecan leaf showing perforations due to the failure of part of the mesophyll to develop. About natural size. From Cairo, Ga., October, 1903.

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Fig. 1.—Pecan shoot in advanced stages of rosette. Note the raised veins, perforations, and reductions in area of leaf blade; also dying back of terminals. From Cairo, Ga., 1903.

Fig. 2.—Normal pecan shoot for comparison with rosetted shoot. From Cairo, Ga., July, 1903.
PLATE XXVII

Fig. 1.—Young orchard pecan tree with a moderate attack of rosette on the left side and seriously dying back from the disease on the other side. Note the cover crop of cowpeas. From Cairo, Ga., November, 1905.

Fig. 2.—Young orchard pecan tree in advanced stages of rosette. Orchard interplanted with corn. From Blackshear, Ga., November, 1905.
PLATE XXVIII

Fig. 1.—Young orchard tree with severe attack of rosette. Note dying back of branches. Orchard interplanted with corn. From Blackshear, Ga., November, 1904.

Fig. 2.—Rosetted pecan tree cut off to the stump the preceding season, with the present season's growth again distinctly showing rosette. From Cairo, Ga., October, 1903.

Fig. 3.—Two seedling pecan trees planted the same day from the same lot of seedlings. The tree in the background was normal, while the one in the foreground had been affected with rosette almost from the time of planting. Note the effect of a severe and long-standing case of the disease. From Tallahassee, Fla., summer of 1912.