# A COMPARISON BETWEEN GRAPE DEGENERATION IN FLORIDA AND PIERCE'S DISEASE IN CALIFORNIA

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Early Spanish settlers in the 16th century became familiar with the native Florida grapes. The wild grape plants grew profusely, and, because of this, they made plantings of the European grape, *Vitis vinifera* L., that they knew so well. These plantings failed and, by and large, one might say this has remained the history of the European grape in Florida to this day.

Many attempts to grow grapes on a commercial basis in Florida have been made since these early times. DePass in 1891 had 60 varieties on trial near Lake City. They all soon failed. Varieties of the species *Vitis labrusca* L. were also tried at this time, but they did little better and were soon gone. By 1894 a considerable grape industry had been built up in the state and perhaps more than a thousand acres of grapes, mostly the variety White Niagara, were planted. It must have indeed seemed at this time that viticulture was at last established in Florida. However, these plantings began to fail and in less than a decade were gone. This period closely coincides with the time that a "condition" called California vine disease, mysterious vine disease, or Anaheim disease was killing thousands of acres of grapes in southern California.

The grape growers in Florida were able to interest the Federal Government in their problem in 1899 and grape tests were started at Earlton, Florida. These tests were being made by Baron van Luttichau almost concurrently with those of Pierce in California, who was attempting to solve the problem of California vine disease there. The Florida plantings of Luttichau failed but they did indicate that certain rootstocks were beneficial, and that the Munson Texas Hybrids were of some value here. By this time Pierce had made extensive studies of the California vine disease problem and reached the conclusion that the condition in California was not due to soil types, or cultural practices. He also believed the vine disease was not being caused by a fungus or bacteria but was due to some obscure contagious disease producing agent that was spreading through the vineyards. By 1900 the incidence of California vine disease, which

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we now call Pierce's disease, had increased sharply and while we now know the disease was present it was more or less static for several years (until about 1935). Hewitt (4) in 1939 showed that Pierce's disease was caused by a virus.

Hawkins, in 1924, planted almost one hundred varieties of European grapes at Eustis, Florida, on land where the grape phylloxera, *Phylloxera vitifoliae* (Fitch), was known to be absent. The planting was not successful and was abandoned. By this time however, a resurgence of grape culture in Florida had occurred and commercial crops of the Munson Texas Hybrid grapes such as Extra, Carman, R. W. Munson, Muench and Bailey were being produced and shipped to northern markets in carload lots.

The fresh grape industry decreased in Florida during the thirties and little replanting was done. The vineyards declined and grape culture in the state waned again.

By 1946 Stover had reason to believe, from wide experience and observation, that a limited number of grape varieties of *V. labrusca* parentage could be grown if grafted to suitable rootstocks. Dickey, Stover and Parris (1) made definite recommendations in 1947 on varieties and rootstocks. Since that time these combinations have not proven up to expectations and Parris (6) in 1951 stated, "We therefore have no grape variety or combination of varieties in a grafted plant which we can recommend for planting in Florida at the present time."

At this point it would seem that the viticulturist in Florida has once more completed the cycle of promise then failure. Again, let us re-examine the known facts of grape degeneration in Florida in comparison to what is known of Pierce's disease of grape in California.

Pierce's disease has been proven to be due to a virus (4) and several insect vectors are known (3, 8, 9). The syndrome has seasonal variations, and varietal responses differ. The disease has in the past indicated a cyclic tendency (12). Extensive field, greenhouse, and laboratory investigations have replaced speculation and pointed the way to a commercial control of the disease in California.

The occurrence of the symptoms of Pierce's disease in a given plant will depend to a certain extent upon when that plant is infected, its state of growth and the variety. Let us take a hypothetical situation for a discussion of the symptoms and presume we have a healthy plant of a susceptible European

grape, *V. vinifera,* that would show the full range of symptoms. We will also assume this plant is inoculated with Pierce's disease virus in the late summer or early autumn. During the remainder of that growing season probably no discernable differences will be noted. The fruit that has set will probably reach maturity and the vine will go into an apparently normal dormancy that winter. The following spring little or nothing may be detected to indicate early the vine is diseased, except that it is slow to start. After two or three weeks growth this vine would appear to be lagging behind the others. (This delayed growth has often been supposedly due to insufficient chilling or "faulty dormancy".) The next difference to be noted would be that one arm of the vine apparently catches up and seems normal while the canes of the other arm are shorter because the internodes are reduced in length. The basal or first leaves of these canes may then show interveinal mottling. This mottling then appears progressively in successive leaves toward the tip. (Irregular dormancy, fertilizer deficiencies, root damage, certain insects and arthropods, and irrigation practices have been thought in the past to cause this.) By the time the early flush of spring growth has slowed, a marginal burn may begin to show on the basal leaves and an occasional cane may suddenly wilt and dry up. (This has been attributed to such factors as sunburn, high temperatures, insect attack, drought and sulfur burn in the past.) During this first season after infection, a fair set of fruit may occur, but a few bunches may shell early and the fruit stems can be seen on the vines all through the year. (This has been blamed on poor pollination, disease, heavy fruit load and other causes.)

As the summer progresses leaf scorching becomes more severe and interveinal chlorosis may be more distinct. Bunches of green fruit that have appeared normal become flacid and withered, color prematurely, and are insipid in flavor. (Again drought, mechanical damage, disease, heavy fruit load, etc., have been blamed for water berry or withered fruit.) By mid-summer many canes on both arms of the plant may have shed their leaves but in an abnormal manner. Instead of the leaf petiole abscissing from the cane the leaf laminae absciss from the petioles and the petioles remain on the canes. (This opens the vine and sunburn occurs on the unprotected fruit.)

After harvest in the autumn when the vine is again returning to dormancy a very definitive symptom of Pierce's dis-

ease can be seen. Canes instead of turning a normal tan or brown will have green areas persisting between the nodes and in some cases these areas will be slightly raised above the surface of the brown areas. (Occasionally one may note a size differential in the diameter of the pruned arms at this time, but this is not a reliable symptom.)

While the top of the plant is showing these symptoms changes are also taking place in the roots. At first the small feeder roots die back from the tips and become necrotic. In this weakened condition the roots are often invaded by secondary organisms, and the thrift of the vine again suffers. In each succeeding season there is greater root death. (Before the viral nature of the primary pathogen was known the disease was often attributed to these secondary root invaders.)

The grape phylloxera, *Phylloxera vitifolae* (Fitch) is frequently found on the roots of vines suffering with Pierce's disease, and for many years the damage caused by the activities of this insect were believed by some to cause the "condition". This insect has been implicated in like circumstances with grape degeneration in Florida, but has never been actually proven the cause.

The following spring the same pattern will start again with increased severity, and often the arm that was weak before will fail to produce any growth or will have only short weak suckers at the base. The other arm will behave much like the weaker one did in the previous season, and while the vine may live on the second year little or no fruit will be produced and death will soon follow.

An idealized description of the symptoms of grape degeneration in Florida would be very much the same as the foregoing of Pierce's disease in California. From a syndrome stand-point the patterns of the two are remarkably similar if not identical.

If one combines certain of the symptoms described by Rhoads (7) in 1926, under root rot, (which he lists as being caused by the fungus *Clitocybe tabescens* [Scop. ex Fr.] Bres.) non-setting of fruit, shelling of fruit, chlorosis, and irregular water relations, (all listed under injuries due to physiological causes) one has a good description of grape degeneration, or Pierce's disease.

Not all of these symptoms are manifested in every vine suffering from Pierce's disease or grape degeneration, but many different varietal and species reactions are known for Pierce's disease (5). These differential reactions also occur in Florida

in a like manner in respect to grape degeneration and are consistent with those known for Pierce's disease in California (10).

Now let us consider transmission of Pierce's disease virus. Frazier and Freitag (3) have shown the close relationship between the 14 species of leafhoppers known to be capable of transmitting the virus. Severin (9) has reported four species and five varieties of spittle bugs also have this ability. Pierce's disease virus is a persistent type virus as Watson and Roberts (11) define this group. This more or less limits field spread to insect vectors and inadvertent inoculation in propagation activities. In the list of insects known to transmit the virus three are believed to be responsible for most of the field spread in California. These are: the blue-green sharpshooter, *Neokolla circellata* (Baker), the green sharpshooter, *Draeculacephala minerva* Ball, and the redheaded sharpshooter *Carneocephala fulgida* Nott. The redheaded sharpshooter *Carneocephala fulgida* Nott. favors bermuda grass, *Cynodon dactylon* (L.) Pers. as a host plant and Winkler (12) includes this grass as one of the hosts of Pierce's disease.

Many investigators have observed that when bermuda grass invades the vineyards in Florida the vines die out very quickly from degeneration. This observation led the author to make sweep collections on bermuda grass in several localities near Leesburg on June 5 and 6, July 10 and 11 and August 20 and 23 of this year. Large numbers of the yellowhead sharpshooter *Carneocephala flaviceps* (Riley)[2] were taken on bermuda grass in locations where degeneration had eliminated grapes or was quite evident in the vineyards. A few individuals of the species *Draeculacephala portola* Ball and *Draeculacephala inscripta* Van Duzee were also taken on grasses adjacent to a grape nursery where there was a high incidence of grape degeneration. None of these species are proven vectors of Pierce's disease but they are closely related to two of its principal known vector species in California, and as Frazier and Freitag have pointed out (3) should be suspected as vectors because of this relationship.

Observations in Florida areas where grape degeneration has been severe in the past show that several species of plants known to be hosts of Pierce's disease virus in California are present. This is especially true of certain grasses and both wild and cultivated legumes.

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[2] The author is indebted to Professor Dwight M. DeLong of Ohio State University, Columbus, Ohio, for determination of these specimens.

Hairy indigo, *Indigofera hirusta* L., and lupine, *Lupinus angustifolia* L., purposely planted as crops in areas where grape degeneration has occurred, have been observed showing dwarfing and witches broom symptoms. While there is no controlled proof that these species are infected with a virus when these symptoms are present the inference is plain. (Pierce's disease virus also causes serious loss in California in alfalfa plantings. The disease in this host is called Dwarf or Witches Broom.)

Esau (2) has shown that infection with Pierce's disease virus can cause the formation of tyloses and the deposition of gum in the xylem elements of the grape. Specimens of grapes suffering from grape degeneration were taken from the field near Leesburg on June 5, 6, and 19th of this year. Fresh sections were made and microscopically examined. Large numbers of tyloses and heavy gum deposition was found in the xylem elements of these Florida specimens.

What can be said in summary then of the comparison between grape degeneration in Florida and Pierce's disease in California.

1. Past history of both are almost concident chronologically.
2. External field symptoms of both are strikingly similar if not identical.
3. Species of sharpshooters closely related to known vectors of Pierce's disease virus occur in Florida where grape degeneration has long been a problem.
4. Plants proven to be hosts of Pierce's disease virus and its vectors are abundant where degeneration occurs and exhibits symptoms typical of Pierce's disease virus infection.
5. Preliminary laboratory studies show that aberrations in the internal anatomy of grapes suffering from grape degeneration in Florida are similar to those induced by infection with Pierce's disease virus in the same host in California.

The overall field ecologies of Pierce's disease and grape degeneration are much too similar to be reasonably ascribed to coincidence. This ecology as set forth in this paper strongly indicates that what has been called grape degeneration in Florida is actually Pierce's disease. Insect vector trials, insect vector field infectivity trials, insect vector location studies, insect vector life history investigations, host range determinations for both insect vectors and the suspected virus, and grafting experiments are now being conducted to test this hypothesis.

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