Unexplained Citrus Declines

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As the Chairman pointed out, my title is too big for anyone to cover in a short time, and I don't intend to attempt a discussion of all kinds of citrus tree declines. I will confine my remarks to the ones I consider most important, which is a subjective way of saying I am going to talk about the unexplained citrus tree declines--those for which we don't know the cause. My purpose also is to introduce the subject, because later I will be joined by Dr. J. F. Childs, Dr. E. P. DuCharme, and Dr. M. Cohen for a discussion session. We are going to give them an opportunity to rebut what I am about to say, if that is necessary or seems desirable, or amplify anything that I may have slighted, or interject new material into the discussion.

Not everyone would agree, but I am going to say first that Florida has been relatively fortunate on tree declines. We have not had any difficulty here with stubborn disease and greening disease; we have not had a catastrophic situation with regard to tristeza, or xyloporosis, or exocortis, or stem-pitting--but perhaps things are changing for us in Florida.

We have had our own unique problems in Florida. One Florida problem has been spreading decline. Since we alone have the problem, we have been unable to get any research help from elsewhere. Spreading decline has already been discussed on this program, so I will say nothing about that.

Another unique problem is the unexplained stem-pitting discussed by Charles Youtsey and Don Bridges in a paper in 1972 (20). Potentially, this could be a very serious matter for us, but so little is known about it that I am going to pass this matter up without any further discussion.

There may be other, more serious disease problems, but in my opinion there is clearly one more important than the others. So, I am going to talk primarily about blight and young tree decline. After I have reviewed the symptoms, the history, and made some speculations about the cause of these declines, then we will have a general discussion.

First of all, I think we should define what it is we are talking about, so I would like to give the symptoms of blight as best I can. Blight is certainly not a new disease. It has probably been known for approximately 100 years. To give you an idea of how old a disease this is, I would like to relate the symptoms for you of the disease called blight as it was described by Swingle and Webber in 1896 (16). It is really a very good description of the same thing we have in our citrus groves today. Here are a few sentences from their account:

"Blight usually appears very suddenly and on trees that have previously seemed perfectly healthy. The first symptom is a wilting of the foliage, as if the trees were suffering from drought. First, the wilting is slight and can be plainly seen only on hot, dry days, but it soon becomes very pronounced and often continues during the wet season in summer, when rains are almost a daily occurrence. Most cases of blight appear in early spring, from February to April, which is usually a dry season. Sometimes, however, cases occur in mid-summer when the ground is thoroughly wetted every few days. After the wilting becomes severe, the foliage begins to drop and in a few weeks to a few months, according to the severity of the case, the affected branches shed nearly all their leaves. In many cases, the whole top of the tree is attacked at one time, but very often only a single branch shows the disease at first. In such cases, however, the entire tree soon becomes affected."

They also indicate that among the symptoms are the numerous water sprouts that come out from the trunk and scaffold branches of the tree, and that in severe cases the tree succumbs so suddenly the leaves do not fall. Certainly we have seen that in a few groves in the East Coast--the leaves simply wither and turn brown on the twigs.

One other interesting symptom is that sooner or later the leaves become small and usually yellowish as the tree begins to decline. However, they do not mention a mottled foliage condition, as for example zinc deficiency patterns. In 1896 the symptoms of zinc deficiency were not identified by that name, but keen observers as Swingle and Webber would not have overlooked the presence of these symptoms.

A most remarkable fact, they go on to say, is that the roots of blighted trees invariably seem to be entirely healthy. At least at the outset, that certainly would seem to fit our observations today, although, of course, after a tree declines to a marked extent, some diminution in the amount of healthy roots also can be observed.

Blight has been studied for a long time in Florida, but we have never been entirely sure among ourselves in research as to when to determine definitely that a tree was affected by blight. In 1968, Dr. M. Cohen (5) stated that in order to have blight present in a tree, you would have to have all of the following seven symptoms:

1. Non-transmissible die-back and decline of trees five years old and older.

2. Normal trees produced when buds from affected trees are propagated.

3. Failure of trees to recover.

4. Irregular spread of the disease in the grove.

5. Absence of specific plant lesions.

6. Normal growth of replants in disease spots.

7. Abnormally low potassium leaf content of the affected trees.

Dr. L. C. Knorr, formerly at Lake Alfred but now in Thailand, has in preparation a book on citrus diseases which is soon to be published by the University of Florida Press (10). In the manuscript of this book, he makes the following statements:

"Most investigators agree that blight is characterized by three symptoms: 1) a wilting of the foliage despite adequacy of soil moisture, 2) by a die-back that is not the result of obvious damage to trunk or roots, 3) by a post-decline emergence of water sprouts."

But he goes on to say that no one of these symptoms is specific. Each can be caused by something else.

As I said earlier, blight is an old disease. Swingle and Webber indicate that the first mention of blight was by Underwood (18), who was sent in 1891 by the USDA to Florida to make a preliminary study of citrus diseases. He described blight very well at that time. Underwood was followed by Swingle and Webber, whose work was terminated by the great freeze of 1894 and '5. In 1896 they reported on their experiments (16), which were unfortunately mostly inconclusive. They had the last word on blight for about two decades, because of the few trees left in the State in the early 1900's.

As soon as the industry became reestablished in Florida, reports of blight conditions again appeared. Consequently, the Florida Agricultural Experiment Station in 1924 stationed A. S. Rhoads at Cocoa to undertake research work on blight. After 12 years, Dr. Rhoads wrote a bulletin on his field work over that period (14), but he had been unable to determine the cause of the disease, unable to transmit it, and unable to propagate it into other trees. But there were still many trees declining from the disease.

In 1943, Dr. R. F. Suit joined the group at the Lake Alfred Station and for several years conducted an on-going survey of the diseases that he was called out in the grove to observe. In 1947 he and Dr. E. P. Du Charme published a paper enumerating the incidence of the diseases (15). At that time, the most frequently reported tree decline was *Phytophthora* footrot. Second, however, was a condition that they called rootrot, which under present day context I believe to be identical to or at least very similar to what we are calling blight today. The third was spreading decline, and since spreading decline soon became very important to growers, they abandoned work on almost everything else and intensified work on spreading decline.

The research work on blight was taken up by Dr. J. F. Childs at Orlando in the 1940's. In 1953 and in 1965, Dr. Childs published two papers, one reporting his observations on citrus blight (3), and the second presenting a description of a new species of *Physoderma* that he found constantly associated with all citrus trees that he studied (4). Childs speculated about

the relationship between this species of *Physoderma* and the occurrence of blight. However, there was nothing entirely conclusive about that investigation.

About 1955, Dr. Mortimer Cohen took up the pursuit of the elusive cause of blight disease. Among the very interesting things that Cohen did was to go back to those dozens of trees that were planted by Rhoads in the '30's and earlier on Merritt Island. Rhoads had left behind quite adequate notes on where those trees were located and the identity of the budwood and rootstocks of those trees. In spite of the fact that Cohen positively identified the trees, he was unable to find that any of the surviving trees (of which there was a good number) had the symptoms of blight, even though they had been budded from blighted trees in the first place and were on a number of rootstocks (5). Cohen has conducted a wide variety of transmission experiments, but to this date neither he nor anyone else has conclusively been able to transmit blight.

Now I would like to make a few comments on the rootstock situation involving blight. Swingle and Webber made a rather positive statement to the effect that rootstocks were not of any consequence to blight (16). However, in all fairness to them, it should be pointed out that they had very few opportunities to make observations on rough lemon, and they were not working within the cultural practices of today. Rhoads in 1936 (14) said that there was equal frequency of occurrence of blight on sour orange, bitter sweet orange, grapefruit, and sweet seedling, but that there was very little or none on rough lemon. Again, rough lemon was not the most common rootstock that he observed, but there was more of it in the 1930's than there had been in the 1890's. In 1953 Childs (3) considered rough lemon to be susceptible. Suit and DuCharme also reported (15) many trees on rough lemon to be susceptible to this condition. And Cohen (5) believes that all stocks are susceptible but that some are more susceptible than others.

Now let's turn to an examination of young tree decline in the same sequence. First of all, the condition that we are calling "young tree decline" has a variety of names, none of which is based on the Rock of Gibraltar. The reason we called this trouble "young tree decline" at the outset was that it seemed to be an unnamed new disease, and that a conspicuous symptom was the young age of the trees. Later, it became apparent that not only some young trees but also some older trees, and not only trees on the East Coast but also trees in the Interior, were affected by what appeared to be the same thing. Consequently, we found ourselves very confused as to just what name should be used and decided simply to call the condition when it occurred on well-drained soils "sandhill decline," and to continue to call it "young tree decline" in the flatwoods locations or in poorly-drained soils. Later on, some of us realized that if it were not for the fact that most of the decline was on 'ough lemon, we wouldn't be so concerned about it, and some advocated the name "rough lemon decline." However, in all these cases we may very well be talking about the same thing, but that cannot be proven or disproven today.

To begin, I would like to read for you the symptoms of young tree decline as described in Carl Knorr's forthcoming book (10). There are other descriptions--one given by Anderson and Calvert in a recent publication in the Florida State Horticultural Society Proceedings (1)--but this description of young tree decline written by Knorr will suffice.

"Earliest symptoms are a dulling of the foliage, a wilting of the leaves, or a delayed flushing of the tree. Any one of these effects may occur either on a single branch or throughout the tree. These initial symptoms are usually followed by the appearance in some leaves of a chlorosis resembling zinc deficiency but differing in that yellowed areas are speckled with green dots the size of pin heads to nail heads. Trees with speckled zinc deficiency patterns decline rapidly whereas adjacent trees with *typical* zinc deficiency patterns, i.e., without green dots unless caused by insect punctures or melanose pustules, decline only to the limited extent usually associated with zinc deficiency. Successive flushes on affected bran thes produce leaves that are erect, dwarfed, leathery, strap-shaped, and chlorotic. Yellowed leaves are conspicuous during autumn, winter, and spring but disappear in summer, presumably due to shedding. A small percentage of mature fruits are reduced to the size of golf balls and contain curved columellas and aborted seed. As defoliation progresses, twigs begin to die back. Trees do not die completely but sooner or later are removed because of unproductiveness. As in early stages of blight, ronts seem at first to be unaffected, though as canopies become thinner, roots commence to starve and deteriorate. The various symptoms associated with young tree decline are much the same as those pictured under sandhill decline except for the earlier appearance of symptoms in young-tree decline and in the occurrence of the two diseases on different soil types."

I think there would be general agreement that this is a reasonable summary of the symptoms of young tree decline. You will note that the most striking thing about the description is that none of the symptoms really is worth much when one has to make a diagnosis of young tree decline. While I have read you almost a page of symptoms, no one today can positively identify the disease. This is a tremendous disadvantage. One of the greatest needs now is for a test that will within a reasonable

period of time definitely tell whether or not a tree is affected by blight or young tree decline. Cohen's water transmission test is the best thing that has come along so far in that area.

As far as history is concerned, young tree decline may not actually be a new condition. In Rhoads' bulletin published in 1936 (14), is a footnote which reads as follows (and remember that he was talking about blight as it occurred primarily on the East Coast):

"A distinctly different form of decline of citrus trees on rough lemon stock, which has become quite widespread and prevalent in parts of the Ridge section of the State during the last few years, is erroneously termed 'blight' by many growers who are not familiar with the latter trouble. Unlike typical blighted trees, these declining trees do not exhibit a chronic wilting of the foliage, but instead a very pronounced mottled leaf or small leaf condition, especially on the ends of the branches. A vigorous growth of sprouts develops from the interior of the tree as the decline progresses. This trouble also often appears to spread to trees immediately adjacent to those first developing it."

It was also in 1934 that Dr. E. R. Parker and others in California published the first work on zinc deficiency (12), and it may be as simple as that to explain Rhoads' footnote, but the condition, in his mind at least, was confined to rough lemon. So, perhaps we have had this disease also for a long time.

In recent years, the first instance of young tree decline, to the best of my information, called to our attention at the Lake Alfred Station was in 1964, near Wauchula. The second report that came to my attention was in a grove near Fort Pierce in 1965 (11).

The first diagnosis was that this decline looked like blight, but the trees were too young to have blight. Along about that time, the International Organization of Citrus Virologists met in Italy. The thing that they were most concerned about at that 1966 meeting was the prevalence and severity of greening disease. There was some tendency to suspect or even to believe that our trouble here in Florida was greening (9). However, this has not been confirmed, and has in fact been generally discounted because the chemical indicator of greening, gentisoyl, glucose, is not found in Florida trees (8), and because of other discrepancies.

From 1968 forward, there was an increasing number of reports from Extension Agents, Production Managers, and virtually everyone else in the industry, and in 1968, we at Lake Alfred began to intensify our program on young tree decline, dropping other projects which seemed to be of less concern.

At first the concern was almost altogether about sweet orange scions on rough lemon rootstock, and little or none was reported on sweet orange or on sour. That is still in a general way true in that the losses are worst among sweet orange scions on rough lemon rootstock. However, there are declining trees in the field where the symptoms are of young tree decline, and where the rootstock is not rough lemon. Probably several stocks may be affected, but rough lemon still appears to be the one most affected.

Now very briefly I would like to compare the symptoms of young tree decline and blight. don't represent this comparison to be a consensus. There may be many discrepancies.

Let's talk first about the similarities. Wilting is an early or first symptom in both cases. Failure to recover is common. No way has been found to get any of these trees to recover fully and permanently. Sometimes affected trees do make partial recovery and at times, if they have not declined very much in the first place, they may look nearly normal--but all these trees eventually decline again, and stay in decline. There is agreement that at the outset the roots look fairly healthy. Later on, of course, they do not, but in both cases, the root system does not have any visible or readily visible symptoms. There is agreement that the condition cannot be propagated at this time. That is, you can take a diseased tree on rough lemon rootstock and propagate from both the root and the scion, make new trees, and those trees will grow in a healthy fashion. Most of us would agree that at the present time we would not know any way in which this disease could be transmitted from one tree to another. There is circumstantial evidence to indicate that it is being transmitted, but it is only circumstantial. There is a similarity in the reduced transmission of water through the trunk and scaffold branches by techniques worked out by Gohen. Of course, the obvious symptom in both cases is die-back. There appears to be a random distribution of individual declining trees, although in some cases it does not appear to be strictly random from the statistical point of view (6). However, it is random in a general way. This random incidence gives the impression that the disease is spreading in the grove, but without having a firm knowledge of the cause, it would be impossible to verify that spread is occurring.

There are a few differences. Young tree decline is different from blight in that young tree decline affects younger trees than does blight. Also, young tree decline is concentrated on rough lemon rootstock, but blight is not, having been a factor in East Coast groves where rough lemon has never been the predominant rootstock. Blight less commonly has leaf mottling symptoms than is the case in young tree decline. Blight trees have more tendency to put up water sprouts than do trees with young tree decline. One of the most interesting differences is that young tree decline has surged into prominence in the last five years while blight has been known about 100 years. When the last chapter is written on young tree decline, someone is going to have to account for the fact that young tree decline did in fact surge into prominence within the past decade.

Now, finally, I want to speculate about the cause of blight and young tree decline. Virtually every known cause of plant disease has been considered as a possible cause of these diseases.

Since young tree decline first originated on newly-planted flatwoods soils, it was logical to speculate about inappropriate soils. Dr. J. O. Whiteside, in July 1968, transplanted a half dozen trees from a declining grove on the East Coast to the Lake Alfred Center (19). He thus moved those trees to a well-drained soil, whereas they came from a poorly-drained soil. Three of the trees were healthy in appearance and three were in varying stages of decline. The three healthy ones still are apparently healthy and the three that were in varying stages of decline in 1968 still are in that stage of decline. Something was brought along with the transplanted trees. The change from one kind of soil to another kind of soil did not bring about recovery. Since then, several other transplanting experiments have been initiated.

Fertilizer practice has been speculated upon very extensively as a cause for this condition, without anything very consequential being pinned down. Dr. D. V. Calvert of the Fort Pierce Center has, in cooperation with one of the growers in that area, run an experiment involving fertilizer rates. The trees that are heavily fertilized in that experiment show a lower percentage of trees with the symptoms of young tree decline than those fertilized more or less along conventional rates (2). However, this doesn't necessarily tell us a great deal about the cause of the decline. Dr. Calvert and Dr. C. A. Anderson (at Lake Alfred) have conducted extensive leaf and soil analysis, but about the only conclusive thing is that many of the trees in decline have a low leaf potassium content. Again, it is not too instructive, because the same thing results from spreading decline (1) and other diseases.

More than one person has looked at the possibility that this is due to a fungal disease, or a toxin produced by a fungus. Dr. Francis Holmes was very much interested in the latter as a possible cause of the young tree decline, but nothing has come forward to substantiate that possible explanation.

Dr. J. F. Child's work on Physoderma has been discussed above.

Virus diseases, of course, were among the first things everyone thought of, but the lack of ability either to see anything in the electron microscopy or to transmit the disease by a wide variety of budding techniques, has left that unresolved.

Moisture imbalance was the reason that Rhoads finally gave for blight, but that has not appeared to be a satisfactory solution. Dr. A. C. Tarjan has sampled many of the declining groves rather extensively and concluded that nematodes are not related to this disease (17). A good possibility is that the disease is due to a planting of variant stocks. It is not possible at the present time either to discount this or to prove its validity.

Finally, the newest thing for plant pathologists these days is mycoplasms, and we have in the room today one of the leading mycoplasm specialists in the United States, Dr. Clare Calavan. However, when Dr. D. E. Purcifull and Dr. S. M. Garnsey copied Dr. Calavan's methods for working with mycoplasm, they came up empty handed (13).

I believe that I have given a fair evaluation of where we stand. Many people on this campus, at the USDA Horticultural Station in Orlando, at the IFAS Fort Pierce Center, and those at the IFAS Lake Alfred Center are putting a large research effort into this problem. I wish I could predict for you a time at which we would conclusively demonstrate the cause, but unfortunately, that information is not available. In any event, I would like now to call upon Dr. J. F. Childs, Dr. E. P. Du Charme, and Dr. M. Cohen to come forward and take these chairs, and make themselves available for your questions.

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