

Texas Phoenix Palm Decline¹

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Summary

- Texas Phoenix palm decline (TPPD) is a new disease in Florida. This disease is caused by an unculturable bacterium that has no cell wall—a phytoplasma.
- The TPPD phytoplasma is similar to, but genetically distinct from the phytoplasma that causes lethal yellowing (LY) disease of palms.
- Texas Phoenix palm decline is a fatal, systemic disease that kills palms relatively quickly. The TPPD phytoplasma is spread naturally to palms by sap-feeding insects, such as planthoppers.
- Palms showing symptoms of more than 25 percent foliar discoloration or a dead spear leaf due to the disease should be removed immediately.
- Management of TPPD includes protection of susceptible palms in disease-active areas by
 - trunk injection with oxytetracycline HCl (OTC) every three to four months and
 - planting palm species that are not known to contract this disease.
- As of June 2016, palm species known to be most severely affected by TPPD were *Phoenix canariensis* (Canary Island date palm), *Phoenix dactylifera* (edible date palm),

Phoenix sylvestris (wild date palm) and *Sabal palmetto* (cabbage palm).

Introduction

Until recently, **lethal yellowing** (LY) was the only palm disease in Florida caused by a phytoplasma. In late 2006, a second phytoplasma disease was identified in the coastal areas of Central Florida (from Sarasota to Tampa), affecting *Phoenix* (date) species. In early 2008, this second phytoplasma disease was confirmed as the cause of declining *Sabal palmetto* (cabbage palm) in Hillsborough and Manatee counties. The disease is known as Texas Phoenix palm decline because it was originally described in the southern coastal region of Texas on *Phoenix canariensis* (Canary Island date palm).

Pathogen and Hosts

Texas Phoenix palm decline is caused by a phytoplasma, an unculturable bacterium that has no cell wall. Among phytoplasmas, the TPPD agent has been classified as a member of 16S rDNA RFLP group 16SrIV, subgroup D (16SrIV-D). The signature DNA sequence obtained from TPPD phytoplasma in Florida is a perfect match to the signature of the phytoplasma known to cause TPPD on *P. canariensis* (Canary Island date palm) in the Corpus Christi area of Texas. Analysis of DNA has determined the TPPD

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phytoplasma is related to but genetically distinct from the phytoplasma that causes LY.

Phytoplasmas systemically colonize the phloem tissue (vascular tissue transporting photosynthates) of plants. Phytoplasmas are transmitted to plants by piercing-sucking insects that feed on phloem sap. The insects spread the phytoplasma from plant to plant as they visit different hosts during their feeding activities. Phytoplasmas are not known to survive outside their host, whether the host is plant or insect. Planthoppers, treehoppers or psyllids are the most-likely groups of insects to transmit phytoplasmas.

It is not known when the TPPD phytoplasma arrived in Florida, but considerable time would have been necessary for both the pathogen and the vector populations to establish and for TPPD disease incidence to increase to its initial level in Florida. By 2008, the pathogen was primarily a problem in Hillsborough, Manatee, Pinellas, and Sarasota counties. By June 2016, the pathogen had been detected in the 22 counties shown in Figure 1. The disease may be occurring in additional counties, but no samples have been submitted to confirm the presence of the pathogen.

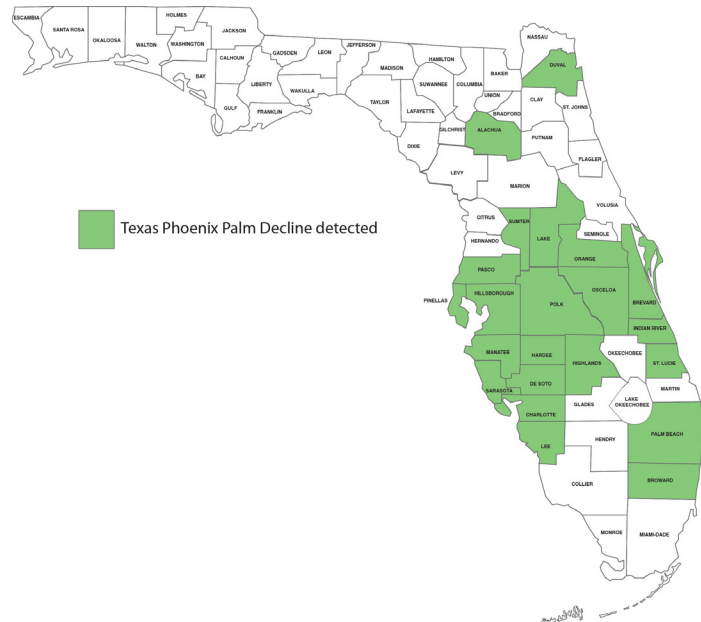


Figure 1. The TPPD pathogen has been detected in 22 counties in Florida.

Credits: Hope Crawford, UF/IFAS

Currently, the known susceptible hosts for the TPPD phytoplasma are *P. canariensis*, *P. dactylifera*, *P. sylvestris*, *P. reclinata*, *Sabal palmetto*, and *Syagrus romanoffiana* (queen palm). Only a few queen palms have been confirmed with this disease, whereas the disease has been observed widely among *Phoenix* species and *Sabal palmetto*. The TPPD phytoplasma has also been detected in *P. roebelenii* and *xButiagrus nabonmandii*, but only once for each species.

Symptoms

The symptoms of this new disease appear to be exactly the same as those associated with LY of *Phoenix* species.

The first obvious TPPD symptom on mature palms is premature drop of most or all fruits at one time (Figure 2). The fruit drop occurs within a few days. The fruit drop is not spread out over a prolonged period of time. Inflorescence (flower) necrosis (death) follows (Figure 3). However, these two symptoms will only be observed if the palm is mature enough to produce fruit, if it is the season for flowering and fruiting, and if the flowers or fruits have not been trimmed from the palm.



Figure 2. Premature fruit drop is an early symptom of TPPD. Virtually all the fruit drops at one time from the affected tree.



Figure 3. Death of the inflorescence (flowers) is an early symptom of TPPD. This photo also illustrates a dead spear leaf (youngest leaf that has not unfolded), which is tan and not green.

The next symptom is discoloration of the foliage, beginning with the oldest leaves. The leaves do not turn yellow (or do so briefly), but quickly turn varying shades of reddish-brown to dark brown or gray (Figures 4 and 5). The discoloration begins at leaf tips (Figure 6).



Figure 4. Discoloration of the lowest (oldest) leaves is an early symptom of TPPD.



Figure 5. Discoloration of the lowest (older) leaves is an early symptom of TPPD in cabbage palm.



Figure 6. Discoloration of the leaves begins at the leaf tips.

Unless the palm is being monitored closely, the onset of leaf discoloration is usually first recognized as a greater number of dead older leaves than is normal for natural senescence. This symptom might be confused with other problems, such as early senescence due to nutrient deficiency (e.g., potassium) or *Ganoderma* butt rot. However, if the dying or dead leaves are being regularly removed (nobody likes an untidy palm!), even this TPPD symptom of foliage discoloration and death may not be obvious.

In *Phoenix* species, when less than one-third (and usually less than one-quarter) of the oldest leaves have discolored and become necrotic, the spear leaf dies (Figures 3, 7, and 8). Death of the spear leaf indicates the apical meristem (bud or heart) has died. Once the apical meristem has died, no new leaves will develop, and the remaining leaves will continue to discolor from the oldest to the youngest leaves. In cabbage palms, approximately two-thirds of the oldest leaves will have discolored before the spear leaf dies (Figure 9).



Figure 7. *Phoenix sylvestris* exhibiting symptoms of TPPD. Note more dead lower leaves than normal for a healthy palm. Also, the spear leaf (youngest leaf that has not unfolded) is tan and not green.



Figure 8. Close-up of dead spear leaf in Figure 7.



Figure 9. *Sabal palmetto* (cabbage palm), exhibiting symptoms of TPPD. Note more dead lower leaves than is normal for a healthy palm. Also, the spear leaf (youngest leaf) has died and is a tan color, rather than green.

In some instances, by the time the spear leaf dies, mature roots of the palm at or near the soil surface are soft in texture and easily broken. The palm may be easily rocked back and forth in the ground because the root system is decaying. This symptom is not typical for palms affected by LY.

Death of the spear leaf may not always be obvious. Both *Phoenix* species and cabbage palms (*Sabal palmetto*) have numerous young leaves surrounding the spear leaf. Unless you see the spear leaf is dead (Figure 3 and 7) or find it hanging from the canopy (Figure 10) or on the ground, you will probably need to physically examine the canopy up close to determine whether a healthy spear leaf is present (Figure 11). Also, since the young spear leaf of *Phoenix* palms—in normal, healthy conditions—is often enclosed in a thin, brown sheath (which tears like paper), be careful not to confuse the normally occurring brown sheath for a dead spear leaf.



Figure 10. Note the spear leaf has died and is hanging down from the canopy of this *Phoenix sylvestris* affected by TPPD.



Figure 11. The spear leaf has already died in this *Phoenix sylvestris*, and the spear leaf has broken off from the canopy. Unlike the examples shown in figures 6, 7 and 9, without a close examination of the bud on this palm, it would not be apparent that the spear leaf had died.

Queen palms have been diagnosed with the TPPD phytoplasma. Leaf necrosis was exhibited on the lowest leaves first and continued upward through the canopy. It is not yet clear at what stage in the disease process the spear leaf dies, but initial observations indicate the spear leaf dies early in the disease process. TPPD symptoms observed in queen

palms are distinctly different from Fusarium wilt, another new disease of queen palms. (For information on Fusarium wilt in queen palms, see the following: <http://edis.ifas.ufl.edu/pp278>.)

Diagnosics

Initial diagnosis of TPPD is based on the palm symptoms described above. Since the phytoplasma is not culturable, a molecular diagnostic test is used to confirm the presence of the pathogen. If pathogen confirmation is necessary, contact your local UF/IFAS Extension office—<http://solutionsforyourlife.ufl.edu/map>—for information on sample submission and cost of laboratory diagnosis. This information is also available at <http://frec.ifas.ufl.edu/media/frecifasufledu/pdfs/LY-TPPD-Trunk-Sampling.pdf>.

Sampling is accomplished by boring into the trunk. The sampling process requires a drill with a long, large-diameter drill bit. Do not obtain samples without first reviewing the complete set of instructions. The quality of the sample is critical for an accurate diagnosis. Samples must be sent via overnight service.

It is currently not known how early in the disease cycle TPPD phytoplasma can be detected via trunk-tissue sampling. However, the TPPD phytoplasma is usually not detectable in palms that are not exhibiting symptoms. This phytoplasma may not be detectable until the spear leaf dies.

Phytoplasma detection by means of testing a trunk tissue sample is like searching blindfolded for a red marble in a bowl that is mostly full of white marbles. If there is only one red marble (phytoplasma) in the bowl of white marbles (trunk tissue), the likelihood of selecting that red marble, if you were blind folded, would be slim. However, the likelihood of selecting a red marble increases as the number of red marbles increase.

The molecular test for the TPPD phytoplasma is best used to confirm the presence of the disease in symptomatic palms in a nursery or community in order to track the spread of the disease and to devise a management program for remaining, susceptible palms. The molecular test does not certify a palm is phytoplasma free.

Remember that palms die or appear to be dying for a number of reasons; Texas Phoenix palm decline is just one reason. Before submitting a sample, take the time to make the best possible field diagnosis.

Disease Management

If the spear leaf has died, the palm should be removed as soon as possible. Death of the spear leaf indicates the apical meristem (bud) has died, so no new growth will occur. Although lower leaves may remain green for a number of months after the spear leaf dies, it is in the best interest of the nursery grower or the community to remove the infectious palm as soon as possible. The diseased palm serves as a source of the phytoplasma that can be transmitted by an insect vector to still-healthy, TPPD-susceptible palms.

If TPPD symptoms are present, but the spear leaf has not died, therapeutic treatment of the disease may be achieved by application of the antibiotic oxytetracycline HCl (often referred to as OTC), administered to palms by liquid injection into the trunk. Treatments would continue for the life of the palm on a three- to four-month treatment schedule.

The most effective use of the antibiotic is as a **preventive** treatment to protect susceptible palms when TPPD is known to occur in the area. Again, these preventative antibiotic treatments should be made every three to four months. However, only palms known to be susceptible should receive the treatments. Palms known to be most susceptible to TPPD, based on the number of samples received, are the following: *Phoenix canariensis* (Canary Island date palm), *Phoenix dactylifera* (edible date palm), *Phoenix sylvestris* (wild date palm), and *Sabal palmetto* (cabbage palm).

The antibiotic (OTC) should be injected at the rate of 1–3 grams per palm. Because there are a number of sources of OTC, be sure to read the label carefully, especially in regards to percent active ingredient, to ensure an adequate amount of OTC is being injected into the palm.

As with LY, landscape disease management of TPPD via control of the vector population is not recommended, especially since the TPPD vector is unknown at this time. Use of host resistance represents the most practical long-term solution. However, the complete palm host range of this phytoplasma is probably not yet known.

References

- Harrison, N. A., E. E. Helmick, and M. L. Elliott. 2008. "Lethal yellowing-type diseases of palms associated with phytoplasmas newly identified in Florida, USA." *Annals of Applied Biology* 153:85–94.
- Harrison, N. A., E. E. Helmick, and M. L. Elliott. 2009. "First report of a phytoplasma-associated lethal decline of *Sabal palmetto* in Florida, USA." *Plant Pathology* 58:792.
- Harrison, N. A., M. Womack, and M. L. Carpio. 2002. "Detection and characterization of a lethal yellowing (16SrIV) group phytoplasma in Canary Island date palms affected by lethal decline in Texas." *Plant Disease* 86:676–681.
- McCoy, R. E. 1975. "Effect of oxytetracycline dose and stage of disease development on remission of lethal yellowing in coconut palm." *Plant Disease Reporter* 59:717–720.
- McCoy, R. E. 1982. "Use of tetracycline antibiotics to control yellows diseases." *Plant Disease* 66:539–542.