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NEWLY EMERGING DISEASE THREATS TO PALMS IN FLORIDA

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INTRODUCTION

The subtropical southern one-third of the Florida peninsula (hardiness zone 10) is conducive to the growth of a great diversity of exotic palm species in addition to the 12 species native to the state and the southeastern United States (Meerow 2006). Although the exact tally of species grown in southern Florida is unknown, the living collection of at least 289 genera and 327 species of palms grown within the confines of Fairchild Tropical and Botanic Garden in Coral Gables, Miami-Dade County (http:www.fairchildgarden.org) provides an excellent indicator of the wealth of species available to palm enthusiasts for use as ornamentals in landscape, amenity, or niche plantings.

Plantings of mature palms provide instant, upscale appeal to landscape architectural designs and are in high demand by both residential and commercial developments. However, despite a broad palette of palm species from which to choose, practical considerations, including the porous, alkaline, nutritionally deficient soils of coastal southern Florida, coupled with a backdrop of both native and introduced pests and diseases (Broschat and Meerow 2000; Elliott et al. 2004), have collectively shaped the common usage of palms in landscapes to about 18 favored species. In the central and northern reaches of the state, hardiness zones 9 and 8, respectively, the choice of palms for plantings is restricted to just a few cold-tolerant species. Furthermore, for palms of large stature, choices of cold tolerant palms are limited largely to highly valued *Phoenix* species and hybrids (*P. canariensis; P. dactylifera; P. sylvestris*), Queen palm (*Syagrus romanzoffiana*), Mexican fan palm (*Washingtonia robusta*), and native sabal (cabbage) palm (*Sabal palmetto*), the most widely used species in urban and suburban plantings.

LETHAL YELLOWING OF PALMS: A LINGERING DISEASE WITH DESTRUCTIVE POTENTIAL

Prior to the 1970s, coconut palm (*Cocos nucifera*) was the most popular and abundant species grown throughout southern Florida. Unique among palms in its ability to evoke an exotic tropical appeal to coastal landscapes, this species is often prominently featured in advertisements to promote Florida tourism. In 1971, the first cases of lethal yellowing (LY), a disease of uncertain etiology at that time, was reported on mainland southern Florida (Seymour et al. 1972). Known to sporadically disrupt coconut production in Cuba and Jamaica since the 1800s (Eden-Green 1997), this fast moving, quickly fatal disease killed most of the resident population of Jamaica tall (Atlantic tall ecotype) coconuts, estimated at 700,000 palms in southeastern Florida, within a

five-year period (McCoy et al. 1983). Mortality of 35 other ornamental palm species attributed to LY was also documented during this time (McCoy et al. 1983; Harrison and Oropeza 2008). Catastrophic losses of Atlantic tall coconuts (>7 million palms) to LY in Jamaica during the same era were followed by similar epiphytotics in southeastern Mexico (Oropeza and Zizumbo 1997) and in northern Honduras during the 1990s (Eden-Green 1997). Recurrent widespread mortality of resistant Malayan dwarf and hybrid Maypan coconuts in Jamaica since the late 1990s indicates that as yet undefined changes within the phytoplasma-vector-palm pathosystem have occurred there (Lebrun et al. 2008). To-date, similar unusual increases in disease among susceptible palm species in southern Florida have not been observed.

Originally confined to just four contiguous southeastern counties, bounded by Monroe County in the south and Palm Beach County in the north, long-distance dispersal to and establishment of LY in Lee County on Florida's southwestern coast occurred during the late 1980s. Although further southward advance of the disease into neighboring Collier County, which contains the largest remaining population of about 80,000 Atlantic tall coconut palms in the state has occurred, palm mortality was effectively minimized by the implementation of a rigorous disease management program based upon prompt identification and removal of diseased palms in concert with proactive treatments of adjacent symptomless palms with oxytetracyline hydrochloride (OTC-HCl) until disease abatement, at which time treatments were discontinued (Fedelem 2000). For the last four decades, the persistent threat of LY has demanded the judicious use of susceptible palms, resulting in the widespread popularity of species, such as Queen palm, Royal palm (*Roystonea regia*), Mexican fan palm, Pygmy date palm (*Phoenix roebelenii*), and sabal palm, for landscaping as they do not to succumb to the disease (McCoy et al. 1983; Meerow 2006).

Phytoplasmas are the accepted cause of LY based on their consistent detection in diseased palms by transmission electron microscopy (TEM) Plavsic-Banjac et al. 1972; Thomas 1979), remission of symptoms in response to tetracycline therapy, and absence of any other pathogen. Phytoplasmas are small, unculturable, cell wall-less bacteria, ovoid to filamentous in shape, with minimal (560–1300 kb) A+T-rich genomes (Christensen et al. 2005; Harrison et al. 2010; Kollar and Seemüller 1989). Phytoplasmas have evolved as obligate, intracellular parasites that inhabit the phloem of plants as well as tissues and organs of their various phloem-feeding insect vectors (Garnier et al. 2001; Lee et al. 2000; Weintraub and Beanland 2006). Due to their small size and obligately parasitic habit, confirmation of phytoplasma diseases has traditionally relied upon *in situ* detection of phytoplasmas in host tissues by TEM. Today, more sensitive molecular assays tailored to detect phytoplasma DNA have largely replaced TEM as the method of choice for confirming phytoplasma infection of plants, especially woody perennial hosts such as palms in which they typically occur in low abundance (Harrison et al. 1994).

Sensitive DNA-based diagnostics employing polymerase chain reaction (PCR) assays capable of detecting phytoplasmas in a "universal" group-specific or pathogen-specific manner have been developed (Lee et al. 2000). Assays that amplify 16S rRNA genes when combined with restriction fragment length polymorphism (RFLP) and sequence analysis of resulting rDNA products have also provided a means to precisely identify and classify phytoplasmas for taxonomic purposes (Lee et al. 2000). Twenty-eight 16S rDNA RFLP (16Sr) groups comprising numerous subgroups of strains have been described thus far (Wei et al. 2007). PCR amplification

and RFLP analysis of rDNA have demonstrated that phytoplasmas associated with LY disease of palms in southern Florida exist as a homogenous population of strains (Harrison et al. 2002a). Originally assigned as the sole member of group 16SrIV (coconut lethal yellows group) subgroup A (i.e., 16SrIV-A), at least five additional subgroups of strains have since been identified within the group, including 16SrIV-B, which includes Yucatan coconut lethal decline phytoplasma (YLD) (Lee et al. 2000); 16SrIV-C, which is represented by Tanzanian coconut lethal disease (TLD) phytoplasma (Lee et al. 2000); 16SrIV-D, which contains *Carludovica palmata* yellows (CPY) phytoplasma; 16SrIV-E, which includes phytoplasmas associated with coconut in the Dominican Republic (Martinez et al. 2008); and 16SrIV-F, which is represented by a phytoplasma detected in a *W. robusta* in west-central Florida (Harrison et al. 2008).

TEXAS PHOENIX PALM DECLINE: A NEWLY EMERGING DISEASE OF UNCERTAIN DESTRUCTIVE POTENTIAL

Prior to 2006, no evidence for northerly dispersal of LY beyond the southernmost subtropical tier of Florida had been documented. This well-established zone of confinement mirrors the geographic boundaries within which coconut palm can be successfully grown and encompasses the native range of the neotropical planthopper vector *Haplaxius* (*Myndus*) *crudus* of LY (Howard et al. 1983; 1984), which is not cold hardy. In light of these observations, discovery of approximately 10-year-old, seed-grown *P. sylvestris*, *P. sylvestris* x *P. dactylifera* and *P. sylvestris* x *P. canariensis* hybrids with fruit and foliar discoloration symptoms and ensuing mortality indicative of LY (McCoy et al. 1983) at a palm field nursery in Hillsborough County, west-central Florida, was a new and unexpected development. Foliar and root decay symptoms similar to those observed on *P. sylvestris* and hybrids were also found affecting immature Queen palm (*Syagrus romanzoffiana*) interplanted with symptomatic *P. sylvestris*. Phytoplasmas were subsequently detected by 16SrIV group-specific PCR assay (Harrison et al. 2008) in all declining palms sampled at this site as well as in declining *P. canariensis* and *P. dactylifera* palms located in surrounding communities. RFLP analysis augmented by sequencing of rDNA products from PCR-positive palms identified the associated phytoplasmas as subgroup 16SrIV-D strains not previously documented to occur in Florida. The fact that these strains were indistinguishable from phytoplasmas associated with Texas Phoenix palm decline (TPPD), a lethal disease of *P. canariensis* in Corpus Christi, Texas during 2000 (Harrison et al. 2002) was also noted.

Surveys for diseased palms were conducted during 2007 in Hillsborough, Manatee, and Sarasota Counties by Florida Division of Plant Industry scientists with assistance from the United States Department of Agriculture's Cooperative Agricultural Pest Survey (CAPS) program. Samples from suspect palms from within these three contiguous counties were also provided by University of Florida Cooperative Extension Service personnel, landscape maintenance businesses, and homeowners for molecular diagnostic analysis. The cumulative diagnostic data indicated a decreasing TPPD disease incidence from north to south, implicating Hillsborough, the most northerly county, as the probable epicenter of infestation by subgroup 16SrIV-D phytoplasmas (Harrison et al. 2008). While the majority of sampled palms in the contiguous tri-county area contained subgroup 16SrIV-D phytoplasmas, two *P. canariensis* palms were found to contain subgroup 16SrIV-A phytoplasmas in Sarasota County thus extending the known northward distribution of LY in the state. Also, in Sarasota County, a solitary *W. robusta* was found to harbor a previously undocumented subgroup 16SrIV-F phytoplasma, as did two *P.*

dactylifera, although this novel strain was detected in the latter palms as a mixed infection along with subgroup 16SrIV-A phytoplasmas. While little is known about this new subgroup 16SrIV-F strain, its close rDNA sequence similarity (99.6%) and occurrence as mixed infections with subgroup 16SrIV-A strains in *P. dactylifera* may be of epidemiological and evolutionary significance, as it suggests that both strains are vectored by *H. crudus*.

A program of continued surveillance and sampling of palms has determined that TPPD currently remains most active in the aforementioned tri-county region. However, isolated cases of the disease, mostly affecting *P. sylvestris* palms, have been identified in Flagler, Highlands, Pinellas, Polk, Lake, and Lee Counties. Solitary cases of TPPD disease affecting three new palm hosts, namely, Pygmy date palm (*P. roebelenii*), Senegal date palm (*P. reclinata*), and mule palm (*S. romanzoffiana* x *Butia capitata*), have since been confirmed in Hillsborough County (unpublished data).

In 2008, unusually large numbers of dead and declining *S. palmetto* palms with reddish-brown leaves were first recognized in Manatee and Hillsborough Counties. Common in urban and suburban ornamental landscapes in both counties, the sabal palm is most abundant and widely distributed in rural and coastal forest habitats, and is considered integral to the ecology of most ecosystems statewide. Etiological study of affected palms using molecular diagnostic techniques attributed this newly recognized disease to phytoplasmas (Harrison et al. 2009). As such, *S. palmetto* represents the first native palm species to be impacted by a phytoplasma disease in Florida, and is a cause of great concern regarding the long-term survival of this stalwart species. Phylogenetic analysis of PCR-amplified DNA sequences has attributed the etiological agent of the disease to phytoplasmas seemingly co-identical with subgroup 16SrIV-D phytoplasmas, which was previously associated with declining palm and palm-like hosts in other studies (Cordova et al. 2000; Harrison et al. 2002; Ong and McBride 2009; Vázquez-Euán et al. 2011).

Early-stage foliar symptoms of TPPD on *S. palmetto* are most difficult to diagnose, especially on palms in natural stands, as they are commonly affected by nutritional deficiencies, especially potassium deficiency, inducing discoloration and premature senesecence of up to half of the lower leaves within the palm canopy. Also, *S. palmetto* flowers only once annually, in mid-year, allowing just a short window of opportunity to evaluate palms for evidence of disease-induced inflorescence symptoms. The species is known to die from a variety of other causes such as Ganoderma basal trunk rot, bud rot due to *Phytophthora palmivora*, infestation by (*Rhyncophorus cruentatus*) palm weevils (Meerow 2006; Elliott et al. 2004) and abiotic factors such as saltwater intrusion and drought (Desantis et al. 2007; Williams et al. 1999). Collectively, these factors conspire to complicate visual estimates of disease incidence or distribution.

Currently, sabal palms most affected by TPPD have been observed along a 32-mile corridor from Brandon in western Hillsborough County to Bradenton in northern Manatee County. The incidence of palm mortality has been highest in and around the towns of Parrish, Ruskin, and Palmetto in Hillsborough County. Isolated cases of TPPD-associated sabal decline have been confirmed in five other counties, namely, De Soto, Hardee, Highlands, Polk, and Sarasota. Research currently sponsored by the USDA/TSTAR-C program to provide estimates of the genetic structure of *S. palmetto* and sabal decline phytoplasma populations and to identify insect

vector(s) should provide the means to gauge the long-term impact of the disease on the survival of this palm species in Florida and the southeastern United States.

FUSARIUM WILT OF QUEEN AND MEXICAN FAN PALMS

Since late 2004, a new disease of Queen palm (*S. romanzoffiana*) and Mexican fan palm (*W. robusta*) has spread across the southern half of Florida The disease is caused by a novel fungus *Fusarium oxysporum* f. sp. *palmarum* and largely affects mature palms in landscape settings (Elliott et al. 2010). Early-stage symptoms on both Queen and Mexican fan palms resemble those previously attributed to Fusarium wilt of Canary Island date palm (*P. canariensis*), a disease that is well established in Florida and is caused by *Fusarium oxysporum* f. sp *canariensis* (Simone 2004). Initial symptoms appear on individual leaves of affected palms as chlorosis and one-sided tan to brown necrosis of the leaf blade together with a distinct reddish-brown stripe along the length of the petiole and rachis. Internal tissues beneath the stripe are also discolored. Affected leaves quickly turn necrotic. Foliar symptoms typically appear first on older lowermost leaves and then progress to successively younger leaves until all leaves in the canopy succumb (a process that is 2–3 months in duration). Necrotic leaves quickly dessicate but remain in place and do not droop, break, or collapse. At this terminal phase of disease development, the apical meristem becomes infected and dies, after which the pathogen then invades the stem tissues.

F. oxysporum f. sp. *palmarum* can be consistently isolated in culture from internal tissues of symptomatic petioles and rachides of affected palms. Pathogenicity tests on seedlings using foliar pour on inoculation have confirmed that isolates of the pathogen from either *S. romanzoffiana* or *W. robusta* are pathogenic to both palm species. Confirmation of pathogen involvement and identity is most efficiently determined by molecular characterization using pathogen DNA in a PCR assay incorporating primers that amplify a portion of the translation elongation factor 1-α, a highly informative region for differentiating forma specialies within the *Fusarium oysporum* complex as well as other *Fusarium* spp. The resulting sequences are then best queried for similarity against sequences archived in the FUSARIUM-ID database (http://fusarium.cbio.psu.edu) (Geiser et al. 2004).

The peninsular wide occurrence of the disease attributed to *F. oxysporum* f. sp. *palmarum* in southern Florida suggests either that the pathogen has been present for a considerable time prior to its initial discovery or that it has been rapidly disseminated across the region since 2004 primarily as airborne conidia, rather than by root infection or on contaminated pruning tools as is typical for spread of *Fusarium oxysporum* f. sp *canariensis*. To-date, the disease appears to be uniquely of Florida origin and is incurable at present. Further research on the disease will be needed to determine whether any other palm species are susceptible. The rapidity of observed palm mortality suggests that fungal toxin may be involved in the host-parasite interaction but remains to be demonstrated. From a disease management standpoint, it is unclear whether fungal spores in the soil can lead to root infection of replacement palm plantings, or whether there are fungicides that could be used to protect susceptible palms in areas of active disease.

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