Phytophthora Bud Rot of Washingtonia Palm

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ABSTRACT


A previously undescribed bud rot of 5-yr-old Washingtonia palms in Florida was shown to be caused by Phytophthora palmivora. The symptoms included wilt; eventual desiccation of the youngest leaves to a straw brown color; and tan-colored, necrotic lesions with brown margins in interior petiole bases adjacent to putrid, gray bud tissue. Mature leaves remained healthy for several weeks after death of the bud. The pathogen was isolated from diseased palms and surrounding nursery soil. Two- to three-year-old palms inoculated with palm or soil isolates developed symptoms identical to those observed in naturally infected palms, and the pathogen was easily reisolated.

Washingtonia palms (Washingtonia robusta Wendl. and W. filifera (Lind.) Wendl.) are important ornamentals in Florida because of their tropical appearance, tall growth habit, and resistance to the palm lethal declines (10) that have destroyed thousands of coconut and other palm species in Florida. In addition, they are only occasionally subject to other serious diseases.

A disorder resulting in the death of 5- to 6-yr-old W. robusta palms in a south Florida field nursery was brought to my attention in April 1979. Symptoms consisted of desiccation of the youngest leaves and bud rot. Preliminary investigations indicated the presence of a Phytophthora sp. in affected tissue. Before the epidemic of lethal declines in Florida, bud rots of coconut palms caused by Phytophthora spp. were particularly important in low-lying areas frequently inundated by water (9). Two reports from California have shown the involvement of Phytophthora spp. in a trunk rot (2) and a collar rot (4) of W. filifera and W. robusta, respectively. The symptoms of the diseases described in these reports differ in several respects from each other and from the disorder observed in Florida. This paper reports the results of investigations on the etiology of the disease in Florida.

MATERIALS AND METHODS

Palsms in the field nursery were planted 2 m apart in 144-m-long, north-south rows about 3 m apart (Fig. 1). The foliage provided a dense canopy in the rows and often nearly touched across the rows. Unpaved roads bordered three sides of the field. On the fourth side were other palm and various dicotyledonous nursery species. Each palm showing symptoms was charted on a map of the nursery. The palms ranged from 1.3 to 2 m tall.

Tissue for isolation was obtained from bud tissue or from petiole bases of young expanded leaves adjacent to the bud of diseased palms. All original isolations from diseased palm tissue were made on water agar, fresh potato-dextrose agar with 1.0% dextrose (PDA), or a medium selective for pythiaceous fungi containing pimaricin, vancomycin, and pentachloronitrobenzene in cornmeal agar (11). A modification of the latter medium (7) to which was added 3-hydroxy-5-methylisoxazole (5) at 50 μg/ml was used for isolations from inoculated, diseased palms. Cultures of the Phytophthora sp. were maintained on clarified V-8 juice agar (8). Cultures grown in V-8 juice broth (13) were used to produce sporangia for identification. Growth response to temperature was determined by measuring the radial growth of the fungus isolates on PDA (2% dextrose) in 9-cm-diameter petri dishes kept for 5 days at 18, 21, 24, 27, 30, or 33 C. Measurements of growth at 35 C were made on cornmeal agar.

Two-year-old W. robusta palms grown

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<th>WASHINGTON PALM NURSERY</th>
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<td>nursery block</td>
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<td>trunk road</td>
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<td>unpaved road</td>
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<td>nursery block</td>
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<td>contiguous nursery block</td>
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Fig. 1. Incidence of bud rot in the Washingtonia robusta palm nursery is indicated by dark circles. Palms were planted 2 m apart in rows 144 m long and 3 m apart.

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Plant Disease/June 1982 517
in 15-cm-diameter pots (about 3 L) were used for inoculation experiments I and II. Three-year-old palms grown in 22-cm-diameter pots (about 6 L) were used for experiment III. Two parts, by volume, of a commercial potting mix formulation (2:5:3 mixture of sand:pygmy tree shavings:Florida peat; one part Canadian peat; and one part perlite composed the soil mix. It was amended with dolomite at 5.8 kg/m² and a resin-coated, 14-14-14 fertilizer (Osmocote, Sierra Chemical Company, Milpitas, CA) at 3.6 kg/m² before being pasteurized at 60 C for 30 min. Subsequent fertilizer applications were top-dressed at 8-wk intervals at the rate of 3.6 kg/m².

Two isolates, one obtained from a diseased palm (E79-2) and one from surrounding nursery soil (E79-15), were each used in inoculation experiments and observed for cultural characteristics. Inoculum was grown on V-8 juice agar for 2 wk in 9-cm-diameter petri dishes, and 1-cm-diameter disks were cut out 1 cm from the edge of the petri dish. One disk per palm was placed on unwounded petiole bases or on petiole bases first wounded with a scalpel by making an incision 1 cm long and 0.5 cm deep. The incisions were made 3-4 cm above the soil line, at which point the diameters of the crowns were 3-7 cm. Strips of plastic film were wrapped around the inoculation sites to hold the disks of inoculum in place and to prevent desiccation. Disks of sterile V-8 juice agar were similarly placed over incisions on control plants. The plants were then randomized and maintained on a bench in a shade house (65% shade).

RESULTS
Symptomatology included wilting of the youngest leaves, first manifested as a change in leaf color to a paler green that progressed to a green brown and eventually to light brown as the leaves became desiccated. The early wilt of the youngest leaves was also evidenced by a refolding of the leaf blades. Careful, progressive removal of the petiole bases of affected palms revealed tan-colored, necrotic lesions, with brown margins. The surfaces of some lesions were covered by layers of white mycelia (Fig. 2), which produced sporangia when incubated in a moist chamber. Pure cultures of the pathogen were obtained upon transfer of mycelia to a selective medium (11). Successive lesions in petiole bases progressed to the diseased bud.

A 1- to 2-mm-wide, brown margin separated gray, slimy, foul-smelling, diseased bud tissue from creamy white, healthy tissue. Only the petiole bases of recently expanded leaves and the undifferentiated tissue of the bud were susceptible to inoculation. In early stages of disease, the petiole bases of the youngest visible leaves were rotted, but the bud was still intact. The oldest leaves persisted for several weeks after the bud had been killed and did not exhibit symptoms of moisture or nutrient stress. Stem tissue was not affected until after death of the bud.

More than 75% of the diseased palms in the nursery were located in the first two rows adjacent to one road and within 44 m of each end of the other rows. Many palms had been removed before our survey of the field because of sales of healthy trees and removal of diseased ones. Therefore, a precise estimate of the number of palms killed by the disease could not be made. None of the adjacent palm species (Phoenix roebelenii O'Brien and Arecastrum romanoffianum (Cham.) Becc.) showed symptoms of disease.

A Phytophthora sp. was consistently isolated from diseased trees and from surrounding nursery soil. Isolates originating from diseased palms or from soil grew best at 27-30 C on PDA and did not grow at 35 C. Sporangia of isolates E79-2 and E79-15 averaged 72.4 x 43.0 µm and 64.2 x 38.6 µm, respectively, and the length-to-breadth ratio for each was 1.7:1. Both isolates were of mating type A' (determined by D. J. Mitchell and M. E. Mitchell). Oospores were not observed in single-isolate culture but were produced when paired with A1 isolates of P. palmivora Butl., P. cinnamomi Rands, and P. parasitica Dust. Based on the above characteristics, the pathogen was identified as P. palmivora according to the description by Waterhouse (12).

Symptoms identical to those observed in naturally infected palms resulted in each of three experiments in which 2- to 3-yr-old palms were inoculated with palm isolate E79-2 or soil isolate E79-15. In inoculation experiment I, three of five and two of five palm wounded with isolates E79-2 and E79-15, respectively, developed symptoms within 3 wk of inoculation. In experiment II, the numbers of palms developing symptoms within 8 wk of wound-inoculation were two of four for each isolate. The number of unwounded palms developing symptoms within the same period was one of four for each isolate. In experiment III, two of each wounded inoculated palms developed symptoms within 6 wk of inoculation. None of the control palms developed symptoms, and the symptoms in experimentally inoculated palms were identical to those observed in the larger plants in the field nursery. Isolations from selected diseased, experimentally inoculated palms yielded fungi resembling those used for inoculation.

DISCUSSION
This is the first published account of a disease of Washingtonia palm caused by a Phytophthora sp. in which bud rot was a primary phase of the disease and not a symptom following the general destruction of other parts of the palm. Previous authors (1,3,9) have reserved the term "bud rot" for a specific disease of coconut and Borassus palm (Borassus flabellifer) that is caused by P. palmivora. The symptoms observed in Washingtonia palms fit the descriptions of these authors except that no leaf spotting occurred in naturally diseased Washingtonia palms. A single diseased, young leaf was occasionally found growing with several healthy leaves having been produced after the affected leaf. Such symptoms were similar to those described in coconut palms (6).

The two other described diseases of Washingtonia palms that are caused by Phytophthora spp. differ from this disease in that one is clearly a trunk rot (2) and the other is a collar rot that completely kills young, container-grown palms (4). Some of the palms inoculated in our studies were the same age as those in the latter study, but only the bud and youngest leaves were killed.

The pathogen was found in samples of nursery soil, but no attempt was made to determine distribution or quantity of the propagules throughout the field. The palms in the perimeter of the field were more open to wind-driven rain and splashing of infected soil particles than those in the center of the field, where the foliar canopy may have provided a moderating influence against movement of soilborne inoculum.

Wound-inoculations of Washingtonia palms conducted by others (4) resulted in 73% diseased plants. The relatively low percentage of diseased plants that resulted from experimental wound-inoculation in our studies suggests that the disease has some specific requirements not always provided in these experiments. Wounds were not essential for infection to occur, but may have aided the infection process. Machete wounds in nursery palms were made by workers removing old, senescent leaves. In one case, a direct link of diseased tissue in petiole bases was found between a machete wound and a diseased bud.

Studies on control of this disease have not been conducted. Preliminary evidence suggests that reducing pruning wounds will reduce disease incidence. Recently available, systemic fungicides specific for pythiaceous fungi offer
potential value for preventing this disease and are currently being evaluated.

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LITERATURE CITED