

Red Ring Disease of Coconut Palm

About 1900, a wilt disease called bud rot of coconut palm (*Cocos nucifera* L.) was causing considerable anxiety in Trinidad, Jamaica, British Honduras, Cuba, and Puerto Rico. The disease caused epidemic destruction of coconut trees in several other countries, also. The government of Trinidad set out to destroy trees with bud rot disease, and during this campaign, in 1905, J. H. Hart, superintendent of the Royal Botanic Gardens, first encountered red ring disease, in the Cedros peninsula. Between 1909 and 1911, the government destroyed 18,068 trees with red ring disease on small holdings throughout the island.

A fungus of the genus *Botryodiplodia* was thought to be the causal agent of the new disease, called root disease to distinguish it from the other two prevalent coconut palm diseases, bud rot and leaf disease. However, another disease of unknown etiology and with different symptoms, known from the Indian state of Travancore, had been called root disease since 1880. In 1919, Nowell (9) named the new disease red ring on the basis of the characteristic red band of tissue always present in the stem of an affected palm.

By 1920, the disease was reported from Honduras, Guyana, Panama, Venezuela, St. Vincent, and Grenada, where Nowell worked. Nowell had determined through Cobb in Panama that the nematode *Rhadinaphelenchus cocophilus* (Cobb 1919) Goodey, 1960 (*Aphelenchus cocophilus*) was the causal agent. Nowell successfully inoculated coconut trees by placing bits of diseased tissue in leaf axils and in holes drilled in the stem or petioles of healthy trees. Nowell concluded that natural infestation began when the nematodes, which he considered to be

soil-inhabiting, crawled up the stem of the tree and entered through cracks. The nematode was believed to be an ectoparasite similar to the rice nematode and the black currant nematode.

Cobb (4) proposed an alternate hypothesis, now supported by experimentation, that implicated the palm weevil (*Rhynchophorus palmarum* L.) as the carrier of the nematode. Apart from the general correlation between the presence of the palm weevil and the disease, about 50% of the adult weevils and larvae Cobb examined carried, either internally or externally, the nematode.

The palm weevil was known in the West Indies before 1827. At that time, the palm weevil was reported as a pest of sugarcane (*Saccharum officinarum* L.) or living in the cabbage palm (*Roystonea oleracea* L.). Other species of the genus *Rhynchophorus* are present in Southeast Asia, India, and Africa, and *R. cruentatus* is found in North America. Only *R. palmarum*, however, is established throughout Latin America and most of the West Indies.

To date, red ring disease has been recorded from the West Indies islands of Tobago, Grenada, and St. Vincent and from the Dominican Republic, Venezuela, Guyana, Surinam, French Guiana, Colombia, Ecuador, Mexico, Brazil, Panama, Costa Rica, Honduras, and El Salvador. In these countries, recorded dates of first observations do not reliably indicate actual times of introduction, which remain unknown.

Symptoms of Red Ring Disease

Coconut palms more than 3 years old are susceptible, especially those 3–10 years old; seedlings are not naturally affected. The major symptom is a band of discolored tissue within the cortex of affected organs, i.e., roots, stems, and petioles (Fig. 1). The ring is almost brick

red in tall cultivars but is usually browner in dwarf and hybrid cultivars. Occasionally, trees more than 20 years old have a solid central cylinder of reddened tissue rather than the characteristic 5-cm wide band in the trunk.

External symptoms are confined to the leaves, which wilt, turn yellow from the tip of the leaflets to the base of the rachis, then turn brown (Fig. 2). Generally, leaf symptoms start on the lowest or oldest leaves close to the point of infection on the stem and progress upward around the tree. Trees between 3 and 10 years of age die within 2 months. Older, heavy bearing trees shed all their fruit as the subtending leaves become infected. Occasionally, the crown of the tree falls off as a result of severe internal damage by the larvae of the palm weevil, which usually tunnel extensively while developing in the diseased crown. Bacterial decay of the heart leaves is not associated with these symptoms, however, nor is necrosis of the inflorescence, which dries out.

The position of the red ring in diseased stems has never been explained plausibly. One suggestion is that aeration and water supply account for the inner limit of the ring and hardness of the tissue accounts for the outer limit. This internal symptom is usually fully developed before external symptoms become visible. First, discrete pink lesions appear in the stem (Fig. 3), eventually coalescing into a red band. The nematodes multiply in these primary lesions (3), then migrate through the intervening parenchyma of the stem and enter the roots and leaves but not the inflorescences and fruit. After invading the roots, some nematodes enter the soil and die within 48 hours.

Histopathology

The red ring nematode is normally confined to tissues containing thin-

walled parenchyma cells in the stems, petioles, and root cortex. The nematodes are found intercellularly throughout the length of the discolored zone; occasionally, some are found within cells. When invaded cells break down, lysigenous cavities form, containing hundreds of nematodes. One gram of infected tissue can contain 10,000 nematodes.

Although the nematodes do not invade the vascular bundles, tyloses occlude the xylem vessels wherever the nematodes have passed through invaded tissue. Viable eggs and adult females are found in small patches of newly invaded tissue showing the tiny pink lesions. Eggs usually occur in batches but sometimes are found singly between cells. The life cycle of the nematode in coconut tissue at 25 C has been recorded as 9–10 days.

Origin of Red Ring Disease

The following findings and hypothesis support a Latin American origin for red ring disease:

1. The disease is present only in Latin America and only in countries where *R. palmarum*, the vector of the nematode, is present.

2. *R. palmarum* is localized in the Western Hemisphere even though other members of the genus are present in

Southeast Asia with enough diversity to indicate Asia as the origin of the genus. Local "red" variants of *R. palmarum* throughout Latin America frequently show the diverse coloration of the several species in Southeast Asia, suggesting that the weevil was introduced to and became isolated in Latin America without bringing in the nematode.

3. The red ring nematode is not known to occur in Southeast Asia, although a similar ectoparasitic species has been reported on areca palm in India. The causal nematode is known to be an obligate parasite on coconut and oil palms in Latin America only.

4. The only ectoparasitic species closely resembling the red ring nematode in Latin America has been reported from Surinam. This species is also called *Rhadinaphelenchus cocophilus* and, when the palm weevil is a pest of the same tree, is associated with little leaf disease of coconut (10). When injected into the trunk, these ectoparasitic forms can cause red ring disease.

5. Evidence strongly indicates that the obligately parasitic *Rhadinaphelenchus cocophilus* is a variant of this ectoparasitic form naturally capable of multiplying in the coconut palm, causing disease, and is carried internally by palm weevils incapable genetically of removing it from the hemocoel. The life cycle of the nematode continues within a coconut palm.

This hypothesis of the origin of red ring disease suggests that the nematode could possibly be transmitted by other members of the genus *Rhynchophorus*. Therefore, quarantine measures are strongly recommended to preclude this form of *Rhadinaphelenchus cocophilus* from entering new countries having other species of *Rhynchophorus* and susceptible palms.

Epidemiology

In addition to the coconut palm, the red ring nematode is an obligate parasite of certain other genera of palm, including the cabbage palm, the date palm (*Phoenix dactylifera* L.), the oil palm (*Elaeis guineensis* Jacq.), and the grugru palm (*Acrocomia aculeata* L.).

Disease in a new or isolated field generally begins with an infected 3- to 10-year-old tree, and the cycle of infection is about 3 months. The likelihood of a particular tree becoming infected varies inversely with the distance from a diseased tree. Where infestation is heavy and healthy trees are not limiting, two trees near each diseased tree usually become infected.

The palm weevil vectors are strong fliers, averaging about 20 ft per second in the field, and may travel up to 1 mile in 24 hours. They generally fly in the understory of the coconut grove but have been observed at an altitude of 200 ft. Their preferred habitat is at the bases of the leaf axils, whether the tree is young or old. The females make several punctures in the soft internodal portion of the crown and oviposit, depositing hundreds of infective juveniles of the red ring nematode in the wounds.

Very often, female weevils are unmated and red ring disease can develop without any overt sign of weevil activity, since no weevil larvae are present in the diseased tree. When fertile weevils are attracted to the diseased tree and oviposit, however, larvae develop and tunnel throughout the stem, causing the crown to break off. Damage depends on the number of insects and the extent of internal injury.

Nematode and Vector Interaction

The red ring nematode is very slender, tapering gradually toward the head and distinctly so from the vulva to the tail. The nematode is about 1 mm long and has faintly visible rings on the body surface. The female narrows to a long, tapering tail with a rounded end, whereas the tail of the male has a pointed tip curving to about four-fifths of a circle (Fig. 4). The red ring nematode is classified in the order *Tylenchida* and the family *Aphelenchoididae*. The genus *Rhadinaphelenchus* was erected by Goodey in 1960, and the nematode was reclassified accordingly when it was removed from the genus *Aphelenchoides*, in which Goodey had placed it instead of *Aphelenchus*, originally given by Cobb in 1919.

The exact nature of the association between the red ring nematode and the palm weevil has been the object of much speculation over the years. In 1921, Ashby, who worked in the West Indies, supported Cobb's view that the palm weevil was the vector (1). In 1953, Martyn suggested that nematodes could be carried by fragments of diseased tissue



Fig. 1. Coconut palm with typical red ring symptoms (A) in the cortical tissues of the trunk and (B) in the trunk and base of the leaves.



Fig. 2. Coconut palm with yellowing and wilt symptoms of red ring disease.

attached to the hairs on the body of the insect. Similar ideas were entertained by Bain and Fedon (2) in Venezuela and Hagley (8) in Trinidad. In 1968, however, I demonstrated that the nematodes on the body surface of the palm weevil, either in frass or on hairs, could not, on their own, initiate infection in a healthy palm tree (6). Thus, nematodes borne on the exterior of the weevil are unimportant to disease initiation or nematode survival.

Survival of the nematode depends on the third-stage juvenile, generally called the preadult larva, which has a conical, rounded head and a mucronate tail. These larvae remain in the decomposed coconut tissue for up to 3 months without developing further. Hundreds are found internally in the weevil vector, either in the proliferated tracheal sacs, from where they move down to the ovipositor, or in the hemocoel among the loops of the gut tract, from where they enter the ovipositor when eggs are being released. This is the infective stage of the nematode.

As wild palm weevil larvae develop in the diseased palm, the majority are able to destroy nematodes entering the hemocoel. This mechanism of active resistance relates to a homozygous recessive condition for an allele that produces an enzyme capable of lysing the invading nematodes in the hemolymph. This mechanism has been shown to cause complete lysis of 360,000 nematodes in living weevil larvae weighing between 9.1 and 12 g at 35 C (7); the mechanism still operates at 2 C. This defense mechanism is absent in about 16% of the wild weevil population, however, and the large quantities of nematodes subsisting within

the hemocoels of such larvae cause a reduction in size of the adults. Such adult females, generally measuring less than 30 mm from the tip of the head to the end of the abdomen (Fig. 5), are the vectors.

Elements Affecting Weevil and Nematode Populations

Several natural events control red ring disease to some extent. The red ring nematode dies in the decomposing tissue of a diseased tree. The larvae of the palm weevil often die when developing in a tree that is attacked by bud rot after contracting red ring disease. Overcrowding of weevil larvae leads to cannibalism, which reduces the number of emerging adults. The bacterium *Micrococcus (agilis) roseus* (Ali-Cohen) associated with Cedros wilt of palm also causes a disease of palm weevils. Finally, some ground lizards feed on the adult insects.

The incidence of red ring disease and the damage caused by only the palm weevil vary. For example, the palm weevil is a major pest in Ecuador, and the adult insects attack healthy trees of any age. The insect is a pest in a habitat providing several other kinds of food, including sugarcane, pineapple, and papaw. Fortunately, the level of the nematode population is low. Such intense weevil attacks without red ring disease are rare in other countries.

Prolonged drought reduces insect attack. Losses to red ring disease are heaviest at the end of the wet season and during the first 2 or 3 months of the dry season, i.e., between December and February.

The palm weevil can be considered the intermediate host to the nematode, whereas the coconut palm, in which the nematode multiplies, is the definitive host. The extent of parasitization of the palm weevil vector by the nematode is critical. When laboratory-reared progeny of vector weevils are allowed to develop in diseased tissue, mortality is always high. As the larvae feed, the number of nematodes accumulating in their body cavities increases. The extent of accumulation and the feeding activities of the nematodes determine their effect

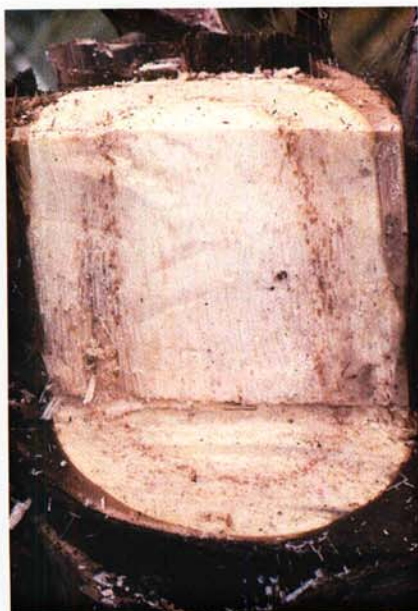


Fig. 3. Primary pink lesions in the stem of a coconut palm infected by *Rhadinaphelenchus cocophilus*. These lesions coalesce to form a red ring.

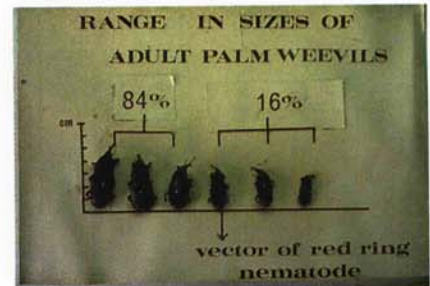
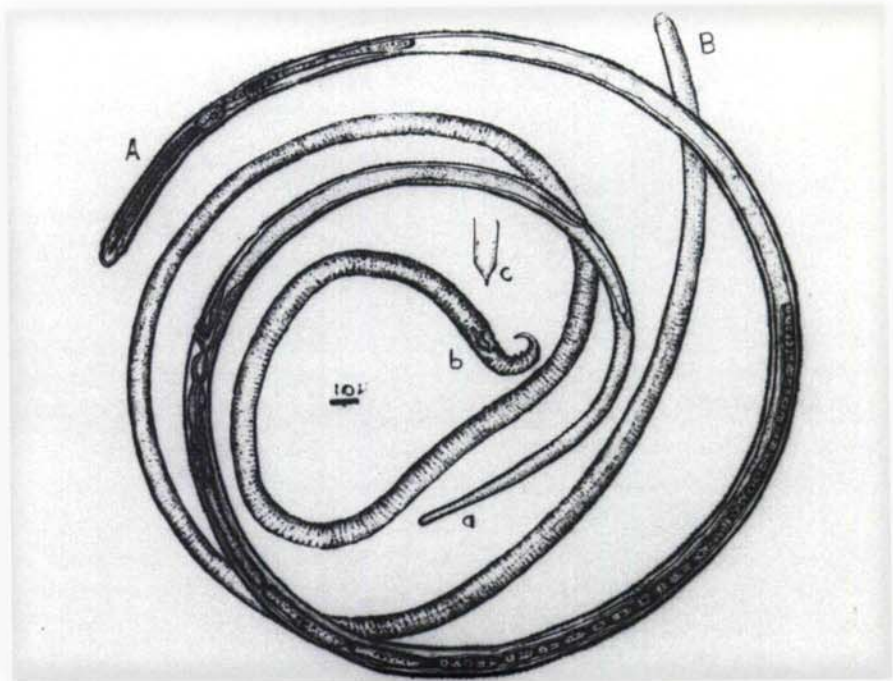


Fig. 5. *Rhynchophorus palmarum*, the palm weevil. Female weevils that vector the red ring nematode are on the right.



Rhadinaphelenchus cocophilus

(Cobb, 1919) J.B. Goodey, 1960.

Syn. *Aphelenchus cocophilus* Cobb, 1919.

Fig. 4. *Rhadinaphelenchus cocophilus*, the red ring nematode.

A Female nematode

a Female tail

B Male nematode

b Male tail

c tail tip of infective stage larva

on the longevity of the weevil larvae and the mortality rate of emerging adults.

The most obvious consequence to the adult insect of hosting large numbers of the nematode is reduced fat body. Normal-sized nonvector palm weevils always have an abundance of fat body; vector insects have almost none. To some degree, this is related to longevity of the adult insect in the field, since adults eat very little. Fecundity is affected, too. Most nonvector females can oviposit between 200 and 400 eggs during 30 days, and many oviposit more than 500. The most frequently recorded quantity for vector insects is 20, and very few oviposit more than 50. Furthermore, many female vectors oviposit unfertilized eggs, since mating might not be accomplished with the larger insects around. Thus, a female weevil, although able to transmit nematodes, may not produce progeny.

Transmission, therefore, depends largely on the attractiveness of the

diseased tree to palm weevils producing fertile eggs with the genetic requirements for susceptibility to the nematode. This replenishment of vectors comes from the homozygous recessive progeny resulting from the heterozygous parents in the population. In diseased estates, the nematode represents a selective force on the palm weevil population. In disease-free estates, this form of pressure is absent. Gene frequency differs correspondingly, and in countries such as Ecuador with low frequencies of the nematode and red ring disease, palm weevil numbers tend to escalate.

The rate of spread of the nematode correlates with the gene frequency of the recessive allele. The intensity of selection against this allele is also important, especially in the small subpopulations that can exist in isolated estates. Efforts to eradicate red ring disease may be aided by determining the rate of gene frequency and altering the recessive allele to make it rare.

Recent Control Measures

The habitat of the palm weevil in the coconut estate is normally the diseased coconut palm to which the insect is attracted for oviposition. Laboratory and field development studies have shown the insect's life cycle in the coconut palm to be about 80 days. Therefore, adult insects begin emerging about 80 days after the tree becomes attractive to other palm weevils. If fertile eggs of vector insects were oviposited with the inoculum of nematodes before the trees become attractive to other insects, vector weevils would emerge before the general population developing in the tree. This has an important bearing on the use of insecticides for control. After many insecticide trials by early workers, Hagley (8) applied insecticides at 50- and 70-day intervals but noted that different timings might have been more effective. Fenwick (5) sprayed at several intervals, including at 2 and 3 months, with no reduction in losses. The reason for the lack of response to these treatments should be clear—the vector insects emerged between sprays and infected healthy trees.

Studies have established that the palm weevil is the only vector of the nematode. Since the nematodes do not multiply in the insect and do not survive for an appreciable time in the dead tree, the only known reservoir of inoculum is the diseased tree in which the vector weevils developed. Starting in 1923, the disease was usually kept at a low level by burning or poisoning diseased trees as soon as symptoms of red ring appeared. In 1959, Webster and Gonzales destroyed both trees and nematodes by injecting a mixture of mevinphos (Phosdrin) and sodium arsenite into infected trees. Blair injected a combination of sodium and potassium arsenite (Weedicide 100) into

diseased trees and obtained the best results by placing 30 ml of the compound into each of two downward-sloping holes 15 cm deep on opposite sides of the stem base; the trees died rapidly and were easier to burn. Fenwick used cacodylic acid (Silvisar 510) to poison the trees. Later, a *cordon sanitaire* was established that included all the farms in a given region when tree poisoning was being practiced over a certain time period. Hague (*World Crops* 1979) believes that a more successful control measure than burning trees is required. In many countries, however, coconut is a small farmer's crop, and removing diseased trees is the most economical and efficacious method of control.

Recently, poisoned baits have been used to control the palm weevil. The adult insect is attracted to such anaerobic fermentation products as ethyl alcohol, to *n*-butyl alcohol, and to certain esters of low molecular weight and high volatility that have been extracted from diseased palm tissue. Since 1970, so-called guard baskets containing chunks of diseased coconut tissue sprayed with 0.1% methomyl (Lannate) that attract and kill palm weevils have been used in coconut estates throughout Latin America.

Further attempts at control will relate to selecting resistant cultivars of coconut and at applying selection pressure against the homozygous recessive vector palm weevil.

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