

Present Status of Research on Root (Wilt) Disease of Coconut

J. J. SOLOMON

*Central Plantation Crops Research Institute,
Regional Station, Kayangulam, Krishnapuram 690533*

Abstract

The root (wilt) disease of coconut has caused an annual loss of over 340 million nuts in Kerala State and has spread to the adjoining Tamil Nadu State. The symptoms and diagnostic tests, etiology and management of the disease are highlighted. Effective quarantine measures are needed to prevent the movement of planting material from disease-prevalent to disease-free areas. Until effective control measures are developed the palms in the affected area should be rejuvenated and the disease contained within its present geographical limits.

I. Introduction

Coconut root (wilt) disease was first reported, following the great floods of 1882, in three isolated pockets about 50 km away from each other in the erstwhile State of Travancore (Butler, 1908; Varghese, 1934). Since then it has spread from the original foci of infection and now occupies a contiguous area covering eight out of the thirteen districts in Kerala. According to a survey concluded in 1972 more than 30% of the 0.5 million ha under coconut in Kerala are affected by the disease causing an annual loss of over 340 million nuts (Bavappa *et al.*, 1982). The disease is non-lethal, but debilitating and palms of all age groups are affected. Recent surveys on the distribution of the disease revealed occurrence of diseased palms in North Kerala far from the core of the diseased tract and also in the adjoining State of Tamil Nadu.

II. Symptoms

The diagnostic symptom of the disease is the characteristic bending of the leaflets termed flaccidity. Yellowing and necrosis are other associated symptoms (Radha and Lal, 1972). As early as 1908 Butler held root rot as a major symptom. Drying up of the spathe and necrosis of spikelets from tip downwards even in

unopened inflorescence was noticed in certain cases. It is worth mentioning here that necrosis of inflorescence is one of the consistent symptoms of lethal yellowing disease in the Caribbean and West Africa.

III. Diagnosis

Two diagnostic tests, one based on positive serological reaction of extracted sap (Solomon *et al.*, 1983a) and another dependent on the differential stomatal resistance (Rajagopal *et al.*, 1984), have been standardised to identify diseased palms even before visual symptoms are manifested.

The sporadic occurrence of the disease and the pattern of spread are more suggestive of the involvement of a pathogen transmitted by biological agents to be the cause of the disease than nutritional or physiological factors. The changes in the physiological parameters in the diseased palms are more indicative of a pathogen mediated host metabolism.

IV. Etiology

i. **Nutritional aspects :** Extensive studies carried out on the nutritional status of coconut palm in relation to the disease tend to rule out the role of any major nutrient in the disease incidence (Cecil, 1975). The effect of micronutrients and heavy metals in the disease syndrome has not been investigated.

ii. **Physiological studies :** Some of the malfunctions encountered are higher transpiration and respiration rates, lower photosynthetic rate and electrolyte release in diseased palms. The accumulation of reducing and non-reducing sugars in leaves as reported is suggestive of impaired translocation normally encountered as a result of infection by vascular pathogens. Accelerated phenol metabolism is yet another feature known for many pathogenic diseases (Mathew and Dwivedi, 1981).

iii. **Mycological studies :** Since Butler's report implicating fungi, possibly *Botryodiplodia* as the probable etiological agent, *Rhizoctonia solani*, *R. bataticola*, *Fusarium equiseti*, *Cylindrocarpon effusum* and *C. lucidum* were isolated from the

roots of wilt affected palms (Lily and Joseph, 1981). These fungi on inoculation to healthy seedlings produced rotting of roots but failed to induce the foliar symptoms characteristic of the disease. These fungi may therefore not have a primary role in the causation of the disease.

iv. **Bacteriological studies:** Srivastava *et al.* (1969) reported bacterial streaming in vascular tissues of roots of diseased palm and isolated *Pseudomonas* sp. However, in subsequent studies an unconventional phytopathogenic bacterium, *Enterobacter cloacae* was isolated from the roots. Culture filtrates of *E. cloacae* induced toxic symptoms on detached coconut leaflets and caused reversible wilting in tomato seedlings (Jayasankar *et al.*, 1981).

v. **Nematological studies:** Implication of a soil bound pathogen or a pathogen transmitted through a soil borne vector warranted nematological studies. Analysis of coconut soils revealed the presence of 35 genera including species of *Xiphinema*, *Longidorus* and *Trichodorus*, known virus vectors, and the burrowing nematode *Radopholus similis* (Mathen *et al.*, 1970). The inconsistent pattern of occurrence and the low population density of these virus transmitters in the diseased tract, when viewed with the pattern of spread of the disease, tend to rule out the role of nematodes in the transmission of the disease. Pathogenicity experiments with *R. similis* on coconut seedlings while establishing the formation of root lesions leading to extensive rotting of roots and stunting of the plants failed to induce the root (wilt) syndrome (Koshy, 1981).

An elaborate pathogenicity experiment with these suspected biological agents - fungi (*Fusarium equiseti*, *Cylindrocarpon effusum*), nematode (*R. similis*) and bacterium (*E. cloacae*), singly and in various combination, is under observation in our Institute.

vi. **Virological aspects:** A viral etiology for the disease was proposed by Nagaraj and Menon (1954) based on the systemic nature of the disease and resemblance of the disease symptoms to other known plant virus diseases. Positive transmission of the

disease on the main host through sap and an insect *Stephanitis typicus* under field and green house conditions implied a sap transmissible insect-carried pathogen, probably a virus. Based on studies at the IARI, New Delhi, Summanwar *et al.* (1969) reported the isolation of a rod-shaped virus. This organism from the leaves and roots of infected palms was described as TMV coconut isolate. These results could not be corroborated and the association of the TMV coconut isolate with the root (wilt) disease has been ruled out through detailed serological studies (Solomon and Sasikala, 1980).

Maramorosch and Kondo (1977) reported the presence of icosahedral virus-like particles in the epidermis and ground parenchyma cells based on the electron microscopy of a diseased palm. These structures were subsequently identified by Parthasarathy (1978) as section of plasmodesmata in tangential plane. Electron microscopic examination of semi purified preparations fractionated by different methods of various plant parts also failed to reveal typical viral particles. Long tubular particles of host origin were detected in preparations of both healthy and diseased palms. They were identified as microtubules - a normal host component not associated with any disease (Randles, 1975). Possibility of a viroid type pathogen with the disease was investigated by me in collaboration with J. W. Randles of Walte Agricultural Research Institute, Australia. Polyacrylamide gel electrophoresis of isolated nucleic acid from limited number of diseased palms also failed to reveal any free nucleic acid akin to that of viroids.

vii. **Mycoplasma - like organisms** : Since 1981 attempts were made to examine ultrathin sections of tissues from different parts of healthy and diseased palms. Emphasis was laid on the EM examination of vascular tissues as the light microscopic studies conducted earlier revealed degenerative changes including chromophily and necrotic obliteration in the phloem tissues of diseased palms (Govindankutty, 1981). EM examination of ultrathin sections from the apical meristem, root tips, rachillae from unopened inflorescence, petiole and tender leaves revealed typical mycoplasma-like organisms (MLOs) in sieve tubes of diseased palms. Such organisms were conspicuously absent in developing tissues of

healthy palms. MLOs were confined to sieve tubes and found in greater concentrations in the sink areas (Solomon *et al.*, 1983b). Moribund forms of the organism were observed in mature tissues. The number and distribution of the organisms varied from palm to palm and also between sieve tubes in different bundles. Such unequal distribution of MLOs were also reported in the case of the lethal yellowing disease in Florida (Thomas 1979). The size of the ovoid bodies of MLO observed in root (wilt) diseased tissue ranged between 250 - 300 nm. They have a triple layered unit membrane with two electron dense layers and translucent one in between, limiting the DNA strands and peripherally distributed ribosomes. The cell walls of the invaded cells and the bordering ones were thickened, cytoplasm granulated and often contained vesicle like structures. True to characteristic pleomorphism they assumed various forms - coccoid, oval to filamentous forms. Interestingly, no other biological agent reported earlier to be associated with the disease were encountered in the developing tissues examined. No viral particles were either observed in the tissues.

Intracellular presence of MLOs, their restriction to the phloem tissues together with the abnormalities encountered in the food conducting channel and the total absence of MLOs in healthy palms favour their role in the etiology of the coconut root (wilt) disease. This theory gains additional impetus from the results of a preliminary experiment conducted a few years ago with oxytetracycline hydrochloride. Application of Terramycin though did not offer any ameliorating effect prevented the treated palms from further decline, whereas the untreated palms deteriorated considerably (Jayasankar *et al.*, 1981).

Based on these findings the root (wilt) disease research at the CPCRI has been given a new direction to study in depth various aspects concerning the role of MLOs in coconut root (wilt) disease.

Constant association of MLOs with the root (wilt) disease has been established beyond doubt with the examination of tissues from 26 diseased and six healthy coconut palms. In all the diseased palms the organisms are present in various proportions and were

totally absent in tissues of healthy palms. Although electron microscopy is the reliable technique for detection of MLOs in tissues it is laborious and time consuming. Hence certain light microscopic methods which have specificity to detect nucleic acid accumulation in short wave ultraviolet fluorescence and a staining method with specificity to detect callose deposition have been developed (Govindankutty, unpublished). The specificity of the tests is being verified with tissues of plants with established mycoplasma etiology.

Plant mycoplasmas are transmitted by phloem feeding insects generally by leaf hoppers and in a few cases by psyllids. In the earlier studies successful transmission of root (wilt) disease was recorded through the lace bug, *Stephanitis typicus*. EM examination of field collected lace bugs subjected to 5 days acquisition and 13 days incubation period on diseased palms revealed certain structures resembling mycoplasmas in the brain and salivary glands. Further study is in progress to establish the vector role of the lace bug. An inventory of insect visitors of coconut gardens is being made through various trapping aids - sticky, rotary suction and light traps, sweep net collections and by direct examination of coconut foliage. This study has brought to light two new records. One a planthopper - *Proutista moesta* and a leaf hopper - *Sophonia greeni* (Rajan and Mathan, 1984 a, b). These records are of great significance as these insects belong to the group which feeds on phloem and transmits phloem seated pathogens. The vector role of these hoppers is also under experimentation.

V. Management of the Disease

A varietal screening programme to evaluate the yield potential and resistance/tolerance to root (wilt) disease has been in progress since 1972 with 92 cultivars/hybrids. The results though not conclusive are not encouraging. A comparative assessment of the performance of West Coast Tall and Dwarf x Tall palms under rainfed and good management conditions in the diseased area showed that DXT hybrids are more productive and have lower incidence of disease compared to WCT (Bavappa *et al.*, 1982).

Field trials using different fungicides and nematicides singly and in combination did not have any ameliorating effect on the

treated palms. Beneficial effect of ascorbic acid and growth promoting substances in masking the symptoms and improving the yield of affected palms has been speculated.

Based on the results obtained on the various aspects of the disease separate strategies for the diseased area and for the border areas have been formulated. The strategy for the diseased area is to "live with the disease" recommending removal of all badly affected uneconomically yielding palms (wherein the input - output cost benefit ratio is one or less), replanting with hybrids and adopting better management practices. Eradication of disease affected palms and sanitation in the farm have been held as beneficial in other diseases of the 'yellows' type also. Experiments have shown that the diseased palms respond to better management like organic recycling, addition of plant materials, etc. A mixed farming experiment with fodder crops as intercrops, maintenance of milch cows and recycling of organic waste in a disease affected garden under rainfed conditions over a period of five years has shown reduction in yellowing and increase in yield of nuts by about 26% (Bavappa *et al.*, 1982). Similarly, mixed cropping with cacao has shown an overall increase in yield of diseased palms by 30% over a period of five years. Regular addition of $MgSO_4$ complimented with normal dose of NPK significantly increased the nut yield and arrested development of foliar symptoms.

The strategy for the border area is "to contain the disease". Experimental evidences indicate that removal of isolated diseased palms in the border areas, where the disease incidence is mild can prevent recurrence of the disease. Based on these findings a programme was launched by the CPCRI Field Station, Trichur to see whether the spread of the disease north of Karuvannur river could be checked. Accordingly, 400 diseased palms from 213 gardens distributed in eight villages situated along the northern basin of Karuvannur river were removed. The boles were dug out, and the leaves and roots were burnt *in situ*. The surrounding palms in 15 m² area (1409 palms) were treated with aldicarb and carbendazin. Of this only fourteen developed disease symptoms in 1981 and 22 palms in 1982, while among the untreated palms 114

manifested symptoms in 1981 and 42 palms may help to reduce further spread of the disease.

In recent surveys isolated pockets of disease have also been located in some of the northern districts of Kerala - Malappuram, Kozhikode and Cannanore and a few palms in Coimbatore in Tamil Nadu (Bavappa *et al.*, 1982). Information collected from the cultivators on the source of planting material revealed that atleast in some cases the planting materials - seedlings have been brought from the root (wilt) affected areas. This highlights the need for effective quarantine measures to prevent the movement of planting material from disease - prevalent to disease - free areas. Until such time when effective control measures are known the alternative is to rejuvenate the palms in the diseased area and to contain the disease within the present geographical limits.

References

- Bavappa, K. V. A., G. B. Pillai and K. Mathan. (Eds.) 1982. Coconut root (wilt) disease A practical approach. In: "*Contain the disease and live with it*". Tech. Bull. 8. Central Plantation Crops Research Institute, Kasaragod, 5 pp.
- Butler, E. J. 1908. *Agric. Res. Inst. Pusa Bull.* No. 9, 1-23.
- Cecil, S. R. 1975. *J. Plant. Crops*, 3, 34-37.
- Govindankutty, M. P. 1931. pp. *Ibid.*, 49-54.
- Jayasankar, N. P., K. V. Joseph and M. George. 1931. *Ibid.*, pp. 25-32.
- Koshy, P. K. 1931. *Ibid.*, pp. 33-48.
- Lily, V. G. and T. Joseph. 1981. *Ibid.*, pp. 20-24.
- Maramorosch, K. and F. Kondo. 1977. *J. Plant. Crops*, 5, 20-22.
- Mathen, K., M. P. Govindankutty, T. Joseph and C. Mathew. 1981. In: *Review of research on coconut root (wilt) disease*. CPCRI, Kasaragod (Mimeo).
- , C. Kurian and S. B. Lal. 1970. *Sci. Cult.*, 36, 159.
- Mathew, C. and R. S. Dwivedi. 1981. In: *Review of research on coconut root (wilt) disease*. CPCRI, Kasaragod, pp. 55-67.
- Nagaraj, A. N. and K. P. V. Menon. 1956. *Indian Cocon. J.*, 9, 161-165.
- Parthasarathy, M. V. 1978. *J. Plant. Crops*, 6 87-89.