

# Root (Wilt) Disease of Coconut

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## 1. INTRODUCTION

In a worldwide survey organised under the aegis of the Food and Agriculture Organisation of the United Nations, Maramorosch (1964) listed 11 coconut diseases of unknown etiology. It also included the 'Kerala Wilt' popularly known as 'Kattuveezhcha' in the local language and is referred to as root (wilt) disease by researchers. This disease was first reported after the great floods in 1882 in three independent locations each about 50 km apart in the erstwhile State of Travancore (Varghese, 1934). Since then, it has spread from the original foci of infection and now occupies a contiguous area covering eight out of the fourteen districts of Kerala, besides in few isolated pockets in the northern districts of the state and the bordering districts of Tamil Nadu. The disease intensity in the contiguous diseased tract ranges from 1.52 to 75.63 per cent. The annual loss due to the disease is estimated to be about 968 million nuts (Anon., 1985a). The disease is non-lethal but debilitating and palms of all age groups are affected. Palms contracting the disease in the prebearing age remain unproductive (Ramadasan *et al.*, 1971).

Pillai *et al.* (1973) observed the disease incidence in all major soil types in Kerala state. The spread is faster in sandy and sandy loam soils of the plains and in clayey alluvial soils of the riverbed than in the laterites. Higher disease incidence is also observed in water logged low lying areas adjacent to rivers and canals.

## 2. SYMPTOMS

### 2.1 External Symptoms

The most consistent and diagnostic symptom of the disease is the conspicuous bending of the middle and outer whorls of leaves and the characteristic ribbing of leaflets termed as 'flaccidity' (Fig. 1) (Radha and Lal, 1972). Foliar yellowing and marginal necrosis are invariably observed in adult diseased palms (Menon and Pandalai, 1958) (Fig. 2). Yellowing of foliage is virtually absent in juvenile diseased palms and in seedlings, where flaccidity is often the only symptom (Radha and Lal, 1972). George and Radha (1973) developed a scoring system for quantifying the disease giving due weightage to intensity and frequency of occurrence of flaccidity, yellowing and necrosis. As early



**Fig. 1 :** Young coconut palm affected with root wilt disease showing flaccidity and yellowing.



**Fig. 2 :** Old coconut palm with middle stage of root wilt disease.



**Fig. 3 :** Old coconut palm with advanced stage of root wilt disease.

as 1908, Butler held root rot as a major symptom. However, other workers could not corroborate this view (Nagaraj and Menon, 1956; Joseph and Jayasankar, 1981). Michael (1964) observed that the capacity to regenerate new roots is drastically reduced with the progress of the disease.

Drying up of the spathe and necrosis of spikelets from tip downwards even in unopened inflorescences was noticed in certain cases (Menon and Pandalai, 1958; Maramorosch, 1964). A high percentage of pollen produced was either sterile or with low viability (Varkey and Davis, 1960). Meiotic irregularities were also observed in diseased palms (Nambiar and Prasannakumari, 1964). Gradual reduction in yield of nuts up to 80 per cent in advanced stages was reported by Radha *et al.* (1962). The nuts in the diseased palms have thinner husk and the fibres are definitely weaker and less firm (Varghese, 1934). Abnormal shedding of female flowers and immature nuts and lack of ability to produce female flowers render the palm unproductive (Varghese 1934; Menon and Pandalai, 1958).

## 2.2 Internal Symptoms

Anatomical studies on the tender unopened leaves of healthy and root (wilt) affected palms revealed general stunting of epidermal cells, reduction in thickness of the cuticle on the adaxial surface and differential rate of division of the upper epidermis in the longitudinal and transverse plains in leaves of diseased palms resulting in slight downward curling of leaflets. Similarly, reduction in wall thickness of cells, sclerenchymatous fibres and bundle sheath was also observed in diseased palms. The distribution of stomata per unit area was found to be more in leaflets of diseased palms (Joseph and Shanta, 1963).

Internal browning of root vascular elements extending up to the cortex and at times accompanied by disintegration of vascular tissues in about 60 per cent of the roots of diseased palms was also observed (Indira and Ramadasan, 1968). However, Dwivedi *et al.* (1978) using an antioxidant in primary fixation of tissues failed to demonstrate vascular browning in externally healthy roots of disease affected palms. Reduction in thickness of the walls of the metaxylem elements resulting in uneven shape of the xylem component, development of tyloses, increased chromophily and necrotic obliteration of phloem are also observed in roots of diseased palms (Govindankutty, 1981).

## 2.3 Diagnosis

Field identification of diseased palms is mainly based on visual symptoms, considering flaccidity as the primary symptom and yellowing and necrosis as other associated symptoms. Since reliable identification of diseased palms becomes difficult under certain conditions, the need for a diagnostic test which could detect the disease status even before the foliar symptoms are evident was felt necessary. A sero-diagnostic test (Solomon *et al.*, 1983) and a physiological test based on stomatal resistance (Rajagopal *et al.*, 1986) have been standardised for detecting the disease. With these

aids, the disease condition could be diagnosed 6 to 20 months before the expression of foliar symptoms (Rajagopal *et al.*, 1988).

### 3. ETIOLOGY

The sporadic occurrence and spreading nature of the disease suggested the involvement of a pathogen.

#### 3.1 Fungi

Bourdillon in 1906 first reported that the disease is 'fungoid' in nature (Varghese, 1934). Butler (1908) on isolation of *Botryodiplodia* spp. from the roots of diseased palms suggested that root rot alone could be sufficient to cause the disease syndrome. Further studies resulted in the isolation of two species of *Rhizoctonia solani* Lutu and *R. bataticola* (Taub.) Butl. besides *Botryodiplodia theobromae* Pat. (Menon and Nair, 1949) from the rotting root system of diseased palms. Pathogenicity experiments with *R. solani* and *R. bataticola* in the field as well as under pot culture conditions, although brought about rotting of roots, yet failed to produce the foliar symptoms characteristic of the disease (Menon and Nair, 1951). In subsequent investigations, *Cylindrocarpon effusum* Bugn. and *Fusarium equiseti* (Corda) Sacc. were isolated from apparently healthy roots of diseased palms (Joseph, 1978). *C. effusum* and *C. lucidum* were also isolated from burrowing nematode lesions of the roots (Sosamma and Koshy, 1978). However, pathogenicity experiments with *C. effusum* and *F. equiseti* singly and in combination on coconut seedlings grown in individual field tanks failed to reproduce the symptoms of the disease (Anon., 1985b).

#### 3.2 Bacteria

Menon and Nair (1951) reported the isolation of two bacteria from roots of diseased palms. Srivastava *et al.* (1969) observed characteristic streaming movement of bacteria in the vascular tissues of roots of diseased palms and isolated a bacterium tentatively identified as *Pseudomonas* sp. Employing an enriched medium, George *et al.* (1976) isolated a bacterium identified as *Enterobacter cloacae* (Jordan) Hormaeche and Edwards from the stelar portions of root tips of diseased palms. However, inoculation of coconut seedlings with *E. cloacae* failed to produce the symptoms of the disease thereby ruling out its involvement in the incidence of the disease.

#### 3.3 Nematodes

Possible involvement of a soil-borne pathogen or a pathogen transmitted through soil-borne vectors warranted nematological studies. Investigations on the nematodes present in coconut roots and soil around resulted in the identification of 35 genera of phytonematodes including species of *Xiphinema*, *Longidorus* and *Trichodorus* known virus vectors and the burrowing nematode *Radopholus similis* (Weischer, 1967; Mathen, 1969; Mathen *et al.*, 1970; Khan *et al.*, 1971). Weischer (1967) opined that low population density of nematodes, their widespread occurrence and the general distribution pattern

of the disease could exclude the nematodes from being considered as primary cause of the disease.

Considering the notoriety of the burrowing nematode, *Radopholus similis* as having caused great havoc in citrus and banana in other countries, its role in the etiology of the disease was assessed in a pathogenicity experiment. Coconut seedlings in field tanks inoculated with up to one million *R. similis* failed to reproduce root (wilt) symptoms even after four years of experimentation. Extensive surveys covering root (wilt) diseased and disease-free areas also could not bring out a correlation between its presence and occurrence of root (wilt) disease (Anon., 1988).

### 3.4 Physiology

Earlier investigations attributed the disease to certain physiological factors, either the lack of nutrients, excess or inadequate water, disturbing the physiological process of the palms and thus predisposing to the disease. Varghese (1934), after carefully examining the various factors associated with the disease, had concluded that the disease is not physiological but fungi, virus and certain toxic substances produced by micro-organisms living around the root zone of the palms might contribute to the disease.

Derangements in the water absorbing capacity of roots in diseased palms (Anon., 1970) so also the uptake and transport of water through the trunk (Ramadasan, 1964) and permeability changes in leaf and root tissues resulting in leaching of metabolites (Anon., 1977) are noticed. It has also been found that the mechanism of stomatal regulation is adversely affected in diseased palms. The stomata in diseased palms failed to close in response to soil and atmospheric drought resulting in excessive water loss (Rajagopal *et al.*, 1986). Thus, an impairment caused to both the facets of water transport, namely, absorption and transpiration lead to internal water stress.

A higher respiration rate (Michael, 1978), derangement in translocation and distribution of sugars (Mathew, 1977), an altered nitrogen metabolism (Varkey *et al.*, 1969) with an increase in amino acid concentration in leaves particularly of arginine (Pillai and Shanta, 1965), an accelerated phenol metabolism with a fall in phenol content and an increase in phenol oxidising and synthesising enzymes (Joseph and Jayasankar, 1973, 1979; Joseph *et al.*, 1976; Joseph, 1983) are the other changes observed. Such malfunctions encountered in diseased palms are rather suggestive of a pathogen mediated altered host metabolism than of a physiological disorder.

### 3.5 Soil and Nutritional Aspects

The impact of soil conditions and associated nutritional factors on the disease incidence has been on investigation since 1939. Based on a survey and study of the disease in relation to the soil conditions in the erstwhile state of Travancore, it was concluded that the disease affected soils are generally poor in major nutrients especially potash and the soils are mostly acidic with less content of exchangeable bases and

poor base exchanging capacity (Menon and Nair, 1949; Sankarasubramoney *et al.*, 1954, 1955; 1956; Pandalai *et al.*, 1958a,b). An accumulation of major nutrients and silica in the leaves of diseased palms was also noticed. However, no perceptible difference in micronutrient level was evident. Cadmium and strontium toxicity was also ruled out (Verghese *et al.*, 1962a,b).

Detailed analysis of soil and tissue samples from healthy and diseased tract of all the coconut growing area in Kerala has ruled out the possibility of the direct involvement of any major nutrients in the incidence of the disease (Pillai *et al.*, 1975; Pillai, 1975). Cecil (1975) observed the Ca and Mg contents of healthy palms significantly higher than those of apparently healthy and diseased palms. He also observed that palms in the root (wilt) affected areas are in a state of unbalanced nutrition as compared to healthy palms. However, fertiliser field trials with the application of different levels of major and secondary nutrients, Ca and Mg could neither control the disease nor prevent fresh incidence although a general improvement in yield was observed (Menon and Nair, 1952b; Chettiar *et al.*, 1959; John *et al.*, 1959; Nair and Radha, 1962; Lal, 1966). The role of micronutrients in the incidence of disease has also been ruled out (Davis and Pillai, 1966).

#### 4. SUB-MICROSCOPIC AGENTS

##### 4.1 Virus or Virus-like Organisms

Nagaraj *et al.* (1954), based on the systemic nature and resemblance of symptoms to other known plant virus diseases proposed a viral etiology for the disease. This theory gained further significance with positive transmission of the disease to coconut by sap inoculation and through the insect vector *Stephanitis typica* under field condition (Nagaraj and Menon, 1956) and under insect-proof condition (Shanta *et al.*, 1964).

Cotyledonary leaves of cowpea plants mechanically inoculated with sap of diseased palms produced malformation and crinkling in emerging trifoliolate leaves. Hence this was claimed as an indicator host for the presumed 'virus' (Shanta and Menon, 1960). The physical properties of the 'virus' was worked out employing cowpea as the test plant (Shanta and Menon, 1961). In view of the peculiar nature of symptoms on cowpea and lack of proof on its passage through bacterial filters, Holmes (1965) suggested that although the pathogen resembled a virus, it might as well be a related organism like a spirochaete or sporozoa. The symptoms on cowpea was found to be very inconsistent in subsequent studies. After detailed investigation, it was concluded that cowpea cannot be used as a reliable test plant (Sasikala and Pillai, 1978).

Summanwar *et al.* (1969) reported the isolation of a virus from roots and leaves of diseased coconut palm. Based on positive serological reaction to three different strains of TMV antiserum, it was identified as a strain of TMV. However, Shanta *et al.* (1975), through detailed electron microscopic, pathogenicity studies and Solomon and Sasikala

(1980) through serological studies, have ruled out the association of TMV with root (wilt) disease of coconut.

Maramorosch and Konda (1977) reported the presence of icosahedral virus-like particle of 56 nm diameter in epidermal and ground parenchyma tissues of leaves of a diseased palm. However, these structures were later identified as plasmodesmata sectioned in oblique or tangential plane (Parthasarathy, 1978). Polyacrylamide gel electrophoretic analysis of isolated nucleic acids from diseased palms did not indicate association of viroid either (Randles and Hatta, 1980).

## 4.2 Mycoplasma-like Organisms (MLO)

### 4.2.1 Microscopy

Solomon *et al.* (1983) observed the presence of mycoplasma-like organisms (MLOs) in sieve tubes of juvenile tissues such as sub-meristem, petiole of developing leaves, rachilla of unopened inflorescence and root apices of diseased palms. Tissues from healthy palms are free of such organisms. Interestingly, MLOs have been found associated with a number of coconut diseases around the globe—lethal yellowing in Caribbean (Plavsic-Banjac *et al.*, 1972). Kaincope and Cape St. Paul wilt disease in West Africa (Dabek *et al.* 1976) and coconut stem necrosis in Malayasia and Sumatra (Turner *et al.*, 1978). Although these diseases differ from root (wilt) disease in symptoms, resistance or tolerance to varieties and mode of spread, they all seem to share the same group of pathogen.

The mollicutes are found in increasing numbers in the sieve tubes in the sink region. Pleomorphic forms ranging from circular to oval and occasionally beaded or filamentous forms are also observed. The coccoid forms are in the size range of 200-450 nm. Generally, they occupy parietal position close to the sieve area. The organisms are bound by a triple layered unit membrane and have DNA strands and peripherally distributed ribosomes. Constant association of the organism with the disease has since been established with the finding of MLOs in tissues of all the seventy diseased palms examined and their absence in tissues of forty healthy palms studied.

Histological staining techniques for detection of MLOs in plant tissues under light microscope also have been standardised. Abnormal bluish colouration in sieve tubes following Dienes' staining and specific fluorescing sites in sieve area consequent to staining with DAPI (4'6-diamidino-2 phenylindole HCl) are observed (Solomon *et al.*, 1987). These staining reactions, indicative of the accumulation of DNA in extra nuclear sites, evidenced the presence of MLOs. Such specific reaction are not observed in tissues of healthy palms. Positive staining reactions are more frequent in junctions of vascular bridges in rachilla where the organisms appear to congregate. The occurrence of the reactions at scattered loci suggests non-uniform distribution of MLOs. This is corroborated in EM observations too.

## 5. TRANSMISSION

### 5.1 Insect transmission

Elucidation of the constant association of MLOs with the disease warranted identification of the insect that transmits the disease in nature. Lacebug, *Stephanitis typica* (Distant) being the single major group of insects on coconut was suspected as a vector of the disease (Shanta and Menon, 1959). Transmission experiments with lace bug on adult palms in the open field (Nagaraj and Menon, 1956) and in seedlings under insect proof condition (Shanta *et al.*, 1964; Mathen *et al.*, 1976) further corroborated its vector role. The report on the association of MLOs with the disease implied a reinvestigation on the vector role of lace bug, as true bugs (heteropteran insects) are not known to be mycoplasma transmitters.

A systematic inventory of all insect visitors to coconut gardens made using various traps and confirmation of their occurrence in coconut foliage by direct examination over a period of two years lead to the identification of a leaf hopper *Sophonia greeni* (Distant) and a plant hopper *Proutista moesta* (Westood) besides lace bug (Rajan and Mathen, 1984, 1985). Survey of representative coconut gardens in the eight southern districts of Kerala where the disease occurs in a contiguous manner and in gardens in northern Kerala where isolated instances of disease are known to occur, brought out the presence of all the three vector candidates. In other words, there is no disease incidence independent of the presence of these insects.

The potential of these putative insects to acquire MLOs while feeding on foliage of diseased palms was studied. MLOs are observed in lace bug subjected to an acquisition and incubation period of 18-23 days and not in insects offered acquisition and incubation periods less than 18 days. These pleomorphic bodies appear similar to those observed in sieve tubes of diseased coconut in structure and morphology. They are confined to the acini of the salivary glands of the infective lace bugs (Mathen *et al.*, 1987).

In the light of the detection of MLOs in tissues of disease affected palms and infective lace bugs, the insect transmission experiment was repeated under insect proof condition with improved techniques. Two years old West Coast Tall coconut seedlings planted in methyl bromide fumigated soil held in field tanks and protected with insect proof cages were inoculated with field collected and infective lace bugs. Nine months after the first inoculation, positive serological reaction was observed in three of the four inoculated seedlings and weak reaction in the fourth indicating the disease contraction. Light microscopy of root tissues subjected to Dienes' staining, DAPI and Hoechst 33258 fluorochromes also indicated MLO infection. Electronmicroscopic observation confirmed the presence of MLOs in all the four lace bug inoculated seedlings between nine to twenty seven months after the first inoculation. Two of the seedlings exhibited flaccidity of leaflets, the decisive and diagnostic symptom of the disease by the seventeenth month (Mathen *et al.*, 1990).

Apart from the direct evidences emerging out of the transmission experiment, a number of direct evidences have also accrued based on studies on pattern of distribution, population and feeding habit of the insect. The lace bugs are found colonising in increasing numbers towards the inner leaves of the crown (Mathen *et al.*, 1969). This pattern of occurrence enhances the chances of acquisition of the organism which is found in higher numbers and in active forms in the juvenile tissues. It is also observed that the number of lace bugs in diseased palms are four times the number in healthy palms (Mathen, 1982). Monitoring the lace bug population for two years, Mathen (1985) reported a direct linear correlation between the number of insects colonising the palms and fresh incidence of disease. Transections of coconut pinnae with lace bugs fixed in feeding position by a cold immobilisation technique revealed the termination of stylet in phloem, thereby, indicating the ability to feed on phloem and to acquire the phloem bound mollicute (Mathen *et al.*, 1988). These direct and indirect evidences unequivocally establishes the role of lace bug as the vector of the disease.

## 5.2 Dodder Transmission

Experimental transmission of the disease to an accepted mycoplasmal indicator host, *Catharanthus roseus*, was also achieved through the dodder laurel *Cassytha filiformis*. Periwinkle plants grown in nylon netted cages bridged to diseased coconut seedlings through the dodder laurel exhibited chlorotic spots in the interveinal areas and at vein endings of fully opened leaves. Ultrathin sections of the tissues of diseased source palm, connecting dodder laurel and periwinkle showed presence of MLOs in all the three samples. MLOs also could be serially transmitted from periwinkle to periwinkle (Sasikala *et al.*, 1988).

## 6. CULTURING

Attempts to culture the organism in cell-free media have not so far been fruitful. More than forty different media with various combination of growth factors, vitamins, nucleic acid precursors, nutrients, sera and vascular sap enriched preparations were tried (Anon., 1989a). None of the media could either ensure long-term maintenance or support growth. Possibilities of cultivating the organism in plant and insect tissues are being explored.

## 7. CHEMOTHERAPY

Since MLOs are not amenable to cultivation, *in vitro* differential chemotherapy is widely advocated as a tool to establish the mycoplasmal etiology of a disease. To ensure that the antibiotic reaches the target site in unaltered state within a reasonable period, a pneumatic pressure injector was devised (Pillai and Raju, 1985). Residue analysis of the antibiotic through bioassay revealed its presence in the foliage within 24 h of application and its retention for more than 12 weeks. However, the concentration of the antibiotic petered out to a minimum with the passage of time.

A field trial was initiated with four concentrations of oxytetracycline hydrochloride (OTC; terramycin tree formulation of M/s. Pfizer India Ltd.), a single concentration each of neomycin, penicillin and distilled water control. Fifty three per cent of palms treated with 3 and 6 g a.i. of OTC showed remission of symptoms. Contrastingly, palms in the distilled water and penicillin treatment deteriorated over their pretreatment condition (Anon., 1989a). Although application of OTC cannot be recommended either for the field control of the disease or as prophylactic measure as recommended in Florida due to possible environmental hazards, the results nevertheless give additional evidence to establish the mycoplasmal etiology of the disease.

Leaf rot disease caused by *Bipolaris halodes* occurs superimposed on about 30 per cent of root (wilt) affected palms (Anon., 1989b). This affects the vigour of the palms bringing about decline in yield. This disease can be controlled effectively by a sequential spraying with 1 per cent Bordeaux mixture, 0.3 per cent Dithane M-45 and 0.5 per cent fytolan in this order at quarterly intervals after removing and destroying all the severely affected leaves (Bavappa, 1986).

## 8. SCREENING OF COCONUT GERMLASM

Field evaluation of 45 cultivars and 62 hybrid combination is in progress in the institute farm and cultivators gardens since 1972 to locate genotypes resistant/tolerant to root (wilt) disease. None of the cultivars/hybrids showed the desired level of resistance/tolerance to the disease. However, the hybrid Chowghat Orange Dwarf (COD) × West Coast Tall (WCT) under ideal management yielded higher number of nuts compared to WCT palms of identical age in the early years of production (Bavappa *et al.*, 1986). The percentage of disease incidence was also comparatively less than the Talls. Seed nut collection made from other coconut growing countries are raised in an offshore quarantine station in the Andamans so that the second generation of plants could be screened against the disease in the heavily diseased area.

In the programme for breeding for resistance/tolerance to root(wilt) disease, healthy high yielding palms identified in the hot spot areas are also being used to evolve ideal plant type.

## 9. DISEASE MANAGEMENT

One of the significant features about the disease is that it is not lethal but a declining malady which responds to good management. Yield of palms can be sustained or even improved through the adoption of integrated management practices consisting of balanced fertiliser application, addition of organic matter, raising green manure crops in the basin and incorporation, irrigation during summer months, leaf rot control and adopting inter and mixed cropping (Bavappa *et al.*, 1986).

Mixed farming in diseased gardens involving the raising of fodder crops in the interspaces and maintaining milch cows and recycling of organic wastes has helped in increasing the mean yield of palms by 26 per cent (Sahasranaman *et al.*, 1983).

Mixed cropping of coconut with cocoa increased the yield by 50 per cent and slowed down the decline of diseased palms (Bavappa *et al.*, 1982). Similarly, cultivation of tapioca, elephant foot yam and yam in the interspaces of palms in disease affected gardens for a period of three years increased the nut yield by 4.96, 15.57 and 8.07 per cent respectively (Menon and Nair, 1978). Irrigation of palms during summer months is also found beneficial in improving both the health and yield of palms (Rajagopal *et al.*, 1987).

Eradication of disease affected palms in the mildly affected areas help in arresting further spread of the disease. Removal of disease affected palms at two isolated pockets, one at Shencottah (Tamil Nadu) and another at Nadathara (Kerala), prevented recurrence of disease (Radha *et al.*, 1985). Encouraged by the results, a Field Station was established at Trichur district in 1979 to arrest the northward march of the disease and to contain it within the present geographical limits (Radha *et al.*, 1981). With the eradication of 400 diseased palms from 209 gardens in eight villages in Trichur district, 92.4 per cent of the gardens were free of disease even after four years.

The strategy for managing the disease is to eradicate all diseased palms in areas of sporadic occurrence, removal of disease advanced and diseased palms in the early age of bearing in the heavily diseased tract, replanting with high yielding hybrids or seedlings from prepotent palms and adoption of the recommended package of practices.

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