

COCONUT ROOT (WILT) DISEASE - AN UPDATE

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INTRODUCTION

Coconut root (wilt) disease was reported back in 1874 from Erattupetta, of Meenachil taluk in Kottayam district. Butler called it 'root rotting disease' probably due to the rotting of roots observed in the affected palms. In 1955 the term 'wilt' was introduced, befitting foliar symptoms and at present it is called 'root (wilt) disease'.^{3,4} Though the disease was first reported from Erattupetta, it originated from three isolated loci in Kerala viz; Karunagapally, Kaviyoor and Kallloopara. The disease is believed to have manifested significantly since the great floods of 1882.⁵ In the course of time, the disease was reported to have spread along the banks of Meenachil, Manimala, Pamba and Kallada rivers.⁶ High incidence was recorded in and around Kayamkulam, Alleppey, Tiruvalla, Mattancherry & Ernakulam. A contiguous distribution of the disease in six districts viz, Trivandrum, Quilon, Alleppey, Kottayam, Ernakulam and Trichur of Kerala state were reported initially.⁷ Sporadic occurrence of the malady was also recorded in the districts of Tirunelveli and Kanyakumari in Tamil Nadu.⁸

Root (wilt) disease is not lethal but causes gradual decline in the yield with time. In 1983, Dr. M.S. Swaminathan termed it 'coconut decline' disease.

OCCURRENCE AND DISTRIBUTION

The disease occurs in all types of soils found in Kerala and under various ecological conditions, from hilly tracts to coastal plains. A survey spanning a six year period from 1971 to 1976 showed the prevalence of root (wilt) in Alleppey district 54.6%, Kottayam 49.8%, Idukki 43%, Ernakulam 26%, and Quilon 27%. Trichur and Trivandrum had very low incidence of the disease.⁹ Another survey conducted in 1984/85¹⁰ showed that 4,10,000 ha in eight southern districts of Kerala was affected. Incidence of the disease was highest in Kottayam district and the percentage of incidence was as follows: Kottayam 75.6%, Alleppey 70.7, Pathanamthitta 38.2%, Ernakulam 34.5%, Idukki 34.2%, and Quilon 28.6%. The percentage of disease was 2.6 and 1.5 in Trichur and Trivandrum respectively. Survey revealed sparse occurrence of the disease in some isolated pockets in the northern parts of Kerala, far away from contiguously diseased area, and also in adjoining districts of Tamil Nadu. Estimated annual loss due to the disease was reported to be 968 million nuts.

TRANSMISSION OF THE DISEASE

Though the disease started in three isolated foci, separated from each other by about 50 kms, it was noted in the nearby areas in course of time. However, a natural spread of

the disease from a diseased palm to the nearest palms was not observed and the transmission was highly irregular. The spread of the disease was conspicuous in sandy, sandy loam and clayey soils and along banks of rivers and canals. As a general rule the spread of the disease can be traced along the course of water bodies like rivers and hence a water-borne pathogen was believed to be the cause of the disease. However, sporadic incidence of disease in places far interior to banks of rivers and the occurrence of several pockets of severe incidence within the healthy belt suggested other modes of spread. In fresh disease pockets near hillocks, initially the incidence is seen at the foot of the hills and subsequently it spreads up the hill. The intensity and spread of the disease was comparatively lower in laterite soils.¹¹

SYMPTOMATOLOGY

Wilting and drooping of the leaves, flaccidity, ribbing, paling/yellowing and necrosis of the leaflets are the typical foliar symptoms of the disease.^{5,6,12} With the progress of disease there is stunting of the crown due to reduction in number of leaves and successive leaves become shorter.^{5,6} Flaccidity is the earliest consistent symptom and it is observed in leaves of the central and outer whorls. It is regarded as the most frequent and common of the three foliar symptoms associated with the disease irrespective of the age of the palm or the soil type.¹²

In general, 67 to 97% palms have flaccidity, 38 to 67% develop yellowing and 28 to 48% show marginal necrosis. In the disease-affected palms of less than 10 years of age 96.8% show only flaccidity.^{13,14}

Rotting of roots is considered to be one of the symptoms of the disease^{1,5,15,16,17} the percentage of root decay varying from 12 to 94% depending on the intensity of the disease.¹⁶ It starts from the tip of the roots and progresses backwards.¹⁸ However, in a recent study no root rot was observed in a transmission experiment conducted under controlled conditions.¹⁸

Anatomical studies of the leaflets revealed degenerated chlorophyll¹⁹, reduction in wall thickness of sclerenchymatous tissues, enhanced division of upper epidermal cells, increased stomata per unit area, proliferated phloem and narrow xylem.²⁰

PATHOPHYSIOLOGY

The diseased palms showed physiological impairment. The rate of respiration was high in diseased palms when compared with healthy palms.²¹ The rate of CO₂ fixation and chlorophyll content in the leaves were significantly low in the diseased palms compared to apparently healthy palms.²² A de-arrangement of translocation and distribution of sugars²³, impaired nitrogen metabolism²⁴ and altered protein content²⁵ are associated with the disease. Concentration of total, reducing and non-reducing sugars were higher in the leaves of diseased palms. A high concentration of certain free amino acids, especially arginine²⁶, was observed in the leaves of wilt-affected palms compared to healthy palms.

A significant decrease in the total phenol content²⁷ in the roots of

disease affected palms was observed. The levels of phenol content were inversely related to the intensity of the disease. Levels of polyphenol oxidase and peroxidase enzymes were found to increase with the intensity of the disease.²⁸

Permeability of the root tissues of diseased palms was impaired, compared to healthy palms²⁹, leading to altered water metabolism. The root sap of healthy palms was acidic, odourless and clear with high K₂O and MgO content whereas the root sap of diseased palms was neutral to alkaline and foul smelling with low K₂O and MgO.³⁰ Tomato seedlings grown in the root sap of diseased palms developed epinasty and bending of leaves, but such symptoms were not observed with sap collected from healthy palms.¹⁴

Absorption of water by roots and its transport in the trunk³⁰ were considerably less in the diseased palms compared to the healthy ones. An abnormal stomatal regulation in diseased palms, leading to higher transpiration rate, was noticed in the diseased palms. The rate of transpiration was proportional to the intensity of disease.³¹ With higher rate of transpiration in diseased palms the water content is expected to be lower in the leaves but a reverse trend was observed. The root (wilt) affected palms had a lower leaf water potential which is one of the early consistent physiological indicators of the disease much before the visual symptoms (flaccidity) appear.³²

Differences in the levels of protein values, carbonic anhydrase, cellulase, and pectinolyase were observed in the diseased palms. Decelerated protein synthesis and accelerated protein breakdown was observed in the diseased palms. A reduction in the availability of biologically active zinc has been indicated

on the basis of the comparatively low levels of carbonic anhydrase activity observed in the leaves of the diseased palms.¹⁴

DIAGNOSIS

Several attempts were made to develop reliable diagnostic tests which could detect the early stages of the disease even before the visual symptoms appear. Differential dehydrogenase activity of leaf tissues³³, measurement of free amino acid arginine²⁶ and tannin³⁴, biochemical tests³⁵ and agglutination tests³⁶ were developed for early detection of the disease but all had its demerits in detecting the disease under varying environmental conditions. Remote sensing using infrared aerial photography was attempted but was not successful either.³⁷

Sero-diagnostic and physiological tests³⁸ proved to be more consistent in detecting the early stages of the disease. Time lag between detection of latent stage of the disease and manifestation of visual symptoms varied from 6 to 24 months based on serological test.

Stomatal resistance and transpiration rate of the last fully opened leaf next to the spear leaf were determined with LiCor 1600 steady state promoter.^{31,39} There was high stomatal resistance rate in the apparently healthy palms whereas it was low in the diseased palms. The transpiration rate was high in diseased palms when compared with apparently healthy palms. Leaf water potential was also found to be a useful diagnostic technique³² and it was low in diseased palms compared to the healthy palms. The symptomless palms which had the leaf water potential similar to the diseased palms developed the foliar symptoms in about 14 months.¹¹

ETIOLOGY

Through the decades various etiological factors were examined and a brief summary is given below.

FUNGI

Fungi were suspected as disease incitant because of common occurrence of *Botryodiplodia theobromae* Pat¹, *Rhizoctonia solani* (Lutu), *R. bataticola* (Taub) Butler and *B. theobromae* in the roots of diseased palms.¹⁵ Other fungi recorded were *Neocosmospora vasinfecta* Smith, *Gloeosporium* sp, *Pestalotia* sp, *Curvularia* sp, *Chaetomium* sp and *Trichoderma* sp.¹⁰

Inoculation experiments with *R. solani* and *R. bataticola* in the field as well as in large size cement tubes brought about rotting of roots, but not the foliar symptoms of root (wilt) disease.¹² The infectivity of these organisms was aggravated by water logging.¹¹ Investigations carried out so far have not indicated any role of the fungi in inciting the disease.¹⁴

BACTERIA

Pseudomonas sp. was observed as common bacteria in the roots of root (wilt) diseased coconut palms¹² and identified it as *Enterobacter cloacae* (Jordan) Hornaeche and Edwards.¹³ The coconut *Enterobacter* isolates were sensitive to streptomycin and oxytetracycline group of compounds. Treatment with these drugs, especially oxytetracycline, was effective in ameliorating the disease symptoms.¹⁴ However, in pathogenicity experiments inoculation with *E. cloacae* failed to produce the symptoms characteristic of the disease.

NEMATODES

Since root rotting was associated with root (wilt) disease and nematodes are known to affect the roots, studies were initiated to detect the presence of nematodes in the roots. Initial investigations showed very high population of *R. similis* from roots of root (wilt) affected as well as healthy palms in disease tracts. However, palms inoculated with one million nematodes, *R. similis*, even after 8 years did not produce symptoms of typical root (wilt) disease. Nevertheless, infestation of nematodes causes heavy root rotting, delay in flowering and reduction in yield.¹¹

VIRUS

Virological investigations were carried out¹⁵, considering the resemblance of root (wilt) disease to plant virus disease symptoms. It gained significance with the positive transmission of the disease through sap inoculation and transmission of disease through the insect vector, *Stephanitis typica* Distant under field conditions and under insect-proof conditions.¹⁶

Flaccidity, paling and slight stunting of young leaves were observed in five out of six seedlings mechanically inoculated with sap of diseased palms and in one out of six seedlings inoculated with *S. typica*.^{17, 18}

Failure to observe any virus particles in the affected palms ruled out the possibility of virus etiology.¹⁴

MYCOPLASMA LIKE ORGANISMS

The mycoplasma-like organism (MLO) theory was proposed since a sub-microscopic agent was suspected to induce root (wilt) dis-

ease in coconut palms.¹⁵ This hypothesis gained support with the experimental transmission of the disease to test seedlings with the insect, *Stephanitis typica* Distant under insect proof condition.^{17, 18}

Presence of MLO was identified in sieve tubes of roots, tender stem, petiole and developing leaf bases of root (wilt) disease palms.¹⁸ Sixty five samples of diseased and 35 healthy palms from disease free area have been studied. Mature tissues exhibited fewer numbers of MLO in degenerated forms.¹⁹ This is in agreement with findings on lethal yellowing disease of coconut in Florida.⁵⁰

Distribution of the organisms within the vascular bundle was sparse and not all the sieve tubes in a phloem patch contained them. Similar trend was seen in lethal disease of palms in Florida and Jamaica.^{50, 51} Failure to find MLOs in all the vascular bundles in root (wilt) affected palm could be attributed either to the low concentration per se or to an uneven distribution within the plant. None of the biological agents reported earlier to be associated with disease could be observed in the vascular tissues examined.^{52, 53} Detection of MLOs by DNA staining showed positive staining reactions in the junction of vascular bridges in rachillas. The occurrence of these reactions at scattered loci suggests the non-uniform distribution of MLOs in root (wilt) disease-affected palms as corroborated in EM observations.¹⁹

Lace bugs were found colonising in increasing number towards the inner leaves⁵⁴ and MLOs were in high concentrations in tender tissues. Lace bugs were 4 times higher in number in diseased palms than control ones.¹⁴ Since arginine, a

free amino acid, has been found at higher concentration in diseased palms, MLOs may be using arginine through the dihydrolase pathway for its energy production.⁵⁵ MLOs could not be propagated in culture media but could be maintained in rachillae explants from diseased juvenile coconut palm for more than 6-8 weeks in certain plant tissue culture media.¹⁴ Partial remission of symptoms in OTC treated palms adds further evidence to the etiological role of MLOs in coconut root (wilt) disease.¹⁴

GEOCHEMISTRY AND NUTRITION

Low pH, reduced microbial activity, nutrient imbalance together with mineral deficiencies, inadequate drainage and poor aeration has been reported as factors responsible for the incidence of root (wilt) disease.⁵⁶ In all the major soil groups studies, disease endemic areas had low available potash, exchangeable calcium and magnesium ions and total exchangeable bases. Water logging is a predisposing factor to the root (wilt) disease in palms.⁵⁶

Studies on the accumulation of major nutrients in the leaves of diseased palms showed a deficiency of calcium and magnesium.⁵⁷ In the diseased palms imbalanced nutrition with wider ratios of N/Mg, P/Mg, K/Mg and Ca/Mg indicating a lower content of Mg in proportion to other major nutrients was observed.

Scanning electron X-ray microprobe analysis indicated high deposition of Al, Mn, Cu & Co in the roots of diseased palms and Cr, Ti, Pb and Ga in the cabbage tissues of diseased palms compared to healthy ones.⁵⁸ Using X-ray fluorescence technique, nickel and strontium were found to be in higher concentrations in the roots of the diseased palms.⁵⁹

Beneficial impact of the correction of magnesium deficiency was significant.^{60,61,62,63} It reduces the prebearing age of the palms by up to 9 months and increases nut yield; the response to magnesium was more pronounced in diseased palms.

However, magnesium deficiency alone cannot be the causative agent as a systematic detailed investigation on the soils and leaves from healthy and disease-affected tracts has ruled out the possibility of direct involvement of major nutrients in the incidence of the disease.¹⁴

Recently, a hypothesis implicating geochemical factors in the incidence of the disease was proposed following analysis of the leaves of healthy and diseased palms from disease-endemic and healthy palms from non-endemic regions.⁶⁴ According to the hypothesis, the lanthanides, especially cerium, in conjunction with magnesium insufficiency impair metabolism leading to the disease.

The geochemical hypothesis calls for a re-examination of the possible role of suspected biological agents. The prevalence of root (wilt) disease in endemic zones or the geographical delineation of the disease¹⁴ suggests the role of some predisposing environmental factor(s). From available data on the transmission of the disease, it is found that disease transmission from the affected palm to the nearest healthy one is seldom observed. A natural transmission to the adjacent palm is not always the case. In field experiments on transmission of disease in the disease endemic region, five out of six seedlings mechanically inoculated with root sap from diseased palms and one out of six seedlings inoculated with insect vector *S. typica* (Distant) contracted the disease, diagnostic criteria being

serological. However, the 20 young palms or 13 seedlings situated in a disease-free area, subjected to mechanical inoculation of root sap from diseased palms for about 2 years at monthly intervals did not develop the disease⁶⁵, indicating deviation from Koch's postulates on infection. It would appear therefore that coconut root (wilt) is not an infective disease.

Further, there is no overlap between sites of MLO infection and sites showing symptoms in the diseased palms. Flaccidity, a consistent symptom, is observed in the middle and outer whorls while MLOs are concentrated in the tender tissues of diseased palms.¹⁴ In the management of diseased palms, the new leaves emerging on removal of the affected ones do not show flaccidity in the initial period, an observation consistent with the above view. Thus, a cause and effect relationship of symptoms and MLO population cannot be established. The augmented levels of MLO population could be the result of increased levels of arginine which is utilised by MLO for energy production.

Against such a backdrop, the geochemical hypothesis would explain some of the phenomena discussed above. The contiguous nature of disease tract or geographical delineation may be explained on the basis of chemical composition of the soils present in the endemic region. In disease endemic region, the charnokites and khondalites, containing rare earth elements⁶⁶, may explain the geological delineation. Though pockets of charnokites are present in the northern parts of Kerala, Dharwar formation and peninsular gneisses which have different characteristics also occur in these regions.

Though lanthanides with atomic numbers from 57-71, are called rare earth elements, they are by no means rare. They occupy the 25th place among elements in the order of decreasing abundance and form $1/6$ th of all elements of the earth. Nevertheless, it gave no less trouble to the chemists to understand them in relation to the remaining $5/6$ th of the elements. In short, abundance of REEs in the earth is 2 times that of tin, 10 times that of lead, 320 times that of antimony, 1600 times that of silver, 2500 times that of mercury or 32,000 times that of gold. Monazite can contain upto 50% of all the cerium group elements.

Normally, lanthanides like cerium are present in nature as insoluble oxides and phosphates. Due to physico-chemical changes, anaerobic microbial activity or the action of phosphate solubilizing fungi, especially in the acid soils, ionic specification might take place. Further, manganese dioxide present in the soil influences the mobility of lanthanides in the environment.^{67, 68} The soluble cerium which is in the Ce(III) valence state is transformed into the (IV) valence state in presence of oxygen to form the less soluble hydroxide which in turn forms a complex and is absorbed onto manganese dioxide.^{67, 68} However, in anaerobic or poorly aerated conditions, manganese may be converted from the insoluble oxide to its soluble form, causing depletion of the former in the environment. Increased levels of manganese in the cabbage leaves of diseased palms as compared to the healthy controls reflects the augmented levels of bioavailable manganese in the diseased tract.³³ Field observation of coconut root (wilt) disease where water logging and poor aeration are obtained^{14, 31} may

further support this mechanism.

The role of lanthanides and their essentiality in plants have not been identified although they are absorbed by the plants. Lanthanides, especially the ones which can exist in more than one state of oxidation, cause flaccidity. They impair cell permeability and inhibit entry of other ions such as potassium without altering the ion pumps. Magnesium entry may also be inhibited. This may explain the low potassium and magnesium in the root sap of diseased palms. To compensate the impaired cation permeability, water molecules have a greater tendency to enter and remain in the cells. The mechanism of water entry due to impaired cell permeability is described elsewhere.⁶⁹ This may explain the higher water content in the leaves, number of stomatal opening in the leaves as a physiological adaptation to eliminate excess water, higher transpiration rate and low leaf water potential in the diseased palms. The primary impairment of water metabolism in the diseased palms could be explained on the basis of interaction of lanthanides with the cell membrane especially at the site of root caps where absorption takes place. Such interaction could be more pronounced under conditions of magnesium insufficiency.

Magnesium is lower in disease endemic acid soils. Magnesium deficiency alone would not explain coconut root (wilt) disease as magnesium supplementation improved the productivity only partially, indicating irreversible damage. The incidence of disease in palms in the endemic region despite magnesium supplementation from the time of planting may be due to the availability of lanthanides to the palms. Bioavailability of trace metals de-

pends on various environmental factors including humates. Humic, fulvic and other organic acids in the humates interact with lanthanides to form organo-metal complexes by adsorption, chelation or other physico-chemical processes. These physico-chemical processes decrease the availability of lanthanides to the palms.^{70, 71} The humus content of soils from coconut farms in Karunagapally, an endemic region, was significantly lower compared to soils from Manavalakurichi^{72, 73}, a region which has high deposits of monazites and free from root (wilt) disease. In the field conditions, generally lower intensity of disease is observed in the palms where organic manuring is practised. Remission of symptoms and higher productivity in the disease affected palms are observed with organic manure application. All these possibly indicate the complexation of lanthanides by humic and other organic acids present in the humus whereby their availability to the palms and consequent toxic effects may be reduced. It is possible that the ameliorative effect of organic manure is due to the complexation of toxic heavy metals and not to its micronutrient content.

Given the implications of the problem for the economy and the challenge it has posed to investigators over the decades, the recent insights into the possible role of lanthanides in the causation of the disease warrant further incisive studies.

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