

POSSIBLE INVOLVEMENT OF BIOLOGICAL AGENTS IN COCONUT ROOT (WILT) DISEASE

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ABSTRACT

Coconut root (wilt) disease is a complex malady. The disease is non-lethal but debilitating in nature. It occurs under varying ecological and soil conditions. Spread of the disease, slow and erratic, is favoured by light soils and high water table. The disease syndrome consisting of rotting of roots, ribbing and marginal necrosis of leaflets and chlorosis of leaves is manifested in different combinations and intensities. Root rot precedes foliar symptoms. Damage to the vascular tissues in the absorbing region of externally healthy roots of apparently healthy palms in the disease affected area also occurs. A sap transmissible virus was suspected to be involved in the disease. *Rhizoctonia solani*, *R. bataticola*, *Fusarium equiseti*, *Cylindrocarpon effusum*, *Enterobacter* sp. and *Radopholus similis* have been found associated with the affected roots. A critical assessment of the involvement of these soil borne pathogens in initiating the symptoms of the disease at the root level is attempted. The delay in symptom expression culminating in debilitation of the palm consequent on infection(s) is attributed to the massive root zone of the palm conditioned by soil environment.

INTRODUCTION

Coconut root (wilt) disease still remains a baffling malady since its appearance after the great floods of 1882 in Central Kerala. Menon (1961) reviewed the results of investigations on this complex problem from the biological, soil and nutritional angles. Based on this background and further recent information obtained on the probable role of biological factors as causal agents of the disease is discussed in this paper.

Epidemiology

A survey conducted on the distribution of the disease revealed that nearly 2.5 lakh ha of coconut gardens are affected (Pillai *et al* 1972). Sporadic occurrence of the disease in outlying areas

of the diseased tract, incidence of disease in all types of soil and pockets of healthy gardens/palms within the diseased areas are points of interest. Spread of the disease is faster and rampant in sandy, sandy loam and clayey soils than in laterite soils. Banks of rivers, canals and areas subject to waterlogging/inundation favour disease development. It was further suggested by them, that the heavy incidence of disease on river banks near foot hills while the lower tidal part is comparatively free of disease indicates its spread through water.

Symptomatology

The symptoms of the disease are systemic in nature, characteristic features being general yellowing and drooping of the outer whorl of leaves, flaccidity, ribbing of leaflets, abnormal bending of petioles which in acute condition tend to twist and marginal necrosis of leaflets. Radha and Lal (1972) reported the frequency and pattern of occurrence of the three major symptoms, namely flaccidity, yellowing and necrosis under different soil conditions and age groups of palms. Flaccidity of leaves is often the initial and only consistent symptom, whereas necrosis is manifested later and yellowing may or may not occur. Heavy shedding of buttons and immature nuts, necrosis of spadices and pollen sterility lead to reduction in the productivity of palms (Menon and Nair, 1951; Varkey and Davis, 1960). Quality of the nut is also adversely affected—husk is thinner, fibres weaken and shell of low hardness. The thin kernel or endosperm fails to dry but remains soft and rubbery when converted into copra.

Rotting of roots is an important symptom observed by all workers starting with E.J. Butler. Butler (1906) reported that the extensive damage to cortical cells as well as collapse and death of root tip is the primary cause of the disease. Menon and Nair (1951) recorded that in the early stage of the disease the bole and the root system are elaborate and healthy but for the rotting of the tips of roots. Later studies, however, showed that the rotting of root tips precedes development of foliar symptoms which increased with the advancement of disease from 50.0—60.0 per cent in healthy and 64.0—90.0 per cent in diseased. Indira and Ramadasan (1968) observed internal browning at times

extending to the cortex and disorganization of vascular tissue and formation of tyloses in metaxylem elements in the tip region of externally healthy roots of healthy palms in the diseased area. A comparative study revealed that this condition existed in 30.0 per cent in healthy and 60.0 per cent in diseased palms. Root rot was negligible and internal damage of roots was rare in healthy palms in disease free area. Further studies on the anatomy of similar roots revealed disorganization of tracheal elements, tyloses in metaxylem vessels and degenerate phloem. Tissue degeneration was less pronounced in metaphloem (Govindankutty and Vellaichamy, 1976).

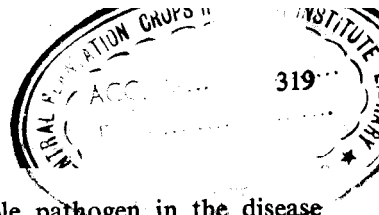
Associated biological agents

a) *Fungi*

Butler (1906) considered that a species of *Botryodiplodia* causing the extensive root rot is the causal agent of the disease. Menon and Nair (1951) observed *B. theobromae*, *Rhizoctonia solani* and *R. bataticola* associated with roots showing deep brown discolouration of the cortex which later dries up in flakes. Pathogenicity trials conducted with these fungi showed that they cause local infections leading to rotting of roots but failed to produce foliar symptoms of disease. *Rhizoctonia* spp. caused rotting of roots in 97.3 per cent of diseased palms as compared to 44.4 per cent only in healthy. Waterlogging favours infectivity of these fungi (Menon and Nair, 1952). Considering the ubiquitous nature of these soil inhabitants it was suggested that they are secondary parasites colonizing the host under conditions of low resistance (Radha and Menon, 1954).

Recent studies indicated association of *Fusarium equiseti* (Corda) and *Cylindrocarpon effusum* (Bugn) in externally healthy roots having internal damage (Thomas Joseph, 1978). Govindankutty and Vellaichamy (1976) observed presence of fungal hyphae and Radha (unpublished) spores of *Cylindrocarpon* in metaxylem. Lily (1979) observed that these organisms can establish infection on roots of potted seedlings.

COCONUT ROOT-WILT ASSOCIATES



b) *Virus*

Involvement of a sap transmissible pathogen in the disease was shown by Nagaraj and Menon (1956). They succeeded in reproducing the disease in a few palms in the field by abrasion method and through a vector, *Stephanitis typicus*. These results were later confirmed by Shanta *et al.* (1964), however, flaccidity of leaflets was the only symptom produced under insect proof conditions in potted plants. Summanwar *et al.* (1969 and 1971) reported the association of a strain of Tobacco mosaic virus in diseased leaf and root tissues. On the contrary Hariharasubramanian and Shanta (1971) did not confirm this, but opined that flexuous non-proteinaceous bodies which break into shorter particles resembling TMV occur in large numbers in root and leaf tissues of healthy and diseased palms. Maramorosch and Kondo (1977) reported the possible association of an icosahedral virus with the disease. Parthasarathy (1978) regarded these as sectional view of plasmodesmata.

c) *Bacteria*

Srivastava *et al.* (1969) observed a vascular streaming movement in roots of diseased palms. The bacterium involved was identified as *Pseudomonas* sp.

Occurrence of *Enterobacter colocacae* in apparently healthy roots of palms from the diseased area and its absence in roots of palms from disease free area is significant (Mathew George and Jayasanker, 1967).

d) *Nematodes*

Presence of *Xiphenema* and *Longidorus* species, known virus vectors of other crops, in coconut soils in the disease affected tract was reported by Weischer (1967) and Mathen (1969). Abundant distribution of the burrowing nematode *Radopholus similis* in the roots of diseased coconut palms as compared to healthy palms in disease free area which is capable of causing damage to the root system has been reported by Koshy *et al.* (1975).

Possible role of biological agents

Shanta *et al* (1964) succeeded in producing flaccidity of leaflets, paling and stunting of leaves in four year old potted coconut seedlings under insect proof conditions by abrasion method. Necrosis of leaves did not develop and roots did not rot. Holmes (1965) described the presence of a sap transmissible agent as virus/virus-like pathogen in coconut root (wilt) disease. Although the nature of this pathogen is yet to be confirmed, later studies also suggest (Summanwar *et al* 1964; Maramorosch, 1977) the association of a virus in the disease complex. Occurrence of necrosis of phloem in the distal ends of roots (Govindankutty and Vellaichamy, 1976) is a pertinent point to be considered in this context. Occurrence of *F. equiseti* and *C. effusum* and *E. colocacae* in the absorbing region of roots exhibiting degeneration of vascular tissues is also significant. Whether any of these organisms are responsible for the derangements of the vascular tissues or they are only secondary invaders of the already infected tissues is yet to be proved. Probably the activity of fungi and/or bacterium under congenial soil environment may also be essential to pave the way for the manifestation of the completed disease syndrome.

Preponderance of *R. similis* population in the maturing regions of the roots, causing lesions in the cortical tissues and association of *Rhizoctonia* spp. again leading to cortical decay lend evidence to the probable role of these agents in aggravating the deterioration of root system infected by a primary agent.

The possibility of a sap transmissible pathogen initiating a chain of physiological aberrations (Ramadasan, 1964), leading to biochemical changes (Jayasankar and Joseph, 1974), nutrient accumulation (Menon, 1961) and histological alterations (Govindankutty and Vellaichamy, 1976) culminating in the disease syndrome aided by the soil borne pathogens at the functional zone of the root system is a valid hypothesis.

Pillai *et al* (1975) reported that the apparently healthy palms and the diseased ones are on par in their nutritional status and quite distinct from that of healthy palms growing in disease free areas.

The incidence of vascular derangement in low percentage of the roots of apparently healthy palms may be the initial expression of disease. With the increasing damage to the roots and impairment of physiological functions, it is presumed that the visual symptoms are manifested. A similar situation exists in the sudden death of clove trees in Zanzibar and Pamba (Nutman and Roberts, 1971).

The extensive and massive root system of the coconut palm enables it to withstand the activity of the pathogen(s). The delay in the expression of foliar symptoms, the slow advancement of the disease and its debilitating nature perhaps depend on the extent of damage to the root system. Observations of Govindankutty and Vellaichamy (1976) on the occurrence of fungal hyphae (?) and tyloses in metaxylem and degeneration of phloem which is less pronounced in metaphloem lend support to this view. They suggested that "degenerative changes in conducting elements could derange the flow of nutrients and thereby contribute to root death due to starvation." The variations in symptom picture may also be due to the differences in pathogenic association of fungi, nematodes and bacteria in roots and the consequent alterations in host reactions. Development of a superimposing disease leaf rot, (*Bipolaris halodes*) only in some of the root (wilt) affected palms (Radha and Lal, 1968) and sporadic incidence of rubbery kernel in diseased palm (Pillai, 1975) indicate that the physiological derangements are not uniform or parallel in all the disease affected palms. Indeed, each one of these biological agents may contribute in creating a complex situation. The variation observed in the manifestation of symptoms may thus be explained as due to involvement of one or more pathogens and their different associations (which is further complicated by the perennial nature of the crop).

Thus the coconut root (wilt) disease is to be considered conglomeration of various entities of which the biological agents have a decisive role conditioned by biotic environmental factors as well as their own synergistic effects. The relative importance of each one of the biological agents and their sequential involvement which are yet to be assessed will provide a better understanding of the problem and pave the way for evolving effective control measures.

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