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RECENT ADVANCES IN RESEARCH ON ROOT (WILT) DISEASE OF COCONUT*

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ABSTRACT

Root (wilt) disease of coconut is a non-lethal but debilitating malady affecting the productivity of the crop. The etiology of the disease was not known for over a century. But recent studies have ruled out the involvement of biological agents such as fungi, bacteria, nematodes and viruses and nutritional and physiological factors in the etiology of the disease. Constant association of mycoplasma-like organisms with the disease has been established. The vector role of lace bug has been confirmed through transmission experiment and other direct and indirect evidences gathered. The disease also could be experimentally transmitted to periwinkle, a known mycoplasmal indicator host through the dodder laurel *Cassytha filiformis*. Remission of symptoms obtained in Oxytetracycline treated palms added additional proof for mycoplasmal etiology of the coconut root (wilt) disease.

INTRODUCTION

Coconut root (wilt) disease was first observed around 1874 from Erattupetta area of Meenachil Taluk, Kottayam District, Kerala. However, it became significantly evident after the great floods of 1882 (Butler, 1908; Varghese, 1934). The disease was then reported from other areas, Kaviyoor and Kalloopara in Thiruvalla Taluk and from Kayangulam of Karthikapally Taluk. It has since then spread in all directions from the original sites of infection and according to a survey conducted in 1984/85, it is prevalent more or less in a contiguous manner in 4,10,000 ha in the eight southern districts of Kerala. The intensity of the disease ranged between 75.6%, the highest in Kottayam district to 2.6 and 1.5% in Thrissur and Thiruvananthapuram districts respectively (Anonymous, 1985a). Isolated incidence of disease occurrence is also noticed in Kannoor, Kozhikode, Malappuram and Palakkad districts (Radha et al, 1985).

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. A sero-diagnostic test (Solomon, Sasikala and Shanta, 1983) and a physiological test based on stomatal resistance (Rajagopal, Patil and Amma, 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).

Etiology

Fungi:

The etiology of the disease remained an enigma until recently. A fungoid concept was proposed by Bourdillon in 1906 (Varghese, 1934). A number of fungi, *Botryodiplodia theobromae*, *Rhizoctonia solani*,

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R. bataticola, *Fusarium equiseti* and *Cylindrocarpon effusum* were reported to be associated with the disease (Menon and Nair, 1951; Joseph, 1978; Lily, 1979). Pathogenicity trials with these fungi however failed to reproduce the symptoms of the disease (Menon and Nair, 1951; Anonymous, 1985 b).

Bacteria:

Isolation of the following bacteria *Pseudomonas* sp. and *Enterobacter cloacae* from roots of diseased palms was also reported (Srivastava, Shekhawat and Rao, 1969; George, Potty and Jayasankar, 1976). Pathogenicity studies however, ruled out the involvement of bacteria in disease causation.

Nematodes:

Analysis of the soil around the root zone of coconut yielded 39 genera of plant parasitic nematodes (Sosamma and Koshy, 1991). Extensive investigations with *Radopholus similis*, the most important nematode, did not prove to be an incitant of the coconut root (wilt) disease (Anonymous, 1988).

Virus:

Although the involvement of a submicroscopic agent, possibly a virus, was implicated by a number of workers (Nagaraj, Davis and Menon, 1954; Shanta and Menon, 1960; Summanwar et al, 1969; Maramorosch and Kondo, 1977) no virus could either be isolated or consistently observed in diseased plant tissues. Polyacrylamide gel electrophoretic analysis of nucleic acid from diseased palms also excluded the association of any viroid type pathogen (Randles and Hatta, 1980).

Nutrients:

Extensive analysis of soil and leaf tissues of palms in healthy and diseased tract and various fertilizer trials conducted clearly indicated the non-involvement of major or micronutrients in the incidence of the disease (Pillai et al, 1975; Davis and Pillai, 1966; Davis, 1969; Khan et al, 1985; Cecil, 1981; Cecil et al, 1982).

Physiological factors:

The physiological and biochemical changes observed in diseased palms such as increased rate of respiration (Michael, 1978), deranged translocation and distribution of sugars (Mathew, 1977), altered nitrogen metabolism (Varkey, Michael and Ramadasan, 1969), accelerated phenol metabolism (Joseph, Potty and Jayasankar, 1976) and permeability changes (Ramadasan, 1964; 1967) account for pathogen induced alterations than of a physiological disorder.

Mycoplasma-like organisms (MLOs):

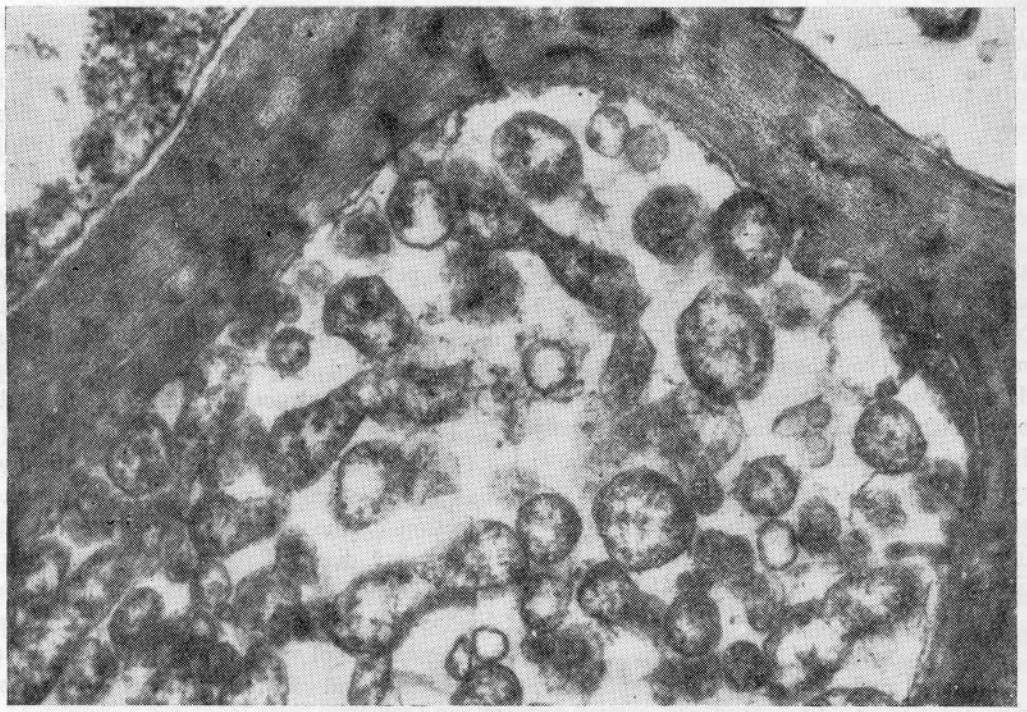
Histopathological studies on palms of different disease intensities evidenced structural changes like disorganisation and degeneration of vascular tissues, increased chromophily in phloem tissues and necrotic obliteration (Govindankutty and Vellaichamy, 1983). Ultrastructural studies of the vascular tissues revealed the presence of mycoplasma-like organisms in sieve tubes of roots, tender stem, petiole and developing leaf bases of root (wilt) diseased palms (Solomon, Govindankutty and Nienhaus, 1983). Location of a phloem bound mollicute is of significance in the light of structural damages noticed.

Constant association of MLOs with the disease has since been established with the detection of the organisms in tissues of all the seventy diseased palms as against their total absence in fifty healthy palms from disease-free area studied. The palms examined are of various age groups, disease intensities and from different locations. The mollicutes are found in increasing

numbers in the 'Sink' region. Degenerated or moribund forms are often observed in mature tissues (Solomon, Govindankutty and Mathen, 1987). Polymorphic forms varying from circular to oval and occasionally beaded or filamentous ones are also observed. The coccoid forms are in the range of 250–400 nm, limited by well defined trilamellar unit membrane and contain DNA strands and peripherally dispersed ribosomes (Fig. 1). The walls of the sieve cells harbouring the organisms and the bordering cells are thickened, cytoplasm granulated and contained paramural bodies. In the sieve tubes MLOs are observed in parietal position and more frequently found to congregate close to the sieve area. None of the biological agents reported earlier to be associated with the disease could be observed in the vascular tissues examined. Distribution of the organism within the vascular bundle is sparse and not all the sieve tubes in a phloem patch contained them. Similar trend of uneven distribution of the organism is also observed in other coconut diseases too. In lethal yellowing diseased palms only 5% of the vascular bundles are, found to contain the organism in very low concentration (Thomas, 1979).

Histochemical studies of juvenile tissues from diseased palms revealed abnormal bluish colouration following Dienes' staining and increased fluorescing sites in sieve area consequent to staining with the fluorochrome 4'6 – diamidino–2 phenyl indole 2 HCl (DAPI) and Hoechst 33258 (Solomon, Govindankutty and Mathen, 1987). These staining reactions indicative of accumulation of DNA in extranuclear sites implicate the

Fig. 1. MLOs in sieve tubes of tender rachilla of root (wilt) diseased palm



presence of MLOs. Such abnormal reaction is not evident in tissues of healthy palms. The staining reaction is found to be more frequent in junctions of vascular bridges in rachillae. Not all the sieve tubes of any phloem patch of root or every vascular bundle of rachilla exhibited positive reaction. This corroborates the EM observation on the non uniform distribution of the organism (Solomon et al., 1987). The specificity of dienes' and fluorescence staining to bind with nucleic acid component of mycoplasma is being utilised as a diagnostic tool for identifying diseased palms.

Insect-vector studies

Data accrued on the constant association of MLOs with the disease warranted the identification of insect vector(s). Lace bug – *Stephanitis typica* Distant (Tingidae) has all along been implicated as the vector of the disease being the single major group of insect on coconut and also based on transmission experiments (Nagaraj and Menon, 1956; Shanta, Joseph and Lal, 1964). MLOs being generally transmitted by leaf hoppers and plant hoppers and rarely by psyllids, the vector role of lace bug in this context had to be reinvestigated.

Record of insects on coconut from India did not include any members of Auchenorhyncha. A systematic inventory of insects in coconut gardens made through various traps and direct examination of over two hundred seedlings for a period of two years led to the identification of a leaf hopper, *Sophonia greeni* (Distant) and a plant hopper, *Proutista moesta* (Westwood) (Rajan and Mathen, 1984; 1985). A rapid survey of the selected gardens in eight districts where the disease occurs contiguously revealed that the disease does not occur independent of these three insects. Potential of these insects to acquire the phloem bound mollicute and sustain its multiplication was determined through EM studies of these vector candidates. MLOs were observed in salivary glands and brain tissues of lace bug given an acquisition and incubation period (A+I) between 18–23 days (Mathen et al, 1987). MLOs were, however, not observed in lace bugs collected from disease free areas such as Kasaragod and Minicoy in Lakshadweep and also in bugs offered A+I less than 18 days. Mycoplasmas have also been recently recorded in plant hopper given A+I of more than 37 days (Anonymous, 1990).

These pleomorphic bodies found in the acini of salivary glands resembled in morphology and structure of the organism found in root (wilt) disease affected palms.

Insect transmission

Transmission of the disease from coconut to coconut through lace bug in the field (Shanta, Menon and Patchu Pillai, 1959) and in insect proof house (Shanta, Joseph and Lal, 1964) had been reported when virus etiology for the disease was implicated. In the light of the present findings on the constant association of MLOs with the disease, the transmission experiment with lacebug was repeated.

Four West Coast Tall two year old seedlings planted in methyl bromide fumigated soil and protected with insect proof field cages were inoculated with lace bugs offered 5 days acquisition feeding on diseased palms and insects offered 18 days A+I on diseased palms as detailed in Table I. Nine months after the first inoculation, three out of four seedlings gave strong positive serological reaction and faint reaction in the fourth indicating disease contraction. Light microscopic staining with Dienes' and fluorochromes, DAPI and Hoechst 33258 indicated positive staining in extra-nuclear

Table I. *Number of lace bugs inoculated on experimental coconut seedlings in field cages numbered* 3, 4, 9 *and* 10

Date	5 days' acquisition				Total	5 days' acquisition plus 13 days' incubation				Total
31 Dec 1985	184	180	180	174	718					
31 Mar 1986	200	196	202	180	778					
30 Jun 1986	631	637	639	664	2571					
Total	1015	1013	1021	1018	4067					
30 Sep 1986						626	621	616	595	2458
31 Dec 1986						427	431	434	458	1750
31 Mar 1987						90	76	80	76	322
Total						1143	1128	1130	1129	4530
30 Jun 1987	100	101	106	97	404					
30 Sep 1987	278	285	257	287	1107					
30 Nov 1987	—	705	—	706	1411					
Total	378	1091	363	1090	2922					
Grand total	1393	2104	1384	2108	6989					

sites confirming MLO infection. EM examination of root tissues further confirmed the presence of the organism in all the four seedlings, 9 to 27 months after the first inoculation. Flaccidity of leaflets, the decisive and diagnostic symptoms of the disease was evident in two of the seedlings by the 17th month. None of the uninoculated control seedlings either exhibited visual symptoms or showed the presence of MLOs (Mathen et al., 1990).

Apart from the direct evidences emerging from the transmission experiment a number of indirect evidences also lent support to its vector role. Lacebugs were found colonising in increasing number towards the inner leaves where the active forms of MLOs are found in higher numbers (Mathen, Mathew and Kurien, 1969). Population of lacebug on diseased palms was found to be four times more than in asymptomatic palms (Mathen, 1982). After monitoring the lace bug population in a garden of over two hundred seedlings for two years Mathen (1985) reported a direct linear correlation between the number colonising the palms and percentage of fresh incidence of disease. Ability of lace bug to feed on phloem tissues and acquire the phloem bound mollicute was established by tracing the stylet course of insect fixed in feeding position by a cold immobilisation technique (Mathen et al., 1988). Locating MLOs in salivary glands and brain tissues of infective lacebugs, transmission of the disease from diseased to healthy coconut seedlings employing this insect and the indirect evidences accrued decisively confirm its vector role.

Dodder transmission

Experimental transmission of the disease from diseased coconut to periwinkle, *Catharanthus roseus* G. Don. a known mycoplasmal

indicator host, employing certain phanerogamic parasite was also achieved. Dodder species, *Cuscuta campestris* Yunck; *C. chinensis* Lam. and *C. subinclusa* Dur. and Hilg. widely used in plant virus transmission studies although established on coconut foliage, failed to put efficient haustorium to reach the vasculature. Probably this accounts for the failure of Tsai (1983) to transmit lethal yellowing with *C. campestris*. A dodder laurel *Cassytha filiformis* was however found to establish well forming intimate organic contact on coconut foliage. Dodder laurel established on periwinkle maintained under insect proof cage and bridged on to diseased coconut seedlings in the field, developed chlorotic spots in the interveinal areas and at vein endings of fully opened leaves three to four weeks after the establishment of the haustorium. Transmission of the disease was confirmed with positive staining reaction and EM detection of MLOs in the mid-vein and petiolar tissues of periwinkle, connecting dodder strands and in the leaflets of diseased coconuts. MLOs were however not observed in dodder established on disease-free coconuts and in control periwinkle plants (Sasikala et al, 1988). Although *C. filiformis* had been employed to transmit citrus mosaic from sweet orange, *Citrus sinensis* (L) Osbeck to acid lime *C. aurantifolia* (Christm, Swingle) (Reddy, Naidu and Gopalaraju, 1985) this is the first instance of MLO being transmitted by an unconventional dodder species.

Culturing

Though the disease could experimentally be transmitted to coconut palms through lacebug and to periwinkle through dodder, culturing of the organism *in vitro* is vital to ascribe its etiological role. Since plant mycoplasmas have so far defied cultivation, medium mimicking the phloem environment may prove useful for culturing of the organism. Phloem sap rich in nutrients has been found as an ideal medium either as such or with serum supplements for culturing *Acholeplasma laidlawii, Mycoplasma fermentans* and *Spiroplasma citri* (Eden, Green and Waters, 1982; McCoy, 1976; 1977) and *Phytomonas davidi* (McCoy, 1978).

A method of collection of vascular sap in unfermented condition has been standardised (Rajagopal et al, 1988). The physicochemical condition of the vascular sap from apparently healthy and diseased palms has been analysed. Sap from diseased palms had higher arginine level than that in healthy palms. It is surmised that the MLOs in root (wilt) diseased palms may be of the non fermentative type which utilises arginine through dihydrolase pathway for energy production (Chempakam and Rajagopal, 1989).

The vascular sap from apparently healthy palms with or without supplements was used for the preparation of culture medium. In addition, about forty different media with various combination of growth factors, nucleic acid precursors, cofactors and vitamins were used for culturing the organism from tissues of diseased palms, symptomatic periwinkle and infective lace bug. A number of methods and cultural conditions were also tried. Embryonated hen's egg was also utilised as a model system. However, the organism could not be cultured in any of the media (Anonymous, 1989). Attempts are being made to maintain/propagate MLOs in tissues of diseased palms, symptomatic periwinkle, dodder parasitised on diseased coconut and infective lace bug. The mollicutes could be maintained in explants of diseased palms cultured in certain plant tissue culture medium for more than 6–8 weeks. This line of investigation is being further pursued.

Chemotherapy

Since plant mycoplasmas are not cultivable *in vitro*, circumstantial evidences such as consistent occurrence of the organism in diseased plants, transmission of the disease through vector and differential chemotherapy are considered as the best evidences to adduce a mycoplasmal etiology for any disease. Results of a field trial with four concentrations of Oxytetracycline hydrochloride (OTC), a single concentration of Penicillin and distilled water control clearly indicated remission of symptoms in palms treated with 3 and 6 g ai of OTC. Contrastingly, palms in both Penicillin and distilled water controls deteriorated over the pretreatment condition (Solomon and Govindankutty, 1991).

Since there are no curative or prophylactic chemicals available which could offer complete protection against MLO infection the alternatives left are (a) breeding for disease resistance (b) prevent spread of the disease through insect control (c) eradication of diseased palms in the mildly affected areas and (d) integrated management of diseased gardens to obtain optimum yield.

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