COCONUT ROOT (WILT) DISEASE

CENTRAL PLANTATION CROPS RESEARCH INSTITUTE
(Indian Council of Agricultural Research)
KASARAGOD - 671 124, KERALA, INDIA

CPCRI

COCONUT
ROOT (WILT) DISEASE

Editors

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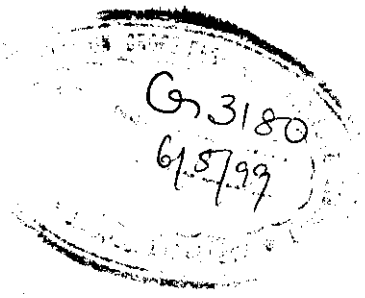

Front cover : Root (wilt) diseased coconut palm

Back cover : Elite palm in diseased tract

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Editors

FOREWORD

One of the main reasons for the low productivity of coconut in Kerala is the prevalance of root (wilt) disease. This malady reported in 1882 has spread from the original focus to eight districts in the state contiguously from Thiruvananthapuram to Thrissur and in isolated localities in a few northern parts of Kerala as well as in areas of Tamil Nadu adjoining Kerala. The mean percentage of incidence is 24.05 per cent, intensities ranging from 2.09 per cent in Thiruvananthapuram to 48.0 per cent in Alappuzha.

Research on root wilt initiated in the late 1940's at Central Coconut Research Station, Kayangulam was further intensified after the formation of Central Plantation Crops Research Institute in 1970 under the Indian Council of Agricultural Research.

The results from breeding for tolerance using disease free high yielding palms located in hot spot areas and the success obtained through management of the disease in diseased areas are encouraging. The research information generated was originally compiled and published during 1991. It is felt necessary to up-date the information gathered since then for the benefit of farmers, students, teachers and scientists and the present publication is an attempt in this direction.

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LIST OF ABBREVIATIONS

Abbreviations	Long form
ALPY	Alappuzha
CEC	Cation Exchange Capacity
CGD	Chowghat Green Dwarf
COD	Chowghat Orange Dwarf
CPCRI	Central Plantation Crops Research Institute
DAPI	4,6-Diamidino - 2-phenylindole
DNA	Deoxyribo Nucleic acid
DTPA	Diethylene Triamine Pentaacetic Acid
EDTA	Ethylene Diamine Tetracetic Acid
ELISA	Enzyme linked immunosorbent assay
EM	Electron Microscopy
IBA	Indole Butyric Acid
IDK	Idukki
KGD	Kulasekharam Green Dwarf
KLM	Kollam
KTM	Kottayam
MLO	Mycoplasma Like Organism
MYD	Malayan Yellow Dwarf
NAA	Naphthalene Acetic Acid
NASA	National Aeronautic and Space Agency
NCD	Natural Cross Dwarf
NPK	Nitrogen, Phosphorous, Potassium
OP	Open Pollinated
OTC	Oxytetracycline
PAL	Phenylalanine Ammonia Lyase
PTA	Pathanamthitta
RLO	Rickettsia-like Organism
TMV	Tobacco Mosaic Virus
TVM	Thiruvananthapuram
VAM	Vesicular arbuscular Mycorrhiza
WCT	West Coast Tall

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ORIGIN, DISTRIBUTION AND PRODUCTION LOSS

JACOB MATHEW, T.S.S. RAWTHER and N.G. PILLAI

Root (wilt) disease of coconut has been known to be present in South Kerala (India) since 1882 (Butler, 1908; Varghese, 1934). Butler referred to it as 'root disease' probably due to the rotting of roots noticed by him in the affected palms. Nagaraj and Menon (1955) felt that the name 'wilt' would be a more appropriate term for the malady taking into account the nature of foliar symptoms and subsequently, the disease came to be known as root (wilt) disease (Shanta *et al.* 1960; Mathen *et al.* 1983).

The root (wilt) disease was first reported from Erattupetta area of Meenachil Taluk in Kottayam District (Butler, 1908; Pillai, 1911). It was believed to have been significantly manifested since the great floods of 1882 (Menon and Pandalai, 1958). Around 1907, the malady was reported from Kaviyoor and Kallooppara areas of Thiruvalla Taluk and later from Kayangulam of Karthikappally Taluk (Butler, 1908; Varghese, 1934; Menon and Pandalai, 1958) (Fig. 1). The disease which appeared more or less simultaneously in the above foci began to spread to adjoining areas also (Varghese, 1934). An attempt to determine the extent of spread of the disease was made by Butler (1908) and Pillai (1911). Their studies indicated that the disease affected about 24,000 ha of coconut plantations in Meenachil, Thiruvalla, Kayangulam and Alappuzha. Later, Varghese (1934) traced the occurrence of the disease upto Kochi in the north and Kottarakara in the South. Isolated pockets of infection were also reported by him in and around Adoor, Mavelikara, Kochi and Moovattupuzha. Rapid spread of the disease along the banks of Meenachil, Manimala, Pamba and Kallada rivers was also indicated. Menon and Nair (1951) in a survey conducted during the period 1937-48 identified the areas bordered by Kollam in the south west, Panalur in the south east, Ochanthuruthu in the north west and Malayattur in the north east as affected by root (wilt) disease. Very heavy incidence was also recorded in places around Kayangulam, Alappuzha, Thiruvalla, Mattancherry and Ernakulam. Verghese (1959) in a reconnaissance survey located pockets of disease incidence in areas beyond Kollam in a few villages east of Attingal on the bank of Vamanapuram river. Pillai *et al.* (1973) reported a contiguous distribution of the disease in six districts viz. Thiruvananthapuram, Kollam, Alappuzha, Kottayam, Ernakulam and Thrissur of Kerala State. The limits of spread of the disease was reported to be Ala, Varandarappally and Kalloor in Thrissur District in the north and Nemom, Ottasekharamangalam and Maranallur of Thiruvananthapuram District in the South. According to them more than 30 per cent of the 7,50,000 ha under coconut in Kerala was affected by the disease. Sporadic occurrence of the malady was also recorded in the districts of Tirunelveli and Kanyakumari of

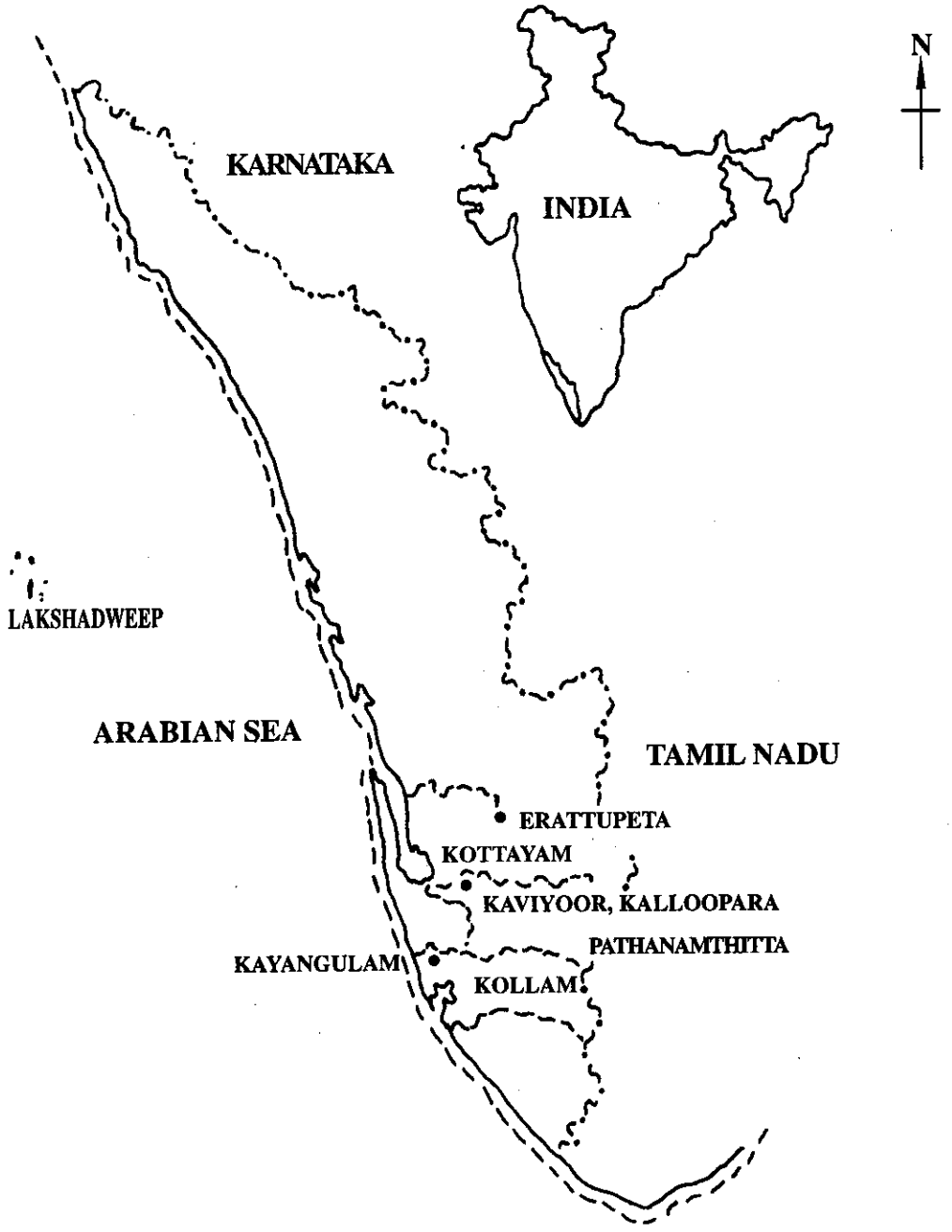

Fig. 1 Foci of initial occurrence of root (wilt) disease (indicated by ●)

Tamil Nadu State lying adjacent to South Kerala (Subba Raja and Ahamed, 1976). Among the disease affected tracts in Kerala State, Karunagappally, Kuttanad, Kanjirappally, Meenachil and Kanayannur Taluks were considered to be disease endemic areas.

George *et al.*, (1979) conducted a survey during the six year period (1971-76) in the districts of Thrissur, Ernakulam, Idukki, Kottayam, Alappuzha, Kollam and Thiruvananthapuram in order to study *inter alia* the incidence of root (wilt) disease. Their survey brought out that the disease incidence was maximum (54.6%) in Alappuzha District followed by Kottayam (49.8%) and Idukki (43.0%) Districts. In Ernakulam and Kollam Districts the disease incidence was 26 and 27 per cent, respectively. Thrissur and Thiruvananthapuram Districts had very low percentage of disease incidence.

Pillai *et al.*, (1980) indicated that the disease was prevalent in all the major soil types in Kerala State. However, the spread of the disease was observed to be fast in light textured sandy loam and alluvial soils as well as heavy textured clayey soils than in the laterites. The incidence was relatively higher in water-logged low lying areas adjacent to rivers and canals and in 'Kari' soils.

A comprehensive survey was undertaken by the Central Plantation Crops Research institute (CPCRI) in collaboration with the Department of Agriculture, Kerala and other agencies in 1984/85 to estimate the spread and intensity of disease and production loss due to the malady (Anon., 1985). The survey brought out that the disease was prevalent in more or less contiguous manner in 4,10,000 ha in the eight southern districts of Kerala. The intensity of the disease in both bearing and non-bearing categories of palms was highest in Kottayam district (75.6%) followed by Alappuzha (70.7%), Pathanamthitta (38.2%), Ernakulam (34.5%), Idukki (34.2%) and Kollam (28.6%). In the districts of Thrissur and Thiruvananthapuram the percentage of disease incidence was 2.6 and 1.5, respectively (Table 1).

The survey also revealed the sparse occurrence of the disease in some isolated pockets in the northern parts of Kerala far away from the contiguously diseased area (Fig. 2) and also in the adjoining districts of Tamil Nadu.

Department of Agriculture, Govt. of Kerala had undertaken a detailed survey in 1996 in the eight southern districts (Anon., 1997). It was a complete garden to garden survey of all the panchayath and municipal areas in the two border districts of Thiruvananthapuram and Thrissur, while for the other six districts, only 10% of the panchayaths were covered under the survey. The results of this survey revealed that the over-all disease incidence has come down to 24.05% (Table 2). Compared to the 1984-85 situation, the reduction in incidence was substantial in Alappuzha and Kottayam districts, while in Thrissur district, the disease has spread to more areas. The decline in overall disease incidence is attributed to cutting and removal of diseased palms (with assistance from Coconut Develpment Board), replanting with quality seedlings, adoption of integrated management practices and replacement of coconut with rubber.

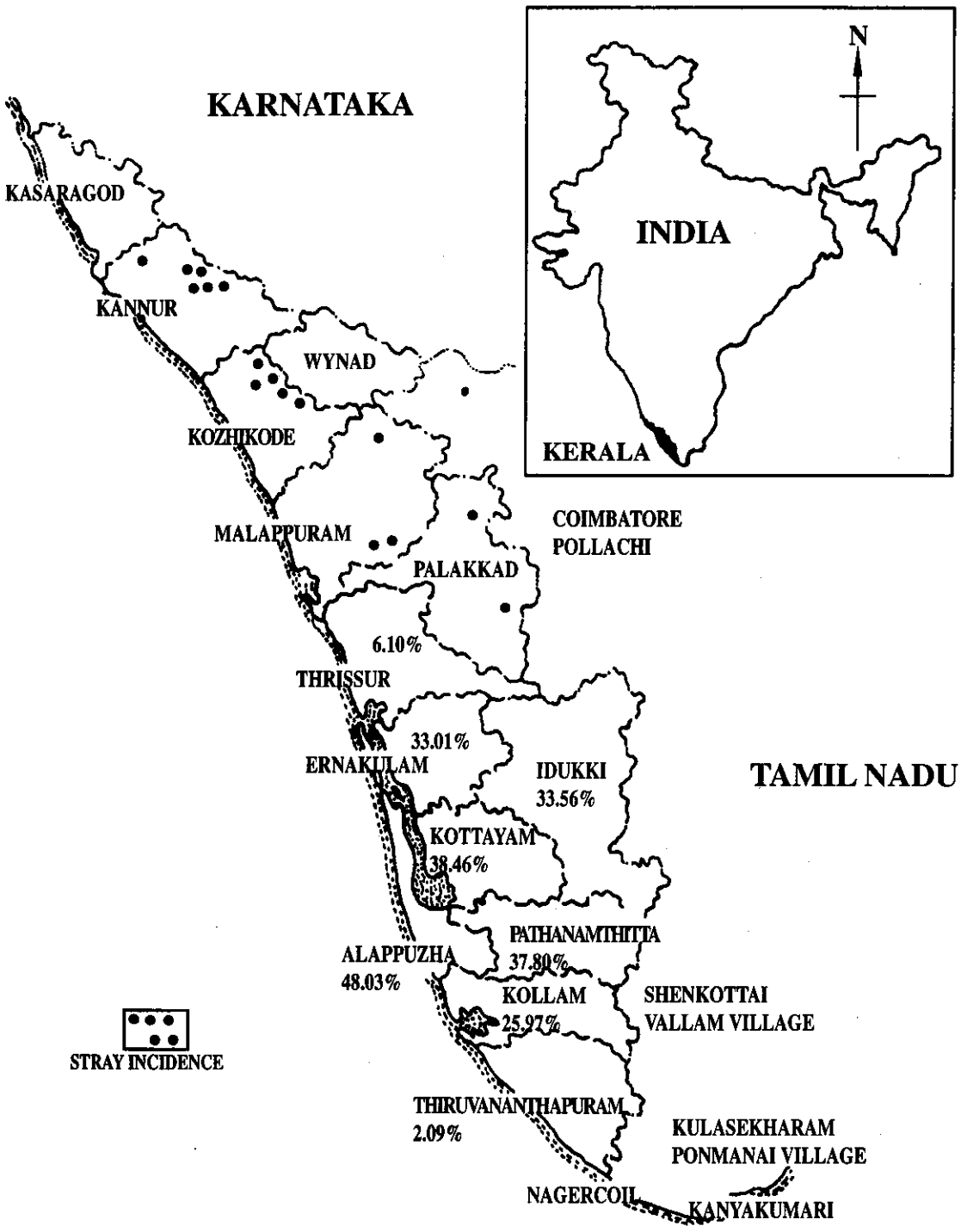

Fig. 2 Distribution of coconut root (wilt) disease in Kerala (1997)

Table 1. Percentage of palms under various categories of disease (Anon., 1985)

Sl. No.	District	Non-bearing palms			Bearing palms			Overall diseased
		DE	DA	Tot. dis	DE	DA	Tot. dis	
1.	Thiruvananthapuram	0.22	0.09	0.31	1.88	0.58	2.46	1.52
2.	Kollam	13.49	3.48	16.97	22.68	0.19	31.87	28.55
3.	Pathanamthitta	12.90	5.84	18.74	29.53	25.59	55.12	38.22
4.	Alappuzha	29.75	10.61	40.36	56.77	23.39	80.16	70.69
5.	Kottayam	37.38	9.43	46.81	61.56	24.00	85.56	75.63
6.	Idukki	4.69	0.41	5.10	27.01	8.29	35.30	34.18
7.	Ernakulam	19.64	1.75	21.39	39.91	8.66	48.57	34.52
8.	Thrissur	0.42	0.16	0.58	3.75	0.86	4.61	2.60
	Overall	13.50	3.26	16.76	29.54	11.36	40.90	32.37

DE - Disease Early; DA - Disease Advanced

Table 2. Coconut root (wilt) disease incidence - District-wise details (Anon., 1997)

Sl. No.	Particulars	TVM	KLM	PTA	ALPY	KTM	IDK	EKM	TCR	Total
1.	Total No. of root (wilt) free palms ('000 Nos.)	20464	9306	3949	7372	5416	4135	10258	16560	77460
2.	Total No. of disease early palms ('000 Nos.)	252	1970	1621	4756	2406	1264	3594	687	16550
3.	Total No. of discease advanced palms ('000 Nos.)	185	1294	779	2057	980	825	1461	407	7988
4.	Total No. of Palms ('000 Nos.)	20901	12570	6349	14185	8802	6224	15313	17654	101998
5.	Percentage of disease incidence	2.09	25.97	37.80	48.03	38.46	33.56	33.01	6.10	24.05

George *et al.* (1979) estimated the annual loss due to the disease as 340 million nuts while the subsequent comprehensive survey (Anon., 1985) revealed that the loss has increased to 968 million nuts. The district-wise split up of production loss due to the disease as per the latter survey is given in Table 3.

Table 3. Estimated production loss due to root (wilt)disease in different districts of Kerala (Anon., 1985)

Sl. No.	District	Production loss (in million nuts)
1.	Thiruvananthapuram	11.34
2.	Kollam	110.56
3.	Pathanamthitta	99.39
4.	Alappuzha	271.02
5.	Kottayam	254.39
6.	Idukki	31.11
7.	Ernakulam	177.13
8.	Thrissur	12.65
	Total	967.59

The survey conducted in 1984-85 also brought out that the loss in husk per nut of the diseased palm was around 25.8 per cent and that of copra and oil per nut was 9.0 and 11.3 per cent respectively. The loss in yield of nuts and leaves is to the tune of 60 per cent.

Separate studies have also been undertaken to estimate the decline in yield due to the incidence of root (wilt) disease. Abraham *et al.* (1987) obtained a significant negative correlation between yield and disease index of affected palms. Jacob Mathew *et al.* (1993) observed in disease affected palms a general yield decline of about 45% in W.C. Tall and about 60% in D x T hybrids, compared to the healthy ones under good management. In palms which contracted the disease in early stages, the loss was still higher. The onset of bearing was also found to be delayed in palms which become diseased in the pre-bearing stage.

It may be seen that the root (wilt) disease is mostly confined to the eight southern districts of Kerala. The strategy developed by CPCRI to contain the disease within this tract and prevent its further spread to northern districts of Kerala and border districts of Tamil Nadu has been encouraging. At present there is no information on hand about the source of infection in isolated pockets of the northern districts of Kerala and in some areas of Tamil Nadu. Periodic survey for identification of fresh incidence of disease in the areas bordering these eight districts and eradication of such palms is felt desirable.

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SYMPTOMATOLOGY

N. G. PILLAI and T.S.S. RAWTHER

The root (wilt) disease is of complex nature and the external and internal symptoms expressed by the affected palms are discussed separately in the following pages.

Varghese (1934) followed by Menon and Nair (1951) and Menon and Pandalai (1958) considered wilting and drooping of the leaves, flaccidity, ribbing, paling/ yellowing and necrosis of leaflets as typical foliar symptoms of the disease (see cover picture). With the progress of the disease, there is a reduction in the number of leaves and successive leaves become smaller, shorter and narrower resulting in the stunting of the crown (Varghese, 1934; Menon and Pandalai, 1958). A better understanding of the symptomatology of the disease was arrived at on the basis of the quantitative evaluation of the foliar symptoms of 7000 palms of varying age by Radha and Lal (1972). They recorded flaccidity, the characteristic bending or ribbing of leaflets, as the earliest consistent visual symptom (Fig. 3). This will be expressed by the leaves of the central and outer whorls. Holmes (1965) pointed out that such affected leaflets were curved along the entire length and formed a structure resembling the ribs of mammals. Rajagopal *et al.* (1986) attributed this to the impaired stomatal regulation resulting in excessive water loss.

Foliar yellowing and marginal necrosis of the older leaves were observed in association with the disease (Menon and Nair, 1952; Menon and Pandalai, 1958). According to Varghese (1934), these symptoms occurred in varying intensities in the outer whorl. Varghese (1934) followed by Menon and Nair (1951) and Nagaraj *et al.* (1954) recorded paling of the younger leaves in advanced stages of the disease. Dwivedi *et al.* (1979) claimed that the initial symptoms of root (wilt) disease were the manifestations of softening and whitening of the leaflets of the spindle.

Expression of foliar symptoms varies both in frequency and association with each other, depending on the soil type and ecological conditions (Radha and Lal, 1972). In general, 67 to 97 per cent of the palms have flaccidity; 38 to 67 per cent develop yellowing and 28 to 48 per cent show marginal necrosis. Intensity of foliar symptoms also varies according to the age of the palms. In palms below the age of ten years, 96.8 per cent have flaccidity while yellowing and marginal necrosis are virtually absent. Flaccidity 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.

Based on the relative contribution of the above three visual symptoms, George and Radha (1973) developed a scoring system for quantifying the disease severity. The formula for arriving at disease index (I) is $I = \Sigma \frac{F+Y+N}{L} \times 10$, where F = flaccidity with

Fig. 3 Characteristic symptoms of the disease - Flaccidity, foliar yellowing and marginal necrosis against the backdrop of healthy trees

Fig. 4 Inflorescence necrosis

0-5 score, Y = yellowing with 0-3 score, N = necrosis with 0-2 score and L = total number of leaves. They categorised the disease into early, middle and advanced stages. This method has been further simplified and made easier by rating the three major symptoms present in the leaves in any of the five spirals (Nambiar and Pillai, 1985). Although assignment of grade points to the symptom expressions is based on visual observations, this system helps in quantifying the disease severity in simple numerical expression.

The vitality of the reproductive system is also adversely affected (Menon and Nair, 1951). Inflorescence necrosis (Fig. 4) and lack of ability to produce female flowers (Varghese, 1934) and pollen sterility (Varkey and Davis, 1960) render the palm unproductive. Nambiar and Prasannakumari (1964) observed meiotic irregularities in diseased palms. The spathes of the affected palm become small, weak and do not open normally (Maramorosch, 1964). The extent of decline in yield as a consequence of these is estimated to be 43 to 80 per cent depending on the stage of development (Radha *et al.*, 1972) and 43 per cent in disease early and 74 per cent in disease advanced palms compared to the disease free palms (Anon., 1985). The fact that the disease is not a fatal one and causes gradual yield decline as the disease progresses has prompted Dr. M S Swaminathan to call it 'Coconut decline' (Swaminathan, 1983).

Shedding of immature nuts and poor quality of nuts/copra is often attributed to be yet another character of the disease (Menon and Pandalai, 1958). The husk becomes thinner, less firm and shell does not properly harden or turn black as compared to healthy nuts (Varghese, 1934). Kernel exhibits uneven thickness, does not dry normally and remains flexible (Varghese, 1934; Maramorosch, 1964).

Rotting of roots is considered to be one of the symptoms of the disease by many workers (Butler, 1908; Menon and Nair 1949; Menon and Pandalai, 1958; Michael, 1964; Radha *et al.* 1971). Percentage of root decay varied from 12 to 94.4 depending on the intensity of disease (Michael, 1964; Radha *et al.*, 1971). Butler (1908) observed that half of the main roots were affected by rot in some disease advanced palms. The proportion of the smaller roots rotten was much higher. According to Menon and Nair (1949) rotting of roots and rootlets starts from the tip backwards. Michael (1964) recorded reduction in the number of roots produced as compared to healthy. Maramorosh (1964) noticed root rotting only in a few cases and not in all palms. Significant difference in the extent and frequency of root decay could not however, be traced by many other workers (Nagaraj and Menon, 1955; Radha and Lal, 1967; Lal, 1969). Joseph and Jayasankar (1981) did not consider root rot as a characteristic symptom of the disease as they could not observe more than 10.6 per cent root decay in diseased palms. It is significant in this context that Mathen *et al.* (1990) also did not observe root rot in the transmission experiment conducted under controlled conditions in field cages.

Anatomical studies of the leaflets revealed degenerated chlorophyll

(Shanta *et al.* 1959). Joseph and Shanta (1964) observed reduction in wall thickness of sclerenchymatous tissues, enhanced division of upper epidermal cells, increased stomata per unit area, proliferated phloem and narrow xylem.

Except tapering of the stem in a few diseased palms, no valid information on the stem anatomy is available (Menon and Pandalai, 1958).

Varghese (1934) reported cortical browning in diseased roots; but Maramorosch (1964) did not observe any discoloration in the cortex. Anatomy of the roots of diseased palms revealed degenerated phloem, disorganised tracheal elements and tylosis in metaxylem (Indira and Ramadasan, 1968; Govindankutty and Vellaichamy, 1983). Internal browning in the root vascular elements was observed by Indira and Ramadasan (1968). However, with the use of the antioxidant while severing the roots from diseased palms, Dwivedi *et al.* (1978) could not observe discoloration.

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3.1

ETIOLOGY - FUNGI

T. JOSEPH and V.G. LILY

Investigations on the association of fungi with the root (wilt) disease of coconut were started by Butler (1908) who observed the common occurrence of *Botryodiplodia theobromae* Pat. in the roots of diseased palms. He suggested that the root rotting due to this fungus could be the cause of the disease. Subsequent investigations (Menon and Nair, 1949) showed the constant association of *Rhizoctonia solani* Lutu and *R. bataticola* (Taub.) Butl. in the roots of diseased palms besides *B. theobromae*. Comparative studies on the occurrence of these two fungi in healthy and diseased areas indicated that *R. solani* being confined to the roots of disease affected palms (Radha and Menon, 1954) was more specific than *R. bataticola*. Other fungi recorded by Radha and Menon (1954) were *Neocosmospora vasinfecta* Smith, *Gloeosporium* sp., *Gliocladium* sp., *Pestalotia* sp., *Curvularia* sp., *Chaetomium* sp. and *Trichoderma* sp.

The occurrence of *Cylindrocarpon effusum* Bugn. and *Fusarium equiseti* (Corda) Sacc. in roots showing vascular discolouration was reported to be 12 to 20 per cent (Anon., 1976). Decayed roots from diseased palms had fungal hyphae in metaxylem (Govindankutty and Vellaichamy, 1983). Joseph (1978) reported occurrence of *C. effusum*, *F. equiseti*, *Monacrosporium bembicodes* (Drechsler) Subram., *Penicillium javanicum* var Beyma, *P. spiculiosporum* Lehman and *Graphium* sp. in diseased palms. *Cylindrocarpon* spp. were also isolated from burrowing nematode lesions on roots from low lying and irrigated plots (Anon., 1979).

Inoculation experiments with *R. solani* and *R. bataticola* in the field as well as in large size cement tubs brought about rotting of roots, but failed to produce the foliar symptoms characteristic of root (wilt) disease (Menon and Nair, 1951). Infectivity of these organisms was aggravated by waterlogging (Menon *et al.* 1952).

Radha and Menon (1957) reported differential behaviour of *R. solani* and *R. bataticola* with regard to their moisture requirement. While *R. solani* preferred soil moisture above 20 per cent for mycelial growth, *R. bataticola* was found to tolerate a wider range of soil moisture. Observations of Lily and Jayasankar (1974) that *R. solani* elaborated pectin methyl esterase, polygalacturonase and pectin transeliminase in culture filtrates further support the role of *R. solani* in causing the rotting of roots.

Apparently healthy roots from disease affected palms harboured *F. equiseti* and *C. effusum*. On inoculations, these fungi established infection in roots of potted seedlings maintained in sterile soil. As the infection progressed, roots of the coconut seedlings exhibited black necrotic patches on the mature parts mainly at the region of formation of breathing pores, branching of rootlet etc. There was a reduction in the

number of surface feeder roots and a majority of them were rotten. Observations on the complete root system of the inoculated seedlings indicated 20 per cent root rot in *F. equiseti* - inoculated seedlings as against 13.5 per cent in seedlings inoculated with *C. effusum*, while there was 5 per cent rotting in the uninoculated check plants (Lily, 1979; 1981 a). The percentage of rotting increased as the infection period prolonged (Lily, 1982). Lily (1983) observed the presence of toxin in culture filtrate of *F. equiseti*. Trials with *C. lucidum* also showed rotting of the root system. Extensive rotting of the tips of main roots and lateral roots was recorded in the inoculated seedlings. On an average 50 per cent of the roots were decayed in inoculated seedlings as against 5 per cent in the control seedling. The inoculated seedlings showed retarded growth and increased rotting of roots (Lily, 1981 b).

Pathogenicity trials conducted on coconut seedlings in microplots of size 1.8m x 1.8m x 1.2m using the fungi *F. equiseti* and *C. effusum* failed to reproduce the symptoms of root (wilt) disease, when inoculated singly and in combination with the burrowing nematode, *Radopholus similis* and the bacterium, *Enterobacter cloacae* (Anon., 1985).

The investigation carried out so far have not indicated any role of the fungi in inciting the disease.

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3.2

ETIOLOGY - BACTERIA

N.P. JAYASANKAR and M. GEORGE

The earliest observation in this regard was the vascular streaming movement of bacteria *Pseudomonas* sp. in the roots of root (wilt) diseased coconut palms (Srivastva *et al.*, 1969). Several attempts were made since then for the isolation of bacteria from the vascular tissue of surface sterilized root bits of diseased and apparently healthy palms. Repeated isolations failed to establish the consistent association of any major group of bacteria in the root (wilt) affected palms. The conditions were altered and stelar portions of freshly collected and surface sterilised root tips were plated in an enriched medium containing 15 per cent (w/v) coconut root extract solidified with agar (George, *et al.*, 1976). An off-white bacterium was isolated that was conspicuously absent in the coconut roots collected from root (wilt) free areas. The bacterium was identified as *Enterobacter cloacae* (Jordan) Hornaeche and Edwards (George *et al.*, 1976). The bacterium does not belong to conventional plant pathogenic genera; however, species of *Enterobacter* have been implicated in plant disease (Rohrbach and Pfeiffer, 1976; Hopkins and Elmstrom, 1977).

The ability of coconut *Enterobacter* isolates to produce polysaccharides was compared with twenty four standard *E. cloacae* cultures obtained from international culture collection centres. The crude toxin extracted from the extracellular polysaccharide like materials from the coconut *Enterobacter* isolates could wilt tomato plant cuttings in 30 min. at a concentration of 2000 ppm. This crude fraction was antigenic showing a serological relation to extracts from diseased materials. Such a relationship was not observed in the standard cultures of *E. cloacae* (George *et al.*, 1976; George, 1983).

The coconut *Enterobacter* isolates were sensitive to streptomycin and oxytetracycline group of compounds. The minimum inhibitory concentration of oxytetracycline and streptomycin as assessed by tube dilution technique showed that oxytetracycline prevented growth at a concentration of 5µg/ml (George, 1983). This observation prompted the initiation of a field experiment with a commercial terramycin tree formulation. Twenty coconut palms (10-20 years) exhibiting the primary symptoms of root (wilt) disease were indexed for intensity by the method of George and Radha (1973). The formulation containing 3 g active ingredient of oxytetracycline in one litre distilled water was injected under pressure to each of the ten experimental palms four times in 1977 (January, March, June and September), followed by a subsequent dose in May, 1979. The remaining ten palms served as controls. The change in disease index of individual palms from 1977 to 1981 is furnished in Table 4. Results indicate the

Table 4. Change in disease index in palms treated with OTC (1977-1981)

OTC treated palms	Control palms
1.2	-6.1
-0.5	-7.4
1.4	-4.4
5.8	-2.3
14.7	-11.0
-3.1	-4.0
1.9	-28.3
2.1	-10.1
6.3	-11.8
	0.0
Mean 3.31	-8.54

't' value 3.81 **

** significant at 1% level OTC - Oxytetracycline

effect of oxytetracycline in ameliorating the disease symptoms (George, 1983).

The exact involvement of the bacterium was assessed by the initiation of a large scale pathogenicity experiment. The bacterium was inoculated alone and in combination with the other biotic agents viz. *Radopholus similis, Fusarium equiseti* and *Cylindrocarpon effusum* to one year old coconut seedlings planted in microplots of size 1.8m x 1.8m x 1.2m containing sandy loam soil fumigated with methyl bromide.

The coconut isolate of *E. cloacae* failed to produce the symptoms characteristic of the disease in the pathogenicity experiment over eight years.

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3.3

ETIOLOGY - NEMATODES

V.K. SOSAMMA and P.K. KOSHY

Root degeneration to the extent of 90 per cent has been reported in root (wilt) affected palms (Butler, 1908; Menon and Nair, 1949; Radha and Menon 1954; Menon and Pandalai, 1958). Increasing evidence on soil transmissible nature of the disease and the suspected involvement of virus in the sixties indicated the possible involvement of plant parasitic nemotodes as probable vectors of the disease (Shanta *et al.*, 1972; Mathen *et al.*, 1976). Weischer (1967) examined a total of 60 soil samples covering six soil types in the diseased tract and four samples from three soil types from healthy tract and reported the occurrence of plant parasitic nematodes belonging to the genera *Criconema, Criconemoides, Dolichodorus, Helicotylenchus, Hemicriconemoides, Hemicycliophora, Hoplolaimus, Longidorus, Meloidogyne, Paratylenchus, Radopholus, Rotylenchulus, Tylenchorhynchus* and *Xiphinema* from the rhizoshphere of coconut. He concluded that the presence of species of *Xiphinema* or *Longidorus* in all soil types could be of importance if viruses were involved in the disease. While *Xiphinema* was present in both the diseased and healthy areas, *Longidorus* was found only in the diseased zone or very near the border between these two areas (Weischer, 1967). Later Khan *et al.* (1971) reported *Dolichodorus pulvinus, Macroposthonia oachirai, Discocriconemella recensi, Longidorus saginus* and *Paralongidorus flexus* from sandy loam soil around the rhizosphere of coconut at Kayangulam. The vector role of *L. saginus* and *P. flexus* in the disease need to be studied in the light of reported soil transmissible nature of the disease (Radha and Menon, 1954; Weischer, 1967; Shanta *et al.*, 1972).

Initial investigations on root (wilt) affected coconut palms showed very high populations of *R. similis* from roots of root (wilt) affected as well as healthy palms in disease tracts (Koshy *et al.*, 1975; Koshy *et al.*, 1978).

R. similis infestation produces small elongated orange coloured lesions on tender creamy white roots which leads to extensive rotting of roots. On merging of lesions, cracks develop on the epidermis of the semi-hard orange coloured main roots (Fig. 5). Lesions and rotting are confined to the tender portion of roots. Lesions are not conspicuous on the secondary and tertiary roots as they are narrow and rot quickly on infestation (Fig. 6). Tender roots of coconut seedlings on heavy infestation become spongy in texture.

Maximum number of nematodes and cavities are seen in the outer cortex. Nematodes have not been observed in the stelar region or in closely packed 4-6 layers of cells outside the strongly suberised endodermis even in heavily infested roots. The endodermis and the 4-6 layers of cells around it appear to serve as an effective

Fig. 5 Tender, white main roots of coconut with various intensities of lesions and rotting on infestation by *R. similis*

Fig. 6 Tender roots at the base of the palm showing lesions and rotting on infestation by the burrowing nematode

barrier against the invasion of the stele. In the early stage of infection, roots have cavities of independent origin separated by several cells. Consequent to nematode multiplication and lysis of cytoplasm and cell walls, adjacent cavities merged with each other. Multiple cavities and their coalescence destroy the cortex to a great extent. The stelar tube remains intact even in heavily infested roots in transverse and longitudinal sections.

In an extensive survey carried out comprising 965 samples each of soil and root from Kerala (836), Karnataka (13) and Tamil Nadu (116) during 1973-1982 the widespread occurrence of the burrowing nematode, *R. similis* on coconut was reported.

Studies on population of the burrowing nematode in coconut plantations in Kerala, show that infested coconut roots yield maximum number of *R. similis* during October to November and minimum or nil during March to July. Factors favourable for nematode multiplication are soil temperature between 23 to 25°C and moist soils coupled with availability of tender fleshy roots. Nematode population in roots of individual palms varies considerably during low and high peaks depending upon the age, variety and disease index of the palms involved (Koshy and Sosamma, 1978 a).

The pathogenicity experiments conducted at CPCRI R.S, Kayangulam have clearly established the pathogenic potential of the nematode on coconut, but the role of the nematode in the etiology of the root (wilt) disease could not be established. A detailed pathogenicity trial was initiated in October 1982. 1.8m x 1.8m x 1.2m field tanks (microplots) filled with sandy loam soil fumigated with methyl bromide were used to facilitate normal growth of the plant to flower, yield and exhibit the disease under natural conditions. The seedlings were inoculated with fungus and different levels of nematode inoculum viz. 100 to one million per seedling.

The growth parameters recorded at six monthly intervals showed reduced growth of palms corresponding to the initial inoculum levels one year after inoculation. The progressive production of number of leaves in various treatments is shown in Table 5. The seedlings that received the highest initial inoculum (T_6) had put out three leaves compared to four leaves in all the other treatments after one year. The seedlings that received 10,000 nematodes (T_4) and above had put out on an average six leaves from third year onwards compared to eight to nine leaves by seedlings that received 100 and 1000 nematodes and nine to ten leaves in uninoculated seedlings. Similar decreasing trend was seen with regard to height as well as girth at the base with increase in inoculum levels.

After eleven years initial inoculum of nematodes caused reductions in height, girth at base, root weight, number of main roots, total number of leaves, lamina length and lamina breadth.

One of the control palms put out the first inflorescence in the 31st leaf axil after 65 months of planting. All the uninoculated palms came to flowering during 65 to 83 months after planting between 31st to 49th

Table 5. Effect of different initial inoculum levels of *R. similis* on growth parameters of coconut

Treatment	Initial inoculum level (cm)	Height (cm)	Girth at base (cm)	Shoot weight (kg)	Total no. of leaves produced	No. of leaves retained	Av. lamina length	Av. no. of leaflets	Middle leaflet length	Middle leaflet breadth	No. of main roots	Root weight (kg)
T_1	0	958	146	514	100	20	364	233	108	6.5	4577	84.8
T_2	100	784	131	329	84	14	362	229	106	6.2	4091	76.4
		(14)	(10)	(36)	(16)	(28)	(1)	(2)	(2)	(5)	(11)	(0)
T_3	1,000	761	124	273	85	16	331	211	94	5.8	4611	75.8
		(21)	(15)	(47)	15)	(20)	(9)	(10)	(13)	(11)	(+12)	(11)
T_4	10,000	649	112	165	75	11	336	207	91	5.5	2834	28.4
		(32)	(23)	(68)	(25)	(45)	(8)	(11)	(15)	(14)	(38)	(67)
T_5	1,00,000	658	94	145	70	10	291	194	83	4.8	1529	15.4
		(31)	(36)	(72)	(30)	(50)	(20)	(17)	(22)	(26)	(61)	(62)
T_6	10,00,000	564	87	109	63	9	289	208	89	4.6	1529	15.4
		(41)	(41)	(79)	(37)	(54)	(21)	(11)	(17)	(29)	(67)	(82)
	G. Mean	729	116	256	79	13	329	213	95	5.5	3239	52.0
	CV%	16.11	18.1	45.6	13.6	36.0	15.3	12.3	14.5	13.5	43.21	47.8
	F. ratio	6.89**	5.76**	8.43**	7.12**	3.70*	2.11	1.55	2.4	4.98**	4.89**	7.33**
	CD (P=0.05)	153.0	27.4	151.9	14.1	6.34	NS	NS	NS	0.99	1823	32.47

Figures in parentheses are per cent reduction over control.

leaf axils whereas four out of the five palms that received an initial inoculum level of 100 nematodes flowered during 67 to 130 months in the leaf axils from 39 to 56. Two palms each that received an initial inoculum level of 1000 and 10,000 nematodes came to flowering after 108 months and one out of five palms that received an initial inoculum level of 1 lakh nematodes also came to flowering after 132 months. None of the palms that received 10 lakhs came to flowering (Table 6). The control palms produced a total of 155 inflorescences compared to 67 inflorescences in palms inoculated with 100 nematodes as initial inoculum level. However, the palms that received an initial inoculum of 1000 nematodes and above did not yield any nut even after eleven years of planting. The control palms produced an average of 125 nuts compared to 37 nuts by palms that were inoculated initially with 100 nemotodes (Table 7). This clearly showed that nematodes in soil reduced the yield under the present experimental conditions. The need for application of nematicides to infested seedlings at planting and later in July and October for proper

growth has been clearly brought out in this experiment.

This pathogenicity experiment, first of its kind on a perennial crop, has clearly brought out the damage potential of the burrowing nematode on growth, flowering and yield of coconut under field conditions over a period of eleven years from planting. The absence of production of typical root (wilt) disease symptoms even after eleven years on palms inoculated with one million nematodes clearly shows that *R. similis* is not involved as an incitant in the etiology of root (wilt) disease. The root (wilt) affected palms may decline at a faster rate on infestation by *R. similis*, which is wide spread on coconut as well as on intercrops like banana, black pepper etc. (Koshy and Sosamma, 1996).

The effect of the biocontrol agents *Paecilomyces lilacinus, Trichoderma viride, T. harzianum, Verticillium chlamydosporium, Pasteuria penetrans* and AMF on the burrowing nematode of coconut was studied in the green house. *P. lilacinus* and *V. chlamydosporium* are found to infect the eggs of the burrowing nematode.

Thirty per cent increase in yield and 5 to 10 per cent decrease in disease indices of palms affected with root (wilt) disease has been recorded by the application of *Hydnocarpus* oil cake @ 4 kg per palm as well as with phorate @ 10 g a.i/palm in June-July and October-November (Koshy, 1986). Maximum increase in yield is obtained with application of phenamiphos 2 10 g a.i./palm. The control palms, on the contrary recorded 10 per cent increase in disease indices and 2-5 per cent reduction in yield.

Table 6. Effect of different levels of *R. similis* on flowering

Treatment	Initial inoculum level	No. of palms flowered	Production of inflorescences in leaf axil	Time taken for flowering in months	Delay in initiation of flowering in months	No. of yielding palms
T1	0	5/5	41.8 (31-49)	73.8	-	4/5
T2	100	4/5	47 (39-36)	84.8	11	2/5
T3	1,000	2/5	55 (42-68)	116	42	0/5
T4	10,000	2/5	58.5 (54-63)	125	51	0/5
T5	1,00,000	1/5	68	132	58	0/5
T6	10,00,000	0/5	0	0	0	0/5

(Range in parenthesis)

Table 7. Effect of different levels of *R. similis* on yield

Treatment	Control							*R. similis* - (100)						
Replication	1	2	3	4	5	Total	Average	1	2	3	4	5	Total	Average
No. of inflo-rescences produced	61	39	26	25	4	155	31	7	32	26	2	0	67	13.4
No. of nuts produced	396	137	38	53	0	624	125	0	99	87	0	0	186	37.0

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3.4

ETIOLOGY - VIRUS

T. JOSEPH, N.G. PILLAI and J.J. SOLOMON

Virological investigations on the root (wilt) disease of coconut were initiated following the unsuccessful attempts of Menon and Nair (1951) to reproduce its characteristic symptoms through inoculation of fungal isolates including *Botryodiplodia theobromae* proposed by Butler (1908) as the causal agent. On account of the resemblance of the symptoms to those of the known plant virus disease, Nagaraj *et al.* (1954) suspected virus as the disease incitant.

The virus theory gained significance with positive transmission of the disease through sap inoculation and the insect vector, *Stephanitis typica* Distant under field conditions (Nagaraj and Menon, 1956) and under insect-proof condition. Flaccidity, paling and slight stunting of younger leaves were observed in five of the six seedlings mechanically inoculated with sap of diseased palms and in one out of six seedlings inoculated with *S. typica* (Shanta *et al.*, 1960; Shanta *et al.*, 1964).

Shanta and Menon (1960) reported crinkling and malformation in the first trifoliate leaf of 71.8 per cent cowpea *(Vigna unguiculata* (L.) Walp) mechanically inoculated on the pair of the simple leaves with crude leaf sap of diseased palms. Cowpea plants also became infected when inoculated through viruliferous lace bug, *S. typica.* Even a single insect could be infective and 16 per cent of the field population was found to be viruliferous (Joesph *et al.* 1972). Pillai, *et al.* (1970) observed that pollen, nuts, husk, kernel and embryo of diseased palms had the virus in them as inoculated cowpea developed typical symptoms.

Positive reproduction of symptoms was reported on one out of two coconut seedlings by incorporating diseased roots in the soil under insect proof conditions and in the open, four out of five seedlings grown in sterilised soil interlayered with diseased roots (Shanta *et al.*, 1964; Mathen *et al.*, 1976). Cowpea plants also showed symptoms when grown in soil collected from basins of diseased palms; in sterilised soil watered with infective leaf/root sap and also sterilised soil incorporated with infected roots (Shanta *et al.*, 1972). Cowpea plants grown in the basin of diseased palms also exhibited typical symptoms (Menon and Shanta, 1962; Pillai *et al.*, 1970).

The virus contained in the expressed sap of the diseased palm was named as 'coconut wilt virus' (Shanta and Menon, 1960). Shanta and Menon (1961) studied the physical properties of the virus using cowpea as an indicator plant. The virus had a thermal inactivation point of 76°C for 10 min. and a dilution end point of 10^{-4}. The virus was most active at pH 5 to 9 in the supernatant

solution of the extract. The longevity of the virus was eight weeks in frozen sap and three weeks at room temperature of 28-30°C at dilution of 1:2.5 (Shanta and Menon, 1961). It had a wide host range infecting many species under Leguminosae, Solanaceae and Arecae. The virus, in general caused necrosis, malformation of leaves and stunting in these hosts. Culture of the virus was maintained in cowpea through serial inoculation (Shanta and Menon, 1961).

The positive results of cowpea inoculation tests were confirmed by Holmes (1965) and Holmes *et al.* (1965). Holmes (1965), however, suggested that the sap transmissible agent may be a virus-like organism similar to spirochaete or sporozoa, in view of the peculiar nature of the symptoms on cowpea and lack of proof on its passage through bacterial filters. Production of symptom on cowpea was found to be very inconsistent in later studies. Environmental factors, especially temperature, were suspected to influence symptom expression (Anon., 1971). However, Sasikala and Pillai (1978) after a detailed study on cowpea indicated that it cannot be used as a reliable test plant.

Summanwar *et al.* (1969) isolated a virus from *Chenopodium amaranticolor* Coste & Reynier which they had infected using purified fraction from diseased coconut leaf. The purified fraction caused chlorotic lesions on *C. amaranticolor* and systemic mosaic on *Nicotiana tabacum* cv White Burley. The particles were 320-360 nm in length and 24-25 nm in width. The longevity *in vitro* was nearly one year; the thermal inactivation point 90°C and the dilution end point 10^{-5} to 10^{-6}. The virus was present in leaf and root sap of diseased coconuts but not in leaf, root sap and nut water of healthy palms. Failure of coconut leaf crude sap to infect *C. amaranticolor* was attributed to the presence of viral inhibitors (Summanwar *et al.*, 1971). The rate of infectivity could be improved by addition of 15 per cent lead acetate and 30 per cent polyclar (Summanwar and Gupta, 1976). Based on its positive reaction to antisera of three tobacco mosaic virus (TMV) strains, the virus was classified as TMV (Summanwar *et al.*, 1971). However, Shanta *et al.* (1975) through detailed pathogenicity and EM studies, indicated the non-association of TMV with the root (wilt) disease of coconut. Solomon and Sasikala (1980) also confirmed the non-involvement of TMV with the disease through serology. Maramorosch and Kondo (1977) reported the presence of icosahedral particles of 56 nm diameter. in the epidermis and ground parenchyma cells of diseased palms. But, Parthasarathy (1978) identified these particles as plasmodesmata sectioned in tangential plane.

Failure to observe any virus particle associated with the root (wilt) disease of coconut and the inconsistency in the response of cowpea to the root (wilt) pathogen presently stand out against a virus etiology for the disease.

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3.5

ETIOLOGY - PHYTOPLASMA

J.J. SOLOMON, C.P.R. NAIR, P. RAJAN, N. SRINIVASAN,
M.P. GOVINDANKUTTY, K. MATHEN and N.G. PILLAI

Coconut root (wilt) disease has long been suspected to be induced by a sub-microsocopic agent (Nagaraj *et al.*, 1954). This hypothesis gained support with the experimental transmission of the disease to test seedlings employing the insect *Stephanitis typica* (Distant) under insect proof condition (Shanta *et al.*, 1960; Shanta *et al.* 1964). Comparative histological studies on palms with different intensities of root (wilt) disease revealed disorganisation and degeneration of vascular tissues. Phloem tissues showed increased chromophily and necrotic obliteration (Govindankutty and Vellaichamy, 1983), implying a vascular limited pathogen.

The presence of Phytoplasma, earlier referred as Mycoplasma Like Organisms (MLOs), was identified in sieve tubes of roots, tender stem, petiole and developing leaf bases of root (wilt) diseased palms (Solomon *et al.*, 1983). Phytoplasmas are plant pathogenic mycoplasmas that are non-helical non-culturable and trasmitted by arthropod insect vectors (Anon., 1984).

Constant association of Phytoplasma with the disease has since been established with the finding of the organism in tissues of all the 75 diseased palms as against their total absence in an equal number of healthy palms from disease free area studied (Table 8). The palms sampled cover the various intensities of disease and different locations. The mollicutes were found in increasing numbers in the sink regions. Mature tissues exhibited fewer numbers of degenerated forms (Solomon *et al.* 1987). This is in agreement with the findings in lethal yellowing disease of coconut in Florida (Parthasarathy, 1974). Conforming to the pleomorphic nature, forms varying from circular to oval and occasionally beaded or filamentous ones were observed in sieve tubes of diseased palms. The coccoid forms were in the range of 250-400 nm; bounded by a trilameller membrane and contained well defined internal structures such as DNA strands and ribosomes (Fig 7). The walls of invaded and closely neighbouring cells were thickened, the cytoplasm granulated and often contained vesicle like structures. Phytoplasmas were observed only in sieve tubes and often found in parietal position and more frequently close to the sieve area. Distribution of the organism within the vascular bundle was sparse and not all the sieve tubes in a phloem patch contained them. Similar trend of uneven distribution was also observed in the case of lethal decline of palms in Florida and Jamaica (Thomas, 1979; Parthasarathy, 1974), in Africa (Gianotti and Dollet, 1983) and coconut stem necrosis in North Sumatra and peninsular Malaysia (Turner *et al.*, 1978). Thomas (1979) after electron microscopic examination of over 36 declining palms belonging to 21 species observed that Phytoplasma concentration in

Table 8. EM examination of root (wilt) diseased samples *

Condition of palms	Location	No. of samples tested	No. of samples with Phytoplasma
Healthy	Disease free area	75	0
Disease early	Disease affected area	40	40
Diseased middle	Disease affected area	25	25
Disease advanced	Disease affected area	10	10

* Tissues sampled : Root, rachilla, leaves in case of non-destructive sampling
Heart tissue, in case of destructive sampling.

coconut was the lowest. Failure to find Phytoplasma in all the vascular bundles in root (wilt) affected palms 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 the disease (Radha, 1979; Lily, 1981) could be observed in the vascular tissues examined .

Govindankutty (1981) reported the occurrence of phloem anomalies in both roots and pinnae of palms affected with the disease. In subsequent studies abnormal bluish colouration in sieve tubes of diseased palms following Diene's staining and increased fluorescing sites in sieve area consequent to 4,6 diamidino -2 phenylindole 2 HCl (DAPI) staining were observed (Solomon *et al.*, 1987). These histochemical staining reactions indicative of accumulation of DNA in extra nuclear sites showed the presence of Phytoplasma. Such characteristic reaction was not evident in tissues of healthy palms. Even in diseased palms positive reaction was not observed in all the sieve tubes of any phloem patch of root or every vascular bundle of rachillae. Such positive staining sites were more frequent in junctions of vascular bridges in rachillae. The occurrence of these reactions at scattered loci suggests the non uniform distribution of Phytoplasma in root (wilt) disease affected palms as corroborated in EM observation (Solomon *et al.*, 1987).

The specificity of Diene's staining (Deeley *et al.*, 1979; Razin, 1983) and fluorescence staining of DAPI (Seemueller, 1976) to bind with nucleic acid component of the phytoplasma has been well documented and is advocated as a diagnostic tool for detecting phytoplasma infection in plants (Nienhaus *et al.*, 1982). These techniques are currently being used to detect Phytoplasmal infection in root (wilt) affected palms especially the symptomless palms in the diseased tract and the disease suspects.

The constant association of phytoplasma with the disease warranted search for insect vector(s). Earlier

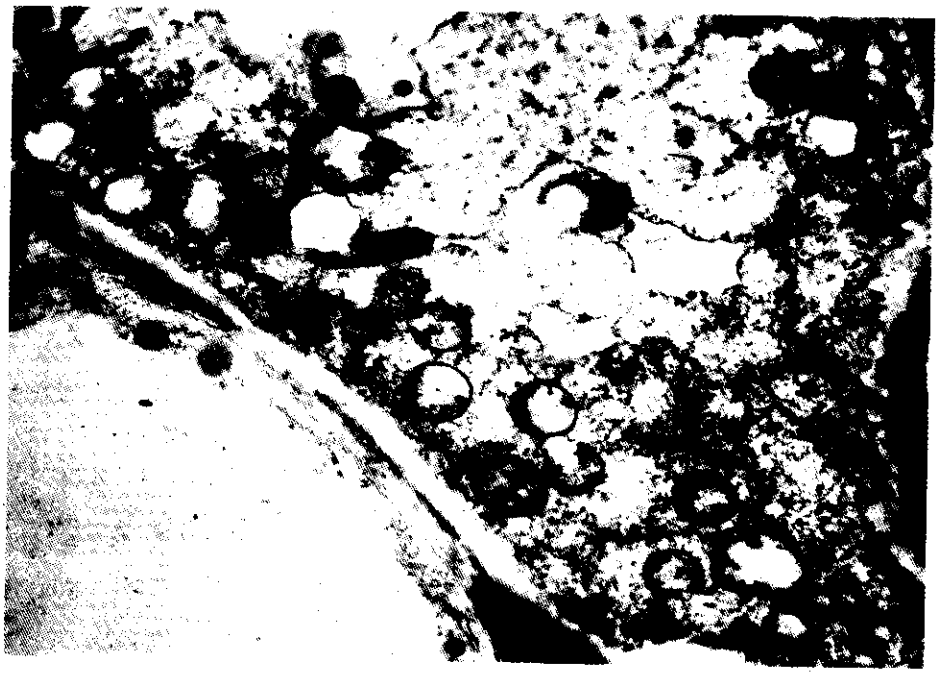

Fig. 7 Phytoplasma in sieve tubes of tender petiole from root (wilt) diseased coconut palm.

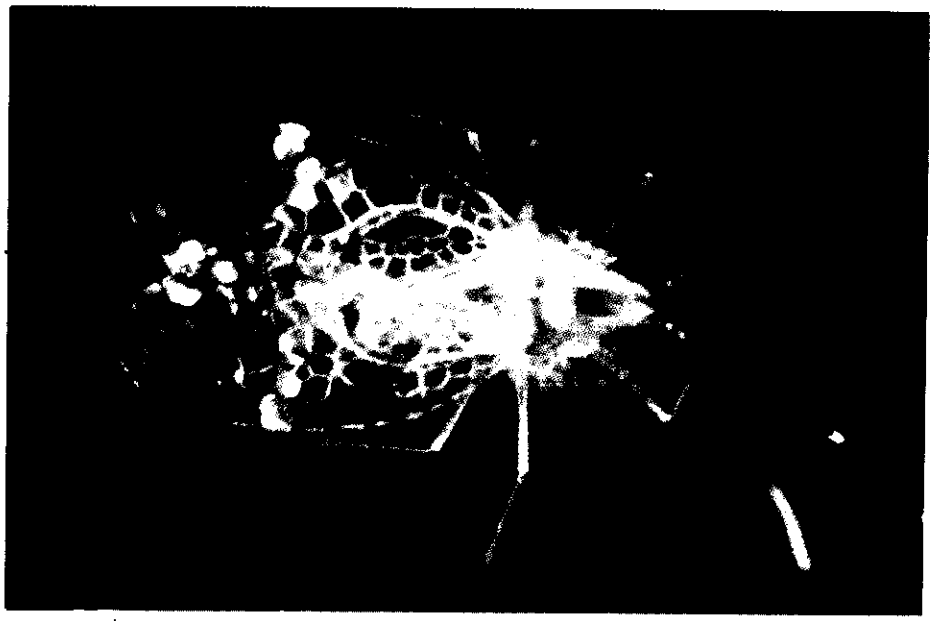

Fig. 8 Lace bug *Stephanitis typica* (Dist.)

transmission experiments (Nagaraj and Menon, 1956; Shanta, *et al.*, 1964) brought out the role of lace bug, *Stephanitis typica* (Distant) - Tingidae (Fig. 8) - the single major group of insects on coconut in the transmission of the disease. These observations were made when a viral etiology for the disease was suspected. But the report of Solomon *et al.* (1983) on the association of Phytoplasma with the disease implied a reinvestigation on the vectoral ability of the lace bug to imbibe and to transmit the phloem bound mollicute since phytoplasma diseases are not known to be transmitted by true bugs (Heteropteran insects). Phytoplasmas are mostly transmitted by leaf hoppers, plant hoppers (*Auchenorhyncha*) and rarely by psyllids. Record of insect fauna on coconut (Kurian *et al.*; 1979) however, did not include insects belonging to *Auchenorhyncha* from India. Therefore, a systematic inventory of insects in root (wilt) prevalent gardens using various traps and confirmation of their occurrence in coconut foliage by direct examination over a period of two years was carried out. As a result, besides lace bug, a leaf hopper, *Sophonia greeni* (Distant) and a plant hopper, *Proutista moesta* (Westwood) were recorded (Rajan and Mathen 1984; 1985). There was no disease occurrence independent of all the three insects. The potential of these insects to acquire the organism was verified electron microscopically. Phytoplasma was observed in brain and salivary glands of lace bug given an acquisition plus incubation period ranging from 18 to 23 days (Mathen *et al.*, 1987). Phytoplasma was not observed in lace bugs collected from disease free areas such as Kasaragod and Minicoy in Lakshadweep and also in bugs offered acquisition plus incubation periods less than 18 days (Mathen *et al.*, 1987). Phytoplasmas has also been observed in the salivary glands of *Proutista moesta* (Fig. 9) given an acquisition plus incubation period of more than 30 days on diseased palms (Anon., 1991).

In both the insects these polymorphic bodies are found in the acini of salivary glands and resembled in morphology and structure to those reported in vectors of other yellow diseases (Sinha and Paliwal, 1970; Nasu *et al.*, 1970). The morphology of Phytoplasma in root (wilt) disease affected field palms, insect salivary glands as well as brain tissues was comparable.

Although transmission of the disease from coconut to coconut through lace bug had been reported earlier (Shanta *et al.*, 1964), in the light of detection of Phytoplasma in the tissues of disease affected palms, transmission experiments were repeated under insect proof conditions (Fig. 10). Nine months after the first inoculation, coconut seedlings inoculated with lace bugs gave strong positive serological reaction in three out of four experimental seedlings and weak reaction in the fourth indicating contraction of root (wilt) disease. Light microscopy of root tissues subjected to Diene's staining, DAPI and Hoechst 33258 fluorochromes also indicated Phytoplasma infection in phloem. EM observation also confirmed the presence of Phytoplasma in all the four lace bug inoculated seedlings between 9 and 27 months after the first inoculation. By the 17th month, two of the seedlings developed flaccidity of leaflets, the diagnostic and

Fig. 9 Plant hopper *Proutista moesta* (Westwood)

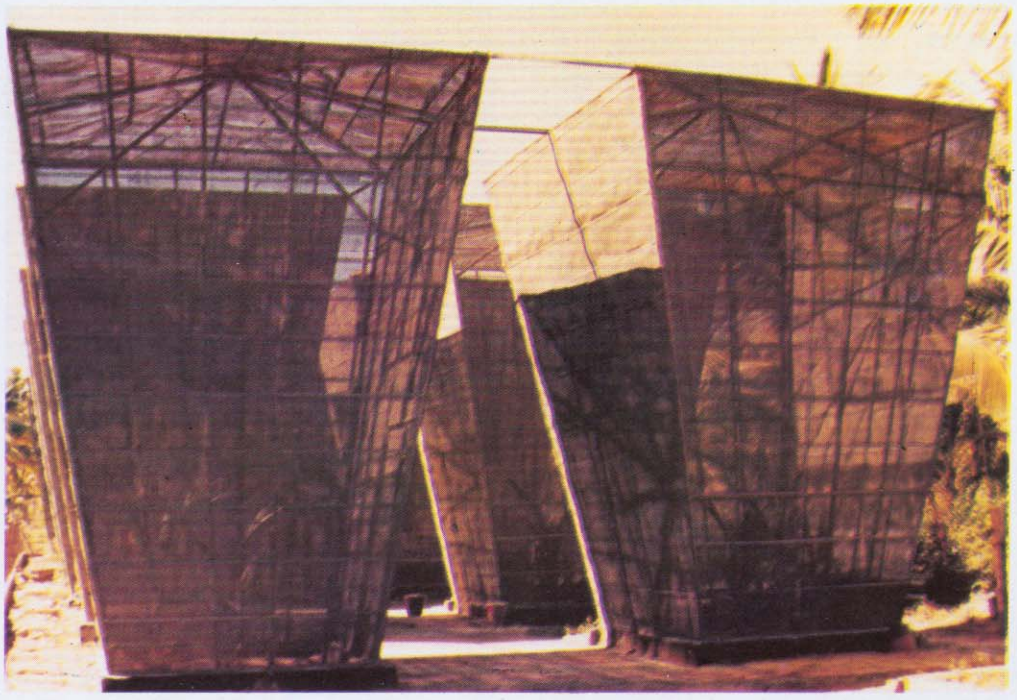

Fig. 10 Insect proof cages for transmission experiment

decisive symptom of the disease (Mathen *et al.*, 1990). However, in control seedlings there were no symptoms and no Phytoplasma was observed either.

Apart from the direct evidences accrued on the vectoral role of lace bugs, a number of indirect evidences also lent support. Lace bugs were found colonising in increasing number towards the inner leaves (Mathen *et al.*, 1969). This pattern of distribution enhances the chances of the organism being acquired more efficiently by the bug since active forms of Phytoplasma in higher concentration were found in tender tissues. It was also reported that the number of lace bugs in diseased palms was four times more than that in symptomless palms (Mathen, 1982). Monitoring lace bug population for two years, Mathen (1985) reported a direct linear correlation between the number colonising the palms and percentage of fresh incidence of disease. Transections of coconut pinnae with lace bugs fixed in feeding position by a cold immobilisation technique revealed the termination of the stylet in phloem, thereby confirming the ability of the insect to pick up the phloem delimited organism (Mathen *et al.*, 1988).

Although, tingids as a group are not conventional mycoplasma transmitters, instances of tingids being vectors are encountered in literature. Sugarbeet savoy disease now recognised as of Phytoplasma etiology (Haris, 1979) is transmitted by *Piesma cinereum* (Say) and sugarbeet latent rosette, a rickettsia-like organism (RLO) induced malady transmitted by *P. quadratum* Fieb. (Proesler, 1980). These vectors belong to Piesmidae, taxonomically very close to Tingidae. The above cited direct and indirect evidences together with the finding of Phytoplasma in the salivary gland and brain tissues of lace bug and transmission of the disease from diseased to healthy coconut seedlings through the bug confirm the insect being a vector of the disease. The vector role of the plant hopper was also experimented in two year old WCT coconut seedlings, under insect proof condition. Phytoplasma was observed in six out of eight plant hopper inoculated seedlings 5-24 months after inoculation. Five of the seedlings exhibited the diagnostic symptom of the disease thus confirming the vector role of the plant hopper (Anon., 1997).

Results of the field experiments on the effect of vector control through foliar/ soil application of insecticides and plant products on the incidence of the disease clearly indicated the disease contraction even in plants sprayed at fortnightly intervals with ten times the normal recommended concentration of the insecticide (Anon., 1993; 1997). Similar results were also reported in lethal yellowing disease in Florida (Howard and McCoy, 1980; Howard and Barrant, 1989). This suggests that prevention of disease incidence through insect control is not a practical proposition.

Experimental transmission of Phytoplasma was attempted also employing certain phanerogamic parasites to periwinkle, *Catharanthus roseus* G. Don, a known mycoplasma indicator host. Dodder species, *Cuscuta campestris* Yunck., *C. chinensis* Lam.

and *C. subinclusa* Dur and Hilg. although established on coconut foliage, failed to put efficient haustorium to reach the coconut leaf vascular bundle. Tsai (1983) also failed in his attempts to transmit the lethal yellowing disease from coconut through *C. campestris*. A dodder laurel, *Cassytha filiformis*, however, established well on coconut putting forth haustorium to reach and form intimate contact with the vascular bundles. Periwinkle grown in sterilized soil in mud pots and protected inside insect-proof muslin cloth cages bridged through dodder laurel established on diseased coconut seedlings in the field, developed chlorotic spots in the interveinal areas at vein endings of fully opened leaves, three to four weeks after the establishment of the haustorium. Passage of Phytoplasma as confirmed by positive staining reactions and detection of the organisms through electron microscopy in the midvein and petiolar tissues of the periwinkle, dodder strands and leaflets of diseased coconut seedlings, established the transmission of the disease from coconut to periwinkle. Phytoplasma was however not observed in dodder on disease free coconut and control periwinkle plants (Sasikala *et al.*, 1980). 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 *et al.*, 1985) this is the first instance of Phytoplasma being transmitted by an unconventional dodder species.

Though the disease could be experimentally transmitted through the lace bug to healthy coconut palms and through the dodder to periwinkle, culturing of Phytoplasma *in vitro* is considered to be one of the pre-requisites to prove the pathogenicity of the organism. Phytoplasmas being restricted to the specialised vascular environment of phloem, a medium simulating the physico-chemical environment of the phloem may be necessary for successful culturing of the organism in cell free medium. Phloem sap which is 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; Mc Coy, 1977) and *Phytomonas davidi* (Mc Coy, 1978).

Rajagopal *et al.* (1988) standardised a method for collection of vascular sap in ice packed vacuum flasks. The physicochemical condition of vascular sap from apparently healthy and diseased palms has since been analysed. The analysis of sap also gave an insight into the type of Phytoplasma found associated with the disease. Sap from diseased palms had higher arginine level than that in healthy palms. It is suspected that, the Phytoplasmas present in root (wilt) diseased palms may be of the non-fermentative type, which uses the arginine through dihydrolase pathway for its energy production (Chempakam and Rajagopal, 1989).

The vascular sap collected from apparently healthy palms was filter-sterilised and used as such or supplemented with growth factors for the preparation of culture media. In addition, about 40 different media with various combinations of growth factors,

nucleic acid precursors, co-factors, vitamins etc. were used for the culturing of the organism from tissues of diseased coconut, symptomatic periwinkle and infective lace bugs adopting a number of methods. Embryo-nated hen's eggs were also employed. However, the organism could not be cultured in any of the media (Anon., 1989). Currently, attempts are being made to maintain/propagate the root (wilt) phytoplasma in explants from diseased palms. Phytoplasma could be maintained in rachillae expants from diseased juvenile coconut palms for more than 6-8 weeks in certain plant tissue culture media (Anon., 1989). Co-culturing of dodder laurel with embryo cultured coconut plantlet for attempting *in vitro* transmission is also in progress.

Various methods of application such as ring barking, root feeding, gravity flow and stem injection with pneumatic pressure injection device were tried (Pillai and Raju, 1985). The pneumatic injector was found to be superior to all other devices/methods as the antibiotic injected with this could be detected in sufficiently high concentration in the foliage within 24 hr of application. Residue analysis of the antibiotic in root tips, un-opened leaves and nuts of the injected palms eventually revealed the retention of the chemical in the foliage for more than 12 weeks with the concentration petering out to minimum with the passage of time (Chowdappa *et al.*, 1989)

A field trial was initiated in 1984 with four concentrations (1,2,3 and 6g ai) of Oxytetracycline hydrochloride (OTC

Fig. 11 Antibiotic therapy : OTC-treated palm

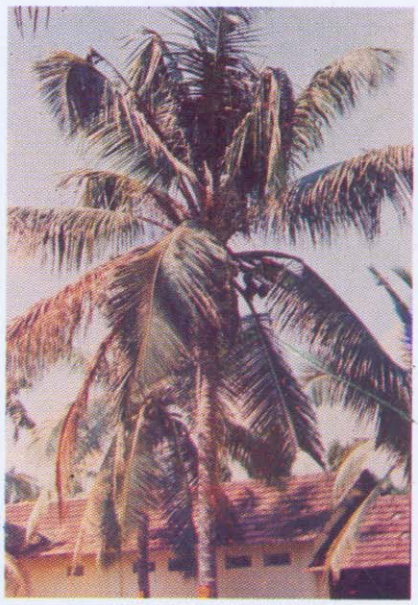

Fig. 12 Antibiotic therapy : Dist. water control

Terramycin Tree Formulation of M/s Pfizer India Ltd.) a single concentration each of Neomycin, Penicillin and distilled water control. Fifteen palms each in the early stage of disease were given the different treatments at quarterly intervals. Fifty three palms treated with 3 and 6/g ai of OTC showed remission of symptoms. Contrastingly, palms in the distilled water (Figs. 11 and 12) and penicillin treatment deteriorated significantly over the pre-treatment condition (Pillai *et al.*, 1991). Thus, the remission of symptoms in OTC treated palms adds further evidence to the etiological role of Phytoplasma in coconut root (wilt) disease.

Non-cultivable nature of phytoplasma is a limiting factor in fufilling Koch's postulates. Nevertheless manifestation of disease symptoms through inoculation of insect vector(s) rendered infective and differential chemotherapeutic response of diseased palms to pencillin and tetracycline are considered adequate to offer the best circumstantial evidence to prove the phytoplasmal etiology.

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4

DIAGNOSIS

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The coconut root (wilt) disease is identified on the basis of visual symptoms. The earliest visual symptom of the disease reported was in 18-month-old palms (Anon., 1981). However, the time lag between infection and manifestation of symptoms vary considerably (Nagaraj and Menon, 1956; Shanta *et al.*,1964; Mathen *et al.*; 1976). Attempts were made to develop reliable diagnostic tests which could detect the palms at very early stages of infection even before the visual symptoms are apparent.

A colour test developed based on differential dehydrogenase activity of leaf tissues was not useful in the diagnosis of the disease (Joseph and Shanta, 1963). Accumulation of free amino acids especially arginine and other ninhydrin positive free amino acids in the tender leaves of diseased coconut palms indicated the possibility of developing a colour test. However, this again gave inconsistent results under varying environmental conditions (Pillai and Shanta, 1965).

A third test based on tannin content was tried following the observation that diseased palms in general had low tannin content in the leaves, although tannin or similar colouring substances get gradually depleted as disease progresses (Lal, 1968). The change was not so marked on the onset of the disease to use it as a diagnostic tool.

A collaborative project of the Indian Space Research Organisation and the Indian Agricultural Research Institute with NASA of U.S.A. was undertaken for the early detection of root (wilt) disease by Remote Sensing Technique using false infra-red aerial photography. The findings by and large indicated that the crown of healthy palms appeared red and those of diseased palms showed paleness as a result of weaker infra-red reflectance on the film as measured by microphotometer. This method could not be used as a diagnostic tool for want of adequate data on ground level (Dakshinamurthy *et al.*, 1971; Dakshinamurthy and Summanwar, 1972).

A biochemical test to detect root (wilt) disease of coconut was developed using ethylene diamine tetra acetic acid (EDTA) as extractant of biologically active organic constituents/pigments present in the diseased palms (Dwivedi *et al.*, 1977). But, this did not give consistent results (Rajagopal *et al.*, 1988).

All these biochemical tests investigated so far were based on altered host metabolism perceptible in the form of either accumulation or depletion of substances consequent to differential enzymatic activity which could be induced under varying conditions. Shanta (1971) observed that agglutination tests were unreliable with coconut leaf and root extracts because of non-specific reaction obtained

with normal serum proteins. Similarly, non-specific reactions were obtained with diseased coconut leaf extracts against all the tested antisera in Ouchterlony's double diffusion test.

A sero-diagnostic test developed by Solomon *et al.*, (1983) and a physiological test standardised by Rajagopal *et al.*, (1986) proved to be more consistent in detecting the disease much before the visual symptoms appear.

The Agar Gel Double Diffusion Test (Solomon *et al.*, 1983) could be used to detect the disease in palms with certainty irrespective of their age group and soil type. The antiserum is specific to root (wilt) disease and it doesn't react with samples from healthy and budrot affected palms (Fig. 13).

Common antibodies from the root (wilt) antiserum were removed by intragel cross absorption technique (Regenmortel, 1967). Since the technique is time-consuming (about 96 hours), a rapid method has been standardised on the basis of serum cross absorption. Total protein isolated from the spear leaves of healthy palms was used for absorbing host antibodies from root (wilt) antiserum. The cross absorbed antiserum could be used for screening samples. By this techique 50% saving in time could be achieved (Anon., 1985a). The test is found to be in agreement with visual identification of diseased palms upto 95.3% (Table 9).

The severity of the disease also could be determined based on the intensity of reaction, the intensity being highly pronounced in the early stage of disease followed by a fall in the intensity as the disease progresses. In all tests irrespective

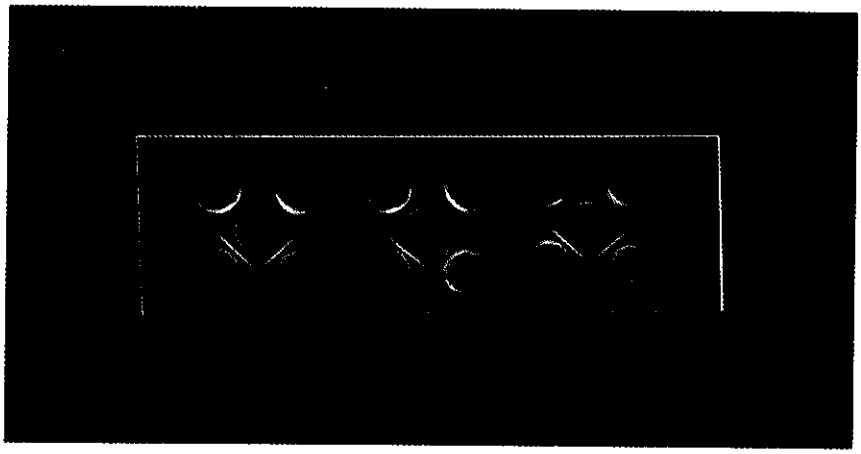

Fig. 13 Double diffusion test

Centre well	**:**	**Root (wilt) antiserum**
Upper well	**:**	**Leaf extracts from healthy palms**
Lower well	**:**	**Leaf extracts from diseased palms**

Table 9. Serological reaction of samples from diseased palms

Year	No. of samples tested	No. of samples with positive reaction
1976	161	158
1977	26	25
1978	30	30
1979	38	38
1980	43	41
1981	--	--
1982	51	50
1983	35	34
1984	92	79
1985	44	42
Total	520	497
Percentage		95.3

of the soil type in which palms are being grown, the antiserum reacted against diseased samples (Table 10). It has been shown based on serological test

Table 10. Precipitin reaction of root (wilt) antiserum against healthy and diseased palms from different soil types

Condition of palm	Soil type	No. of samples tested	No. of samples reacted
Healthy	Laterite	90	Nil
"	Alluvial	60	Nil
"	Sandy loam	58	Nil
"	Clayey	40	Nil
Diseased	Laterite	112	108
"	Sandy loam	921	898
"	Reclaimed Sandy loam	58	56

(Anon., 1985 a) that the time lag between the detection of latent stage of the disease and manifestation of visual symptoms varied from 6 to 24 months.

Enzyme Linked Immunosorbent Assay (ELISA), a more sensitive and rapid diagnostic test, has been standardised for the quick detection of root (wilt) disease (Anon., 1996). Plate coated indirect ELISA is performed with crude leaf extracts of coconut, root (wilt) antibody, Horse radish peroxidase as enzyme conjugate and tetra methyl benzidine as the substrate. Samples of diseased palms recorded four times higher absorbance value over healthy samples. The test could be completed within 44 h and can be used to screen atleast 36 samples at a time using microlitre quantity of antiserum. The serological test is extensively used for confirming the health status of apparently healthy high yielding palms identified in the hot spot area of Alappuzha, Kottayam, Kollam and Pathanamthitta districts. Of the 2304 samples disease status (Table 11).

The stomatal resistance and transpiration rate of last fully opened leaf next to the spear leaf were determined with Li-Cor 1600 steady state porometer (Rajagopal *et al.*, 1982; Rajagopal *et al.*, 1986). Studies on diurnal fluctuations (6 to 18 hrs) and seasonal variations (dry and wet) in the stomatal resistance and transpiration revealed that the determinations of these parameters during the mid-day in the dry season to be the best for distinguishing the diseased palms from the apparently healthy ones. There was high stomatal

Table 11. Serological reaction of samples from elite palms

Year	No. of samples tested	No. of samples with positive reaction
1986	170	69
1987	30	9
1988	56	30
1989	50	18
1990	34	15
1991	177	33
1992	174	49
1993	222	44
1994	233	117
1995	278	173
1996	525	243
1997	190	54
1998	165	101
Total	2304	955

resistance with a correspondingly low transpiration rate in the apparently healthy palms whereas diseased palms exhibited low stomatal resistance and high transpiration rate (Table 12).

Based on the characteristic changes in the leaf water potential components of different whorls of leaves between the apparently healthy and root (wilt) diseased palms (Rajagopal *et al.*, 1987), the determination of leaf water potential was also found to be useful as a diagnostic technique for early detection of the disease (Rajagopal and Amma, 1989). The leaf water potential was lower in clearly diseased palms than in apparently healthy palms. However, there were symptomless palms which had the leaf water potential similar to diseased

Table 12. Stomatal resistance and transpiration rate in the first fully opened leaves (Rajagopal *et al.*, 1986)

Season	Status of disease	Stomatal resistance $sec.cm^{-1}$	Transpiration rate $\mu g.cm^{-2}\ S^{-1}$
Dry	Apparently healthy	14.33	0.053
	Diseased	4.52	0.189
Wet	Apparently healthy	1.82	0.233
	Diseased	1.58	0.300

palms and hence were 'suspected' to be diseased. All such suspected palms developed the foliar symptoms in about 14 months.

A comparative study was undertaken between the serological and physiological tests. At the start of the experiment 19 palms had visual symptoms of the disease and reacted positively to serological test and showed low stomatal resistance and low leaf water potential (Table 13). The foliar symptoms were absent in the other 25 palms, out of which only 9 palms turned out to be free of latent infection (at that given time) based on serological negative reaction and high stomatal resistance indicative of healthy nature of palms (Rajagopal *et al.*, 1987). There were 16 palms which did not show foliar symptoms but had positive serological reaction and low stomatal resistance and high water potential. These 16 palms were designated as disease suspects.

Table 13. Serological reaction (spindle leaf) and stomatal resistance and leaf water potential (LWP) (middle leaf) from a total of 44 palms

No. of palms observed	Visual symptoms	Serological reaction	Stomatal resistance sec.cm-1 (range)	LWT Bars	Remarks
19	Present	Positive	2.3-3.9 0.13	-2.15	Diseased
9	Absent	Negative	5.8-9.1 0.17	-1.64	Apparently healthy
16	Absent	Positive	2.6-4.4	-2.24 0.28	'Suspects'

Table 14. Appearance of visual symptoms in palms subjected earlier to diagnostic tests

	No of palms with disease symptoms in months after tests.						
No. of palms observed	2	4	6	8	10	12	14
16 Disease Suspects	Nil	Nil	Nil	Nil	3	9	14
09 Apparantly Heathy	Nil	Nil	Nil	Nil	Nil	3*	1*

* These four palms had shown low stomatal resistance and LWP between 2nd and 3rd month.

The development of disease symptoms was monitored regularly in both 'suspects' and apparently healthy palms for 18 months. Table 14 shows the time taken for the appearance of visual symptoms. While it took 10 months for three disease suspect palms to exhibit the symptoms within the next two to four months (i.e. total of 12 to 14 months) the remaining palms, nine and four respectively, also had developed the characteristic symptoms of the disease. The disease index of these palms ranged from 15% to 31% i.e. early to middle stage of the disease. The fact that four of the apparently healthy palms had also contracted the disease in about 12 to 14 months preceded by low stomatal resistance and water potential reveal that periodic diagnosis of palms would be beneficial in ascertaining the latent infection of the disease.

It is thus clear that the two parameters of water relations and sero-diagnostic test could compliment each other in detecting the diseased palms prior to manifestation of visual symptoms.

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5

SOILS AND NUTRITION

S.R. CECIL and P.G.K. AMMA

The coconut root (wilt) disease has been found to occur on all soil types of Kerala under varying ecological conditions ranging from foot hills of the Western Ghats to the coastal plains (Pillai *et al.*, 1973). Investigations on soil conditions and nutritional factors associated with the disease were initiated in 1939 (Menon and Nair, 1949). Menon and Nair (1949), Menon *et al.* (1950) suggested that the disease might also be associated with nutritional deficiencies. They reported that the soils of disease affected areas were generally deficient in major nutrients, particularly K, and had a lower content of exchangeable cations and a low pH and cation exchange capacity (CEC). The silica/sesquioxide ratio was reported to be higher.

Soil sickness characterised by low pH, inadequate activity and nutrient imbalances together with mineral deficiencies, probably those of K, Ca and Mg were reported to have a decisive role on the incidence of the disease (Menon *et al.*, 1950, 1952; Pandalai *et al.*, 1958a, 1958b; Menon, 1961; Verghese, 1961; Lal, 1964; 1968). An intensive study of the major soil groups of erstwhile Travancore Cochin State representing healthy and diseased pockets was conducted by Sankarasubramoney *et al.* (1954, 1955, 1956) and Pandalai *et al.* (1958a, 1958b, 1959a, 1959b). Their studies showed that in general, the soils in disease affected areas were low in available K, total Ca and Fe, exchangeable Ca and Mg, total exchangeable cations, CEC, pH and percentage base saturation. Waterlogging was found to favour disease incidence and majority of the diseased areas had a high water table. Cecil and Verghese (1959) observed that the reduction products formed in soils under waterlogged conditions were not responsible for disease incidence. Verghese (1966) indicated the association of faulty nutrient ratios in soils, particularly K/Mg, K/Ca and N/K with disease incidence. Pillai and Pushpadas (1965) observed that coconuts growing on Kari tracts (peaty soil) having high acidity, often in the pH range of 3-4, had less incidence of disease.

Pillai *et al.* (1975) carried out a nutritional survey of all the districts of Kerala and studied the major and micro-nutrient status of all soil groups of the State. There was no consistent difference in major and micro-nutrient status of soils between healthy and diseased areas. However, the total N status was generally higher and the available N status lower in the diseased tracts. Verghese (1959b) suggested that probably the minerological composition of rocks and some toxic products of weathering could be responsible for the disease and the possibility of water acting as their carrier. Biddappa and Khan (1985) studied the heavy metal

status of coconut growing soils of Kerala and found that the contents of Diethylene triamine-penta acetic acid (DTPA) extractable Ba, Cr, Cd, Pb, Sr and V were significantly higher in diseased soils as compared to healthy.

Menon and Nair (1952) were the first to examine the major nutrient status of leaves in relation to the disease. Subsequent studies by Sankarasubramoney *et al.* (1952), Verghese *et al.* (1959a), Pillai (1959) and Pandalai (1959) showed that there was a tendency for N, P and K to get accumulated in the leaf tissues of diseased palms and the accumulation increased with the advancement of the disease. Compared to healthy, the diseased palms contained more of N, P, K and silica to the extent of 5.0 to 13.0; 0.0 to 13.0; 5.0 to 39.0 and 59.0 to 134.0 per cent respectively (Verghese *et al.*, 1959a). Cecil (1969) suggested that the nutrient accumulation was only apparent, possibly the result of a low dry matter content of leaf tissues consequent on disease incidence.

Verghese *et al.* (1957, 1959b) ruled out the possibility of Cd and Sr toxicity in the disease complex. Cecil (1975) found that the N, P and K contents did not differ between healthy and diseased palms in the early stage of infection. But Ca and Mg contents of healthy palms in disease free areas were significantly higher than those of apparently healthy or diseased palms in the affected tracts. He also reported that the palms in the diseased areas were in a state of imbalanced nutrition with wider ratio of N/Mg, P/Mg, K/Mg and Ca/Mg indicating a lower content of Mg in proportion to other major nutrients. A comprehensive study on the nutritional factors of the disease by Pillai *et al.* (1975) indicated that the palms in the disease affected areas, whether apparently healthy or visibly diseased, were in a state of imbalanced nutrition, possibly the result of a relatively higher content of N, P and K on the one hand and a lower content of Ca, Mg and S on the other.

Khan *et al.* (1985) did not observe any relationship between the micro-nutrient composition of diseased palms and the disease index compared to healthy palms. Biddappa and Cecil (1984) and Biddappa (1985) studied the deposition of heavy metals in the root and cabbage tissues respectively, of diseased palms by employing scanning electron X-ray microprobe analyser. High deposition of Al, Mn, Cu and Co in the diseased roots and Cr, Ti, Pb, Bi and Ga in the cabbage tissues of diseased palms were also observed compared to healthy tissues. This was also confirmed by the chemical analysis of a large number of soil and tissue samples under identical conditions (Table 15).

Table 15. Heavy metal concentration in cabbage tissues of healthy and root (wilt) diseased palms (Biddappa, 1985)

Condition of the palms	Heavy metal concentrations (µg/g)				
	Bi	Cr	Pb	Ti	Ga
Healthy	7	6	tr	12	10
Diseased	19	34	2.8	17	29

Biddappa (1984) also observed higher contents of heavy metals in the crown of diseased compared to healthy palms. Wahid *et al.* (1983) studied the non-nutrient elemental composition in soil (0-30 cm) and plant tissues of healthy and root (wilt) diseased palms from a few selected locations employing energy dispersive X-ray fluorescence technique and found that Ni and Sr were present at a higher concentration in the root of diseased palms compared to healthy.

The first field fertiliser trial conducted at Kayangulam (Menon and Nair, 1952) indicated that N and K were the limiting factors in the disease affected gardens. John and Jacob (1959) reported that in disease affected areas of West Coast, NPK application along with fungicides and insecticides markedly increased the yield. Nair and Radha (1959) and Lal (1964) reported reduction in the foliar yellowing and increase in yield of diseased palms by applying NPK, lime and farm yard manure and spraying with Bordeaux mixture, micro-nutrients and magnesium. Sahasranaman *et al.*(1964) found that application of NPK fertilizers higher than the optimum dose generally aggrevated the symptoms and reduced the yield of diseased palms while lower levels helped to maintain an economic yield. The results of a trial with three levels of NPK, two levels of Ca and Mg on diseased palms showed that the lowest level of NPK tried viz., 350-300-600 g along with 500 g MgO per palm per year could be the economic dose for the management of diseased palms (Anon., 1981). Application of lime and ash (Chettiar *et al.*, 1959) and Chilean nitrate (John *et al.*, 1959) showed no positive effect on the disease. Continued application of three levels of N (highest level - 1.362 kg N), P (highest level - 0.908 kg P_2O_5) and K (highest level - 2.724 kg K_2O) in factorial combinations was not effective in either curing the disease or preventing fresh incidence on young healthy palms (Cecil, 1969).

Davis and Pillai (1966) and Davis (1969) reported that the application of micronutrients and Mg did not prevent fresh incidence of disease. However, Mg application had decidedly a favourable response on the yield of diseased palms. Similar responses of Mg treatment on diseased palms were also reported by Varkey *et al.* (1979); Cecil (1981) and Anon. (1981). Concluding his nutritional studies, Cecil (1981) recommended the application of Magnesium for the management of diseased palms. The pre-bearing age was reduced by 9 months by the addition of Mg, and the response of Mg was more pronounced on diseased palms compared to healthy ones. Lal (1968) reported that the yellowing associated with the disease might be largely due to Mg deficiency. Cecil (1981) and Cecil *et al.* (1982) based on field fertility trials concluded that the disease was not caused by deficiency of any major nutrients. Application of sulphur, calcium sulphate and magnesium sulphate along with NPK was found to increase the yield of affected palms (Lal, 1964). The foliar yellowing associated with the disease decreased markedly when the palms were sprayed with 2.0% magnesium sulphate solution at quarterly intervals (Anon., 1966; Varkey *et al.*, 1979).

The nutritional requirement of Chowghat Orange Dwarf (COD) x West Coast Tall (WCT) hybrids in the root (wilt) affected area was investigated by Amma *et al.* (1982) The dose of 500 g N, 300 g P_2O_5 and 1000 g K_2O along with 500 g MgO per palm per year could be taken as ideal for optimum productivity of the hybrid under rainfed conditions. The seedlings which received fertilizers, started yielding 3 years earlier than those grown under control (Table 16). A comparative study on the performance of WCT (Cecil, 1981) and CODxWCT (Amma *et al.*, 1982) under rainfed conditions and regular fertilization with N, P, K, Ca and Mg, since field planting shows that the hybrid was superior to WCT with respect to reduced disease incidence and increased nut yield (Table 17).

Zinc and Mo both as soil application and foliar spray had no effect on incidence or intensity of the disease, even though the tissue levels of Zn and Mo increased considerably (Mathew *et al.*, 1986).

A systematic micronutrient manurial experiment consisting of all combinations of two levels each of Fe, Mn, Cu, Zn, B and Mo since field planting had shown that the disease was not related to micronutrient nutrition of the palm (Anon., 1986).

Table 16. Effect of fertilizers on precocity and yield of hybrid palms in root (wilt) affected areas (Amma, P.G.K. unpublished)

Year after planting	Mean yield of nuts/palm/yr. under different treatments					
	(NPK)*$_1$	(NPK)*$_2$	(NPK)*$_3$	Mean of (NPK)	FYM only	Absolute control
5th	58.9	64.5	44.2	55.9	Nil	Nil
6th	97.5	106.3	98.8	100.9	18.3	Nil
7th	80.3	79.2	68.1	75.9	20.8	Nil
8th	87.7	90.3	95.4	91.1	19.9	10.1
9th	112.5	110.2	92.3	105.0	44.9	26.7
10th	77.0	55.2	53.4	61.9	46.4	27.7
11th	112.8	107.8	89.6	103.4	50.1	29.8
12th	33.5	19.8	26.5	26.6	34.9	24.1
13th	151.2	130.5	130.7	137.5	92.9	59.0
Mean	90.1	84.9	77.7	84.2	34.2	24.2

* (NPK)1 = 0.5 kg N, 0.3 kg P_2O_5 and 1.0 kg K_2O /palm/year

* (NPK)2 = 1.0 kg N, 0.6 kg P_2O_5 and 2.0 kg K_2O/palm/year

* (NPK)3 = 1.5 kg N, 0.9 kg P_2O_5 and 3.0 kg K_2O/palm/year

12th year (1984) - after the unprecedented drought in 1982-83.

Table 17. Comparative performance of COD x WCT and WCT in root (wilt) affected area. (Cecil 1981; Amma *et al.*, 1982)

Year after planting	Cumulative disease incidence (%)		Nut yield/palm (Nos.)	
	COD x WCT	WCT	COD x WCT	WCT
4th year	1.8	2.2	-	-
5th year	3.6	4.3	55.9	-
6th year	5.0	8.8	100.9	-
7th year	5.0	22.5	75.9	16.8
8th year	8.9	29.3	91.1	39.6
9th year	22.1	35.5	105.0	49.4
10th year	22.8	42.6	61.9	44.8
11th year	40.9	50.7	103.4	62.9
* 12th year	41.1	62.2	26.6	52.1
Cumulative yield/palm upto the 12th year.			620.7	265.6

* 12th year (1984) - after the unprecedented drought in 1982 - 83.

The effect of slow release fertilizers in coconut growing soils of root (wilt) affected area was evaluated by Amma *et al.* (1993). The results of the study revealed that slow release N source are superior to ordinary untreated urea for gradual supply of nitrogen to the soil. When urea mixed with indigenous materials like coir dust, neem cake etc ammonification/nitrification was taking place at very low pace, resulting in higher status of NH_4-N/NO_3-N in the soil for a longer period.

The field experiment to study the effect of different organic manures on the growth and productivity of root (wilt) affected palms is in progress (Anon., 1996). The study revealed that by application of organic manures alone or in combination with inorganic fertilizers, the incidence of root (wilt) disease could not be controlled/ arrested. By the end of fifth year of planting 21.8 percent of the palms had contracted the root (wilt) disease. However the growth parameters of the palms like height, girth at collar, number of leaves produced/year, length of the oldest functioning leaf were found to be significantly higher under the treatment of organic manures along with inorganic fertilizers.

Valiathan *et al.* (1992) reported lower level of magnesium and higher concentration of cerium in the leaves of root (wilt) diseased

coconut palms than in the healthy - looking palms. From the Table 18 it is clear that the element lanthanum also showed higher levels in the diseased than in the healthy palms. According to the authors, the reciprocal enhancement of cerium, the most bioactive member of the lanthanide series, is consistent with the synergistic role of magnesium deficiency which increases the cytotoxicity of metals by various mechanisms, including the increase in membrane permeability. They could visualize the similarity in the reciprocal relationship between magnesium and cerium in the diseased palms and the relationship in the cardiac tissues of patients with endomyocardial fibrosis.

Besides the macro and micronutrients, rare earth elements (REEs) like lanthanum (La), Cerium (Ce), Praseodymium (Pr), Neodymium (Nd), Samarium (Sm) and Gadolinium (Gd) were also estimated in the root (wilt) diseased, apparently healthy and healthy palms (Wahid, 1998 personal communication). Among these, the foliar levels of Gd were significantly less in palms of the disease affected tract than in palms of the disease free tract, indicating the deficiency of Gd in the disease endemic areas. This was observed in pooled analysis and in the soil type-wise analysis of laterite soil. The author had also reported significantly less ratios between nutrient - REEs namely Ca/Ce, Mg/Ce, Mn/Ce, Zn/La and Zn/Ke in palms growing on alluvail soil in the disease affected tract than in healthy palms growing on the same type of soil in the disease free tract. According to the author the diseased tract and disease free tract are geochemically different and perhaps it is due to this, that the disease is confined to a particular region.

Table 18. Elemental concentration in the palms after Valiathan *et al.*, 1992

		Healthy looking controls			
Element	Diseased (n=30)	Kollam and Alappuzha (n=30)	Bombay (n=3)	Manavalakurichi (n=10)	Statistical significance
Mg	252±95.7	261±94	336.7±162	377.4±80.1	DG lower than 3 (P<0.001).
Ce	856±320	622±215	16.9±5.8	195.7±81.4	DG higher than 1, 2 and 3 (P<0.001). 1 higher than 2 and 3 (P<0.001).
La	476±170	359±116	<2	70.4±29	DG higher than 2 and 3 (P<0.001)

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6

PHYSIOLOGY AND BIOCHEMISTRY

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Diseased plants generally exhibit impaired metabolism. A number of physiological and biochemical derangements occur consequent on disease development. In root (wilt) affected palms also derangements were noticed in the root functioning, water relations, mineral nutrition, respiration and photosynthesis, phenol metabolism and similar other processes.

Michael (1964) studied the extent of root damage caused by root (wilt) disease in two to ten-year-old palms and reported that the number of functioning roots and the diameter of the bole (root forming portion of the stem) were drastically reduced in diseased palms. In diseased palms, the percentage of dead roots was higher and the regenerating capacity of roots was also considerably reduced.

An attempt to regenerate roots in root (wilt) disease affected palms was made by Amma and Patil (1982) using hormones, phenols and amino acids. Application of these chemicals in the debarked region of the stem of coconut palms just above the bole induced production of new roots. The palms which received indole butyric acid (IBA) 500 ppm+phenols 400 ppm produced maximum number of roots (Table 19) followed by naphthalene acetic acid (NAA) + glutamic acid. Palms in which regeneration of roots was noticed, the disease indices also tended to decrease with an improvement in the foliar condition of the palm (Table 19).

A method for collection of root sap was devised by Davis (1964). His studies on the nature and composition of the root sap indicated that the root sap of apparently healthy palms was acidic in nature, odourless, clear and rich in K_2O and MgO contents. The root sap of diseased palms was neutral to alkaline in nature, foul smelling and poor in K_2O and MgO contents. Ramadasan (1964) noted that the root sap of diseased palms contained 65.72% more solid contents than that of healthy palms. Tomato seedlings placed in root sap collected from diseased palms developed epinasty and bending of leaves, while no such symptoms were noticed on seedlings placed in the root sap of healthy palms. Davis (1964) reported the absorption of 250 to 500 ml water per day by a single root of healthy palm, as against less than 150 ml per day by that of diseased palms. The uptake and transport of water through the trunk in diseased palms was reported to be 35% less than that of healthy palms (Ramadasan, 1964). Studies on the profile of soil moisture depletion by the roots of healthy and diseased palms in irrigated plots also revealed poor depletion by wilt affected palms (Rajagopal *et al.*, 1986b). This was further supported by studies with labelled phosphorus (Dwivedi *et al.*, 1979).

Table 19. Effect of growth hormones and phenols on root regeneration and foliar condition of root (wilt) affected coconut palms after one year of treatment (Amma and Patil, 1982)

Sl. No.	Treatment	No. of palms treated	No. of palms producing roots	Total No. of roots	Disease index	
					Pre-treatment	Post-treatment
1.	IBA 500 ppm	3	Nil	-	19	23
2.	IBA 1000 ppm	3	1	3	32	28
3.	IBA 500 ppm + Thiamine 250 ppm	3	1	4	38	35
4.	IBA 500 ppm + Phenols 400 ppm	3	3	51	35	26
5.	NAA 500 ppm + Glutamic acid 500 ppm	3	2	33	38	30
6.	Control	3	2	2	21	22

Root (wilt) diseased palms were seen to have higher stomatal frequency than that of the healthy ones (Mathew, 1981). Rajagopal *et al.*, (1986a) found abnormal stomatal opening in the infected palms with impaired regulation leading to excessive water loss, irrespective of the time of the day or season or growing condition (Fig. 14). Thus, the stomatal resistance at 14.00 h was only 5.5 sec. cm^{-1} in the leaves of diseased palms, as against 14.9 sec. cm^{-1} in those of healthy palms. With the advancement of disease, there was greater disturbance in stomatal regulation resulting ultimately in excessive water loss. They found palms in the early stage of disease with an index of 20 and a transpiration rate in the range of 4.20 to 4.35 mg. cm^{-2} sec^{-1} while those in the advanced stage with the index above 50 transpired at the rate of 9.15 to 10.50 mg. cm^{-2} sec^{-1}.

Root (wilt) affected palms had consistently lower leaf water potential than the healthy palms at any given time (Rajagopal *et al.*, 1987). The nature of symptoms on the leaflets of different whorls of leaves reflected the changes in leaf water potential (Table 20).

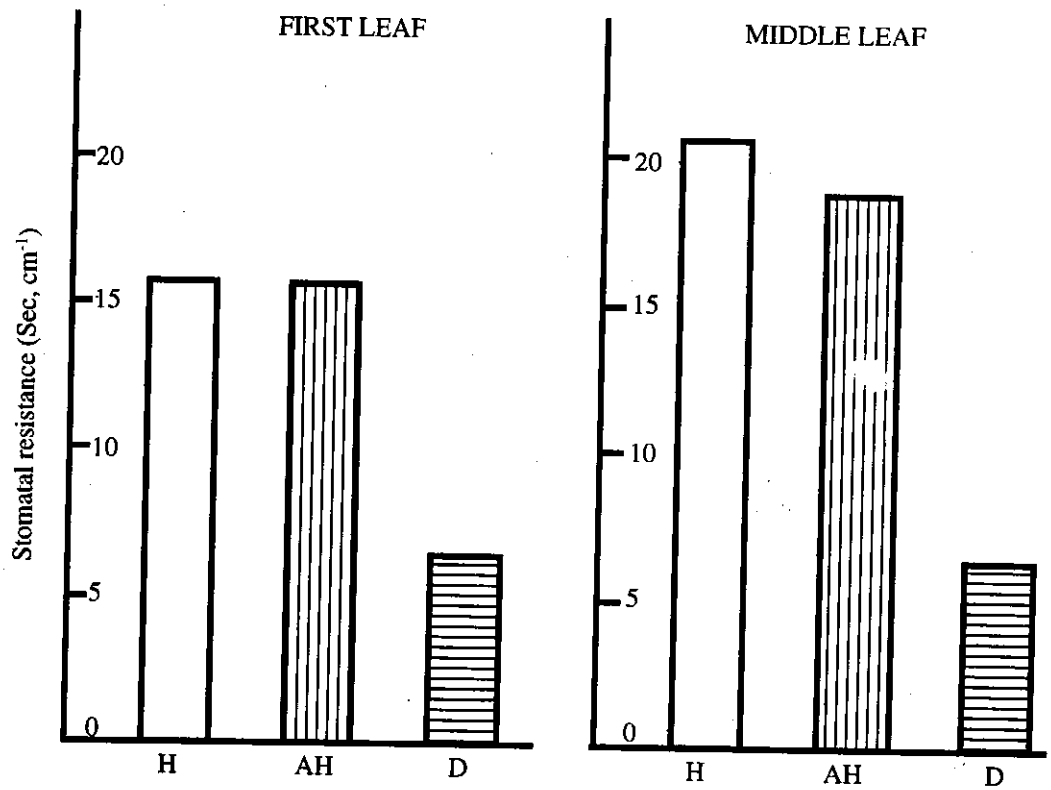
Permeability of leaf and root tissues was found damaged in diseased palms. This was revealed by the release of more electrolytes from root and leaf tissues of diseased palms than those of healthy palms (Anon., 1977).

Studies on the effect of N, P and K nutrients on the growth and incidence of root (wilt) disease of coconut seedlings revealed that nitrogen had a significant effect on the growth. The symptoms noticed on seedlings due to deficiency of N P K nutrients were not, however, comparable to the root

Table 20. Changes in leaf water potential with leaf age and leaflet condition in root (wilt) affected palms (Rajagopal *et al.*, 1987)

Position	Apparently healthy palm	(MPa)	Diseased palm	(MPa)
Spindle	Yellow to light green thick, stiff	-0.37 ±0.02	White to dull cream, thin papery, brown spots	- 0.2 ±0.06
First whorl	Green, normal, erect	-0.68 ±0.06	Light green, slight flattening and bending at the tip	-1.24 ±0.08
Middle	Dark green normal, erect	-0.79 ±0.07	Flaccidity, yellowing necrosis	-1.28 ±0.08
Outer whorl	Dull green, senescent but normal	-0.89 ±0.07	Ribbing, necrosis abnormal, senescence	-1.26 ±0.09

(This experiment was carried out in the 'wet' season (September, 1983). Values are means of six palms ± SE of the mean). * MPa = Mega-Pascal



H Healthy coconut, AH Apparently healthy coconut, D Root (wilt) diseased coconut

Fig. 14 Stomatal resistance in coconut leaves (Rajagopal *et al.*, 1986)

(wilt) disease syndrome (Mathew and Ramadasan, 1971).

Pandalai (1958) discussed the association of a high K/Mg ratio with foliar yellowing and suggested K induced Mg deficiency as a probable reason for the same. Studies on the control of yellowing revealed that 2 per cent $MgSO_4$ given as foliar spray at quarterly intervals, completely cured yellowing in 16 per cent palms, while reduction was noticed in another 60 per cent of palms (Mathew and Ramadasan, 1971). The effect of $MgSO_4$ in reducing foliar yellowing was also confirmed by Varkey *et al.* (1979). They further observed that basal application of $MgSO_4$ (500 g/palm/year) and foliar spray with 1% $FeSO_4$ helped in checking foliar yellowing in coconut.

Michael (1978) reported a significantly higher rate of respiration in root (wilt) diseased palms compared to healthy palms (Table 21).

Table 21. Respiratory rate in leaf and root tissues of healthy and root (wilt) affected palms (expressed 0_2 consumed per $mm^3mg^{-1}hr^{-1}$) (Michael, 1978)

Condition of palms	Summer season	South West monsoon season
Leaves		
Healthy	0.30	0.20
Apparently healthy	0.69	0.55
Diseased	0.70	0.62
Roots		
Healthy	1.70	1.60
Apparently healthy	3.50	2.90
Diseased	4.90	3.60

A marked variation in the photosynthetic CO_2 fixation was noticed between apparently healthy and root (wilt) affected palms, the former recording significantly high rate of CO_2 fixation than the latter. The increase in the CO_2 fixation rate was 11% in the first fully opened leaf and 70% in the middle whorl leaf of apparently healthy palms. Percentage increase of the chlorophyll content in the first fully opened and middle leaves of healthy palms was 16 and 70 respectively, over that in diseased palms (Dwivedi *et al.*, 1978).

The total reducing and non-reducing sugars were significantly higher in the leaves of infected palms. But, a depletion of these sugars occurred in the roots of diseased palms which indicated a possible derangement in the translocation and distribution of sugars in diseased palms. In spite of higher sugar content in the leaves total carbohydrates and starch contents were lower in both the leaves and roots of diseased palms (Mathew, 1977).

Padmaja *et al.*, (1981) reported an increase of 34.2%, 15.9%, 19.9% and 10.1% in the alkali extractable protein fractions in the spindle leaf, first fully opened leaf, middle leaf and outer leaf, respectively of healthy palms compared to diseased palms. The increase in the water extractable protein and ethanol extractable protein was 59.0%, 50.2%, 49.5% and 47.8% for the former in the four whorls of leaves, respectively, and 21.6%, 42.8%, 4.1% and 12.4% for the latter in the respective leaves. The low protein values obtained in the diseased palms may be the net effect of decelerated protein synthesis and accelerated protein breakdown.

Gross derangement in the path of nitrogen resulting in considerable increase in the non-protein nitrogen content, with a concomitant sharp decrease in the water soluble nitrogen and protein nitrogen fractions was observed in the diseased tissue. A reduction in the C/N ratio was also noticed in the roots and leaves of root (wilt) diseased palms (Varkey *et al.*, 1969).

Pillai and Shanta (1965) reported accumulation of certain free amino acids in root (wilt) affected leaves and they opined that accumulation of amino acids might predispose the diseased palms to 'leaf-rot' infection. The quantity of amino acids in the leaves was found to increase with the incidence and intensity of the disease. Arginine increased from minute traces in healthy to considerable quantity with increase in the intensity of the disease.

The activity of carbonic anhydrase (CA) enzyme was studied with a view to finding out the disorder of zinc. Carbonic anhydrase activity was found to be low in the leaves of diseased palms as compared to that in healthy palms (Anon., 1979). This indicated reduced concentration of biologically active zinc in palms.

The higher enzyme activity observed in diseased palms indicates that cellulase might be one of the factors responsible for the vast decay noticed in diseased palms (Padmaja and Amma, 1979).

Pectin lyase activity was found to be nearly six times higher in the roots of diseased palms as compared to the healthy ones (Amma and Patil, 1985).

Radioactive $KH_2{}^{32}PO_4$ was fed to coconut leaves and palms under laboratory and field conditions. In healthy palms the absorption of phosphorus by roots and its accumulation in spindle and first fully opened leaves was found to be significantly higher for nine hours after application, as compared to diseased palms; but at later stages, reverse pattern was observed. The time required for ^{32}p to reach the spindle situated at 9.5 M height of both healthy and diseased palms was found to be three hours only.

The analysis of total phosphorus and different fractions of the same indicated that although total P was more in diseased palms, the organic P, especially the nucleic acid phosphorus, was significantly less in diseased palms than that in healthy palms. This revealed less utilization of absorbed phosphorus in the synthesis of P constituted organic substances in diseased palms (Dwivedi *et al.*,1979).

Joseph and Jayasankar (1973) recorded that the highest concentration of polyphenols was recorded in the samples of roots collected from healthy tract as compared to that in apparently healthy (healthy palms in diseased tract) or diseased palms. With the increase in disease intensity the concentration of total phenol was found to decrease to half.

Studies on phenol oxidising enzymes such as polyphenol oxidase and peroxidase indicated a corresponding increase in their activities with the incidence and increase in intensity of the disease. A positive correleation has been noticed between the activities of these enzymes and disease index (Joseph *et al.*,1976). The

orthodihydroxy phenol content in the roots of coconut palms has also decreased with increase in intensity of the disease. A marked increase in the level of phenylalanine ammonia lyase (PAL) in the apparently healthy, disease early and disease advanced palms has been observed with a negative correlation between enzyme activity and disease indices (Joseph and Jayasankar, 1979). The activities of polyphenol oxidase and peroxidase have a negative correlation with orthodihydroxy phenol while the activity of phenylalanine ammonia lyase has a positive correlation. The isozyme pattern of polyphenol oxidase has indicated distinct difference between the healthy, apparently healthy and diseased palms, resolving into one, three and five bands, respectively (Joseph, 1983).

The rate of flow of phloem sap from the inflorescences of coconut depends on the nature of palms. Apparently healthy palms had a rate of flow of 5 ml/hr until 25 days, but shot up at 32 days with a rate of flow of 22 to 25 ml/hr. This trend continued for 70 days followed by a rapid decline. With the intensity of disease, the rate of flow decreased (Rajagopal *et al.*, 1989). The rate of sap collection differed between day and night, in that the flow was greater during the day than during the night. The data in Table 22 clearly indicates the differences in pH, osmotic concentration and sugars between the apparently healthy and diseased palms. Chempakam and Rajagopal (1989) reported the biochemical constituents of the phloem sap. Diseased palms had much less content of reducing sugars, proteins and free amino acids, phenols, lipids and sterols than in the apparently healthy palms. Arginine content alone was relatively high in the sap of diseased palms (Table 23). Further analysis with gas liquid chromatography revealed the levels of individual amino acids, organic acids and sugar (Chempakam *et al.*, 1991). Arginine and aspartic acid among amino acids, malonic acid and lactic acid among organic acids and glucose and galactose among sugars registered higher values in the sap collected from the inflorescences of diseased palms than that from the apparently healthy ones (Table 24). These biochemical constituents of the phloem sap might have favoured the growth of MLOs in the diseased palms.

Table 22. Differences in the phloem sap collected during the day and night from the inflorescences of coconut palms (Rajagopal *et al.*, 1989)

	Apparently healthy		Diseased	
	Day	Night	Day	Night
Rate of sap flow ($ml.h^{-1}$)	17.9	11.4	9.9	4.1
pH	6.9	6.1	7.2	6.5
Osmotic concentration (m mol Kg^{-1})	720	765	660	690
Total sugars (mg g^{-1} sap solid)	250	180	180	190
Reducing sugars (mg g^{-1} sap solid)	52.0	45.8	51.1	24.6

Table 23. Biochemical constituents of vascular sap. Values expressed as mg g^{-1} sap solids (Chempakam *et al.*, 1991)

Constituents	Apparently healthy	Diseased
Total sugars	299.50	286.20
Reducing sugars	98.10	65.90
Proteins	4.90	2.50
Free amino acids	45.90	18.30
Arginine*	0.41	0.68
Phenols	0.60	0.20
Lipids	0.83	0.57
Sterols*	0.40	0.22

* Values expressed as mg ml^{-1} sap.

It has, thus, become evident that the root (wilt) disease has altered many of the physiological and biochemical processes of the coconut palm. The flaccidity symptom of the disease could be correlated with the development of internal water stress, obviously caused by the pathogen.

Table 24. Composition of amino acids, organic acids and sugars identified in the phloem sap from the inflorescences of apparently healthy and root (wilt) diseased coconut palms. Values are expressed as mg g^{-1} sap solids. (Chempakam *et al.*, 1991)

Compounds identified	App. healthy	Diseased
A) Amino acids (average of 5 palms) :		
Cystine + Cysteine	0.25	0.20
Arginine	0.19	0.28
Serine	0.13	0.09
Glycine	0.11	0.09
Aspartic acid	0.19	0.25
Glutamic acid	0.12	0.06
Leucine	0.14	0.12
Methionine	0.17	0.13
Phenylalanine	0.27	0.24
Tyrosine	0.06	0.23
B) Organic acids (average of 5 palms) :		
Oxalic acid	1.54	1.03
Malic acid	1.29	1.02
Fumaric + succinic acids	0.41	0.29
Maleic acid	0.90	0.64
Malonic acid	75.07	82.05
Citric acid	11.73	12.93
Lactic acid	13.67	23.70
C) Sugar (average of 4 palms) :		
Sucrose	135.50	128.30
Glucose	25.50	39.40
Galactose	18.10	28.90
Mannose	11.50	9.50
Lactose	16.30	14.70
Raffinose	20.40	17.50

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7

MICROBIOLOGY

N.P. JAYASANKAR, M. GEORGE, V.G. LILY and G.V. THOMAS

The nature and activities of the micro-organisms in a soil environment depend on the crop grown, infestation by pests/ pathogens and management practices. The root surface microflora of the coconut palms in relation to coconut root (wilt) disease have been determined under varying conditions. The pioneering efforts of Radha and Menon (1954) has indicated a consistent reduction in the micro-organisms on the root-surface of root (wilt) affected palms compared to the apparently healthy palms in the disease affected tract. Radha and Rawther (1959) subsequently reported a comparative reduction in the soil microflora during rainy season in the same tract. Rawther and Radha (1963) reported a low soil microbial activity in terms of CO_2 evolved from a unit quantity of soil in unit time.

However, the results of the investigation carried out by Potty (1977) revealed significantly higher population of actinomycetes and bacteria in coconut soils of root (wilt) affected region compared to the healthy (Fig 15). The occurrence of these organisms was higher in the top soil at a depth of 0-25 cm. Irrespective of the diseased condition the coconut rhizosphere harboured higher numbers of actinomycetes, bacteria and fungi (Fig. 16). The number of different microflora was more in the rhizosphere of diseased palms compared to healthy. The difference in the quantitative distribution of

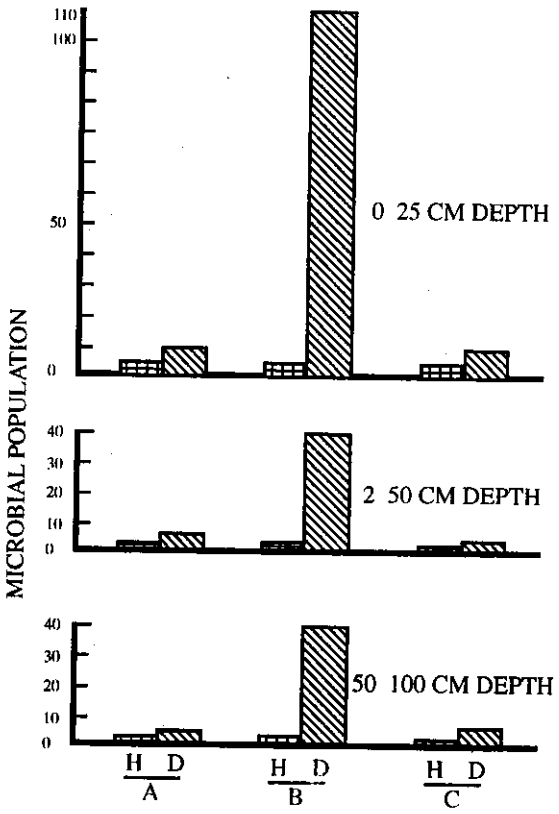

Fig. 15 Distribution of actinomycetes, bacteria and fungi in the healthy and root (wilt) affected regions

H	Healthy region	D Root (wilt) affected region
A	Actinomycetes	(10^3 per g of oven dry soil)
B	Bacteria	(10^6 per g of oven dry soil)
F	Fungi	(10^4 per g of oven dry soil)

the various micro-organisms between the young and old roots was not consistent. Similarly, the pattern of occurrence of the different microflora in the coconut rhizosphere varied in different soil types irrespective of the disease condition of the palms. Within the root (wilt) affected region a higher population of the different microflora was noticed with higher levels of water table (Fig. 17). This trend was more pronounced in the case of bacteria.

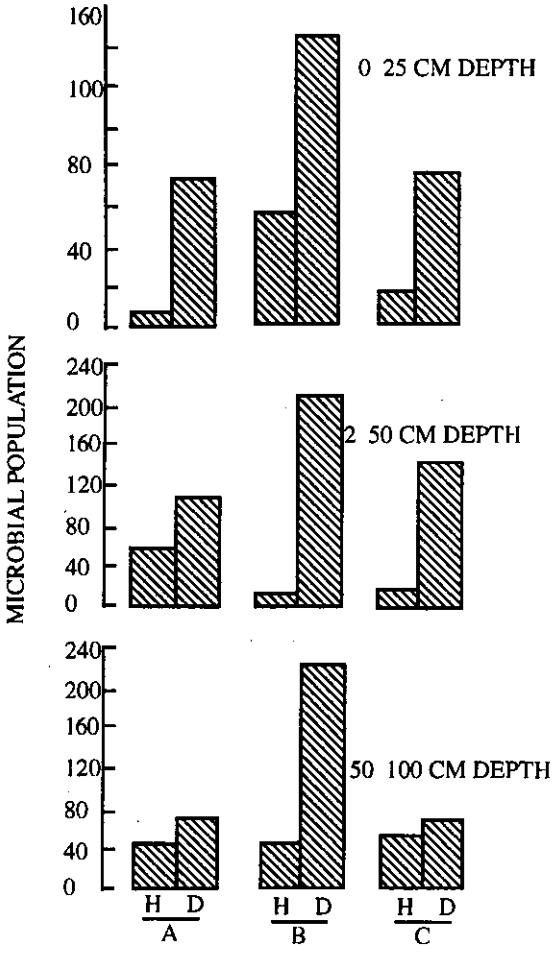

Fig. 16 Distribution of rhizosphere microflora in healthy and root (wilt) affected palms

H Healthy palms D Root (wilt) affected palms
A Actinomycetes (10^3 per g of oven dry soil)
B Bacteria (10^6 per g of oven dry soil)
F Fungi (10^4 per g of oven dry soil)

Observations were also recorded on the microflora of the coconut palms under mixed cropping with fodder crops in the root (wilt) affected region. Preliminary studies revealed higher values of total bacteria and ratios of nitrogen fixing organisms to denitrifiers in coconut soils cultivated with the fodder crop *Stylosanthes gracilis* alone and in combination with Hybrid Napier

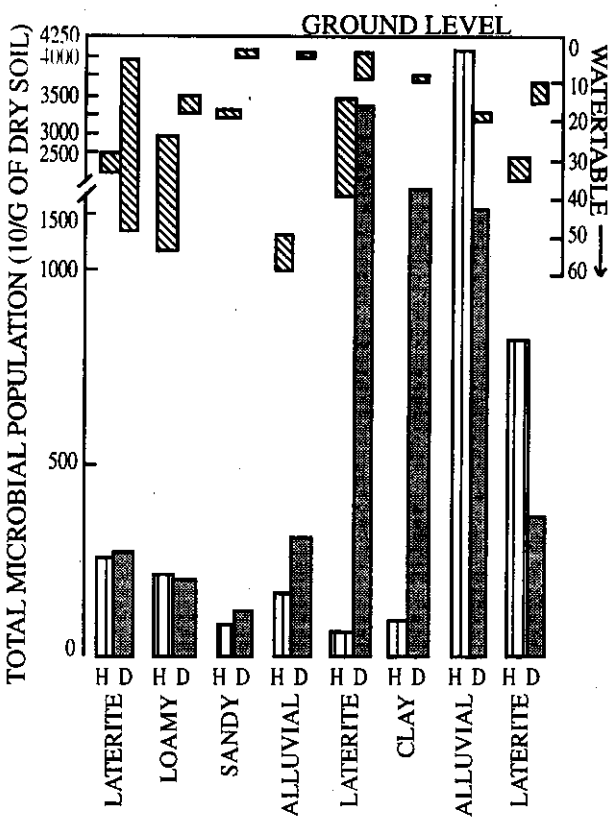

Fig. 17 Distribution of total microflora in relation to water table in healthy and root (wilt) affected regions

H Healthy palms.
D Root (wilt) affected palms.

(Sahasranaman *et al.*, 1983). Mixed cropping with Hybrid Napier enhanced the total bacteria and nitrogen fixing organisms in the coconut rhizosphere irrespective of the disease condition of the palms. There was also an increase in the number of phosphate solubilising bacteria in the rhizosphere of healthy and diseased palms; but the diseased palms harboured a higher number (Potty *et al.*, 1977). These trends were, however, reversed after the application of inorganic manures (Potty and Jayasankar, 1983) (Table 25). Studies on the influence of growing fodder legumes (*Centrosema pubescens* and *Stylosanthes gracilis)* besides Hybrid Napier on the rhizosphere microflora of the coconut palms showed similar trends.

Physiological classification of the actinomycetes isolated from the root surface of healthy and root (wilt) affected coconut palms under mixed cropping with Hybrid Napier (Ag. I), *Centrosema pubescens* (Ag. II), and *Stylosanthes gracilis* (Ag. IV) yielded positive indications (Potty, 1977). Traits studied were phosphate solubilisation (P), nitrate reduction (N), cellulose digestion (C), gelatin hydrolysis (G) and starch hydrolysis (S). A distinct difference was noticed in the combinations PG, PS, PC and PN between the crop mixed and control plots. In Ag. III treatments GS, GC and GN combinations were absent in the isolates. In the combination among three different characters, PGS, PGC, PSN and PCN were not observed in any of the isolates from AG. II treatment. The combined ability of all the five characteristics PGSCN was noticed only among the isolates from diseased palms in Ag. I Treatment. Result indicated the possibility of altering

Table 25. Influence of crop mixing Hybrid Napier grass on the coconut rhizosphere microflora, before and after fertilizer application.

Organisms	Coconut + Napier grass				Coconut alone			
	Pre-application		Post-application		Pre-application		Post-application	
	Healthy	Diseased	Healthy	Diseased	Healthy	Diseased	Healthy	Diseased
Total								
Bacteria	51.84	21.25	21.83	10.28	27.00	5.51	5.15	7.81
Fungi	37.62	29.46	28.67	206.71	6.76	76.11	1.05	20.48
Nitrogen fixing								
Bacteria	84.20	35.86	9.92	8.25	35.86	8.71	3.47	7.88
Phosphate solubilizing								
Actinomycete	4.95	2.33	0.83	1.30	1.45	3.04	0.33	0.55
Bacteria	8.15	130.78	0.61	0.87	8.85	3.95	0.63	0.93
Fungi	6.58	5.26	1.12	1.21	2.40	2.40	1.52	1.46

the microflora of the coconut rhizosphere by appropriate inter and mixed cropping.

The feasibility of intercropping tuber/rhizome crops with coconut palms in the root (wilt) affected tracts of Kerala has been brought out by Antony (1983). The population of the different microorganisms including nitrogen fixing and phosphate solubilising ones was enumerated during the first and second years at the time of harvest of the intercrops. In addition, enzymatic activities like dehydrogenase, invertase, urease and phosphatase were estimated at the time of planting, during vegetative phase and at harvest during the third year. Beneficial influence on the number of asymbiotic nitrogen fixing bacteria was noticed both in the coconut root surface and non-rhizosphere soils. During the vegetative phase there was a significant increase in the activities of dehydrogenase and invertase. Intercropping significantly stimulated phosphatase activity in the non-rhizosphere soils.

Thomas and Shantaram (1984) conducted studies to find out the ability of different green manure legumes to grow and establish in the basins of root (wilt) affected coconut palms in sandy loam soils. Among the nine legumes *Pueraria phaseoloides, Mimosa invisa* and *Calopogonium mucunoides* yielded 19.43, 17.00 and 14.71 kg of green matter/basin, respectively, in a period of four months (Table 26). *In situ* there was a significant increase in the microbial populations as also in the activities of soil enzymes viz. dehydrogenase, urease and phosphatase (Tables 27,28) due to the incorporation of legumes in respective coconut basins.

The biomass production, nitrogen contribution and nodulation characteristics of ten species of legumes were evaluated in

Table 26. Growth and nodulation of green manure crops in basins of root (wilt) affected coconut palms (Thomas and Shantaram, 1984)

Legume species	Growth		Nodulation	
	Fresh weight (kg/basin)	Total N added (g/basin)	Nodule number/five plants	Nodule dry weight (g/five plants)
Calopogonium mucunoides	14.71	102.61	145	0.485
Macrotyloma axillaire	0.95	6.67	58	0.132
Mimosa invisa	17.00	153.19	125	1.860
Pueraria phaseoloides	19.43	121.29	132	1.128
Leucaena leucocephala	2.95	16.55	0	0.000
Sesbania aegiptica	1.30	6.98	56	0.535
Macroptilium atropurpureum	9.10	66.64	60	0.108
Glycine weighti	2.35	19.20	95	0.205
Stylosanthes guianensis	3.50	12.70	350	0.055
CD at 5 %	7.59	53.24	65	2.905

a root (wilt) diseased garden in laterite soil type (Thomas and Shantaram, 1993). *P. phaseoloides, C. mucunoides* and *M. invisa* yielded higher quantities of biomass to the level of 28.4, 27.2 and 24.9 kg per basin with a nitrogen contribution of 196.2, 186.5 and 187.6 g per basin, respectively, during a growth period of 130-140 days in rainy season. Zymogenic response of microflora and enhanced dehydrogenase enzyme activities were observed in root region soils of coconut at maximum vegetative growth of legumes in cocônut basins and at 30 days of incorporation of green manures (Thomas, 1987). *In situ* cultivation and incorporation of legumes were also effective in enhancing the vesicular-arbuscular mycorrhizal (VAM) symbiosis in coconut.

Subsequently, Thomas (1988) reported the adverse effect of root (wilt) disease on the mycorrhizal symbiosis of coconut and showed the efficacy of intercropping system with hybrid napier in increasing the

Table 27. Effect of green manure incorporation on microbial population in coconut rhizosphere soil (Thomas and Shantaram, 1984)

Green manure incorporated	Microbial population per g oven dry soil					
	Bacteria ($X10^5$)	Fungi ($X\ 10^4$)	Actinomycetes ($X10^4$)	Asymbiotic N_2 fixers ($X10^3$)	P. solubilisers	
					Fungi ($X10^3$)	Bacteria ($X10^4$)
P. phaseoloides	39.25	8.80	18.78	69.30	7.85	7.51
C. mucunoides	16.00	11.39	14.67	63.49	4.15	6.16
M. invisa	2.84	10.12	5.27	30.12	6.38	4.47
Control	18.61	2.30	6.86	41.44	1.17	1.75
CD at 5%	10.08	2.75	2.98	NS	2.73	NS

Table 28. Effect of green manure incorporation on enzyme activities in coconut rhizosphere soil (Thomas and Shantaram, 1984)

Green manure incorporated	Dehydrogenase activity (µg TPF/g over dry soil)		Phosphatase activity (µg PNP/g oven dry soil	Urease activity (µg NH_4+/g oven dry soil)
	Endogenous	Response to glucose		
P. phaseoloides	4.072	22.226	62.22	93.08
C. mucunoides	3.281	29.550	82.58	97.04
M. invisa	3.310	11.975	54.11	69.75
Control	1.568	5.450	50.93	68.29
CD at 5%	1.411	8.400	14.75	11.41

TPF = Triphenylformazo; PNP = Para nitrophenol

mycorrhizal status of root (wilt) affected coconut palms.

The field trial conducted with *P. phaseoloides, M. invisa* and *C. mucunoides* for a period of five years indicated the effectiveness of basin management with the legumes alongwith the application of recommended dose of NPK and MgO in increasing the productivity of root (wilt) diseased palms without causing further deterioration in the disease condition of the palms (Thomas *et al.* 1993).

Inoculation studies on *P. phaseoloides* and *C. mucunoides* with eight isolates of *Rhizobium* from different forage legumes indicated the promiscuous nature of rhizobium requirement of the two legumes (Thomas and Shantaram, 1987). A comparative field trial with promising local isolates of *Rhizobium* and NifTAL cultures in a root (wilt) diseased coconut garden indicated the effectiveness of composite NifTAL cultures in increasing nodulation and dry matter production in *C. mucunoides. P. phaseoloides* did not respond to rhizobial inoculation due to effective nodulation by native soil rhizobia. *M. invisa* also showed profuse nodulation by native rhizobia in different soil types.

Cropping systems involving inter cultivation of crops resulted in an improvement in the microbial population and biological activities in the root region soils of root (wilt) diseased coconut palms indicating a modification in the soil environment to the benefit of plant growth. The cultivation and incorporation of green manure legumes in coconut basins can be considered a simple and less expensive input to enhance the soil fertility and yield of diseased palms.

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8

LEAF ROT DISEASE

N. SRINIVASAN, M. GUNASEKARAN, T. JOSEPH and T.S.S. RAWTHER

Coconut leaf rot disease (LRD) is important as it is widely prevalent in association with root (wilt) disease (RWD) in Southern districts of Kerala state. The extensive destruction of leaf in LRD infected so palm is so conspicuous that the RWD palms draw the farmers' easy attention after the onset of LRD. The disease is over a century old and investigations had begun almost in parallel with the investigations on RWD. The results on different aspects of LRD investigations are summarised in this chapter.

Inter-relationship of LRD with RWD

Since the beginning of the century LRD seemed to have been considered as a part of RWD. Varghese (1934) observed that leaf disease of coconut occurred along with the root disease and generally the same palm showed symptoms of both diseases. Radha and Lal (1968) reported close relationship between occurrence of LRD and RWD in the field as well as in inoculation trials. They observed that nearly 16 to 40% of the palms in the root (wilt) affected areas developed leaf rot and its intensity varied in different types of soils. Srinivasan (1991) reported that the LRD was generally confined to RWD affected palms and on an average 65% of these palms were superinfected with LRD; the incidence of LRD increasing with the intensity of RWD. Thus a strong inter-relationship of LRD with RWD incidence is evident. Young palms with RWD symptoms were readily attacked by LRD irrespective of soil types (Table 29).

Table 29. Incidence of LRD in relation to RWD

Soil type	Non-bearing (young) palms			Bearing (adult) palms		
	No of palms sampled	No diseased*	RWD with LRD (%)**	No of palms sampled	No diseased*	RWD with LRD (%)**
Sandy loam	354	126	85.71	1496	1316	50.61
Sandy	161	86	90.70	860	597	58.29
Alluvial	153	90	82.22	645	529	63.89
Clay	186	113	85.84	719	549	73.95
Laterite	105	47	93.62	806	589	74.36
Total	959	462	-	4526	3580	-

* Sum of RWD alone and RWD superinfected with LRD

** Out of diseased palms

Severe natural incidence of LRD in RWD infected seedlings in field conditions was observed (Anon., 1996). Inoculation experiment on two year old RWD affected seedlings showed severe infections under field conditions. The palms weakened by Phytoplasma, the causal agent of RWD, might result in the breakdown of defence machanism leading to susceptibility to LRD. The occurrence of LRD with RWD seems to be a distinct Phytoplasma - fungal disease complex, a phenomenon so far not known in other diseases of crop plants.

Symptomatology and Indexing

Symptoms

LRD appears initially as minute, water-soaked lesions on the emerging spindle with different shades of colour and shapes. These lesions enlarge, coalesce freely leading to extensive rotting. The rotten portions dry

Fig. 19. Leaf rot symptoms on the midrib

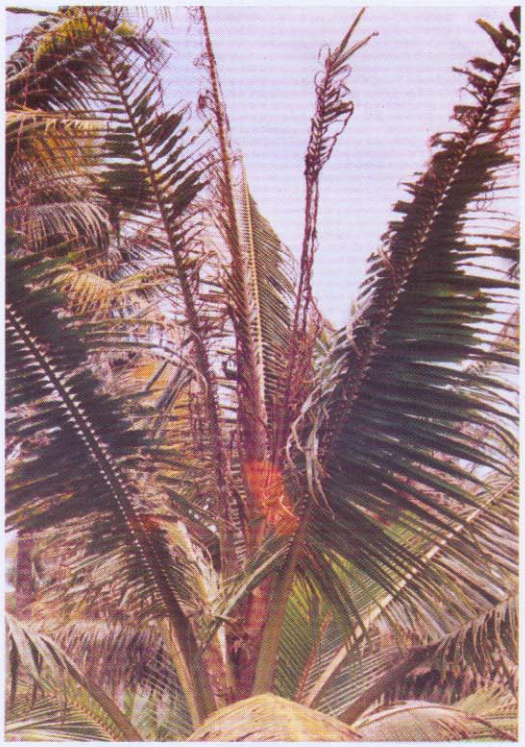

Fig. 18. Rotten spindle of an affected palm infected with leaf rot.

Fig. 20. Susceptibility of diseased palm to rhinoceros attack

Table 30. Symptoms of LRD observed in different whorls

Soil type	No palms	Palms exhibited LRD in different whorls/whorl combinations(%)*				
		Inner (I)	Outer (O)	I+M	I+O	I+M+O
Sandy loam	774	58.53	4.65	0.26	12.14	24.42
Sandy	426	26.06	18.54	-	12.44	42.96
Alluvial	412	32.53	16.50	-	18.69	32.28
Clay	503	19.48	29.62	-	13.12	37.78
Laterite	482	21.99	12.86	-	26.97	38.18
Total	2597	34.73	15.17	0.08	16.17	33.85

* Symptoms only in Middle (M) whorl or M+O whorl combination not observed

up, turn black and fall off. Tips of leaflets and mid-ribs often become blackish and shrivelled. The progress of rotting slows down with the maturity of leaflets (Fig. 18-20). The quantitative pattern of the LRD symptoms in different whorls of affected palms revealed that the innerwhorls of leaves are vulnerable to infection. Out of 2597 diseased palms sampled representing different soil types, 24 to 43% expressed the disease symptoms in the entire crown accounting approximately one third of LRD affected palms. These palms had contracted successive infection of freshly emerged leaves which resulted in varying degrees of rotting of the leaflets and hence the 'fan-like' appearance of all the leaves in the crown (Table 30) (Srinivasan and Gunasekaran, 1992). Younger leaves, especially the spindle, play a critical role in LRD incidence. In severely affected spindle tissues, mould growth is also commonly seen on the surface of the affected leaflets. (Srinivasan *et al.*, 1995) Rapid expansion of lesions relate to infection in the early period of spindle leaf emergence and the rotting may extend into the interior of the spindle. The symptoms of LRD on parts such as leaf petiole and mid-rib/mid-vein of leaflets was observed in naturally infected palms and such symptoms have been reproduced by artificial inoculations also (Anon., 1996).

Disease indexing

For quantification of disease intensity a four point grading system was adopted for LRD indexing (Srinivasan and Gunasekaran 1996a).

Disease grade	% leaf area infected
0	No infection
1	up to 25
2	26-50
3	51-75
4	Above 75

$$\text{Disease Index (DI)} = \frac{\text{Total Numerical ratings}}{\text{No of leaves x Max No. of grades}} \times 100$$

The above disease indexing method could be utilized for comparison of disease intensity. However, LRD indexing may be refined by using more grades with narrow intervals of disease ratings and also an intergrated disease indexing has to be evolved for LRD and RWD.

Yield Loss

Information on the loss due to leaf rot disease alone is not available as it is generally associated with the RWD. However, Menon and Nair (1948) estimated the loss at Rs. 5.6 million annually. This is besides the loss in quality of the leaves rendering them unfit for thatching and other purposes. Applying the above criteria the loss due to leaf rot may be computed at present as 461 million nuts annually as the area affected by the disease in Kerala extends to 0.41 million hacteres (Anon., 1985 b).

Etiology

McRae (1916) and Sundararaman (1925) isolated a salamon coloured *Penicillium* like fungus from diseased leaves in Kochi. *Helminthosporium halodes* (= *Bipolaris halodes*) *Gleosporium* sp., *Curvularia* sp., *Gliocladium roseum*, *Pestalotia* sp., and *Fusarium* sp. were isolated from the diseased leaves (Menon and Nair, 1948, Anon, 1985). Srinivasan and Gunasekaran (1993, 1996 b, c) made isolations from over 500 infected spindles and revealed the association of a number of fungal species, and attributed LRD to a fungal complex. The fungi isolated were identified at CAB International Mycological Institute, United Kingdom as *Colletotrichum gloeosporiodes* (Penzig) Penzig and Sacc., *Exserohilum rostratum* (Drechsler) Leonard and Suggs, *Gliocladium vermoeseni* (Biourge) Thom. (Srinivasan and Gunasekaran, 1994a), *Cylindrocladium scoparium* Morgan (Srinivasan and Gunasekaran, 1995a), *Fusarium solani* Martius (Sacc.), *F. moniliforme* Sheldon var. *intermedium* Neish and Legget (Srinivasan and Gunasekaran, 1998a), *Thielaviopsis paradoxa* (Dade) C. Moreau, *Rhizoctonia solani* Kuhn, *Mortierella elongata* Linnem, *Curvularia* sp., *Acremonium* sp., *Thielavia microspora* Mouch, *T. terricola* (J. Gilman and E.V. Abbott) Emmons and *Chaetomium brasiliense* Batista and Pont. (Srinivasan and Gunasekaran, 1994b).

Studies on the pathogenicity of the fungi were initiated by Menon and Nair (1948, 1951). Inoculating bits of tender leaves and leaflets of mature leaves with spore suspensions of *H. halodes*, *Gloeosporium* sp., *G. roseum* and *Pestalotia* sp., *in vitro*, they found that *H. halodes* induced infection with in 12 hours and the rest in 48 hours. They considered *H. halodes* as the most virulent and the rest only as secondary parasites aggravating the rotting initiated by *H. halodes*. They confirmed these findings through *in vivo* tests and established the pathogenicity of *H. halodes* using single and mixed inocula. Later Radha and Lal (1968) also confirmed the infectivity of *B. halodes* on coconut. Culture filtrate of *H. halodes* when applied on tender leaves of coconut failed to demonstrate any toxic effect (Anon., 1981). The pathogenicity of *C. gloeosporioides* (Cg), *E. rostratum* (Er), *G. vermoeseni* (Gv), *F. solani* (Fs), *F. moniliforme* var, *intermedium* (Fn), *T. paradoxa* (Tp), *R. solani* (Rs), *M. elongata* (Me) and *Curvularia* sp. was established (Srinivasan and Gunasekaran 1994a, b, 1995a, 1996d, 1998a). The fungi associated with LRD were grouped

into three categories (Group A, B and C) based on the frequency, pattern and relative association with the disease (Anon., 1994).

In diseased spindle tissues, profuse mycelial growth and spore masses are common especially in between infected leaflets. Hence mycoflora of spindles and older leaves of 120 palms were observed by leaf scrapings as well as isolations. The fungi were present either independently or in association with other species. *C. gloeosporioides, E. rostratum, G. vermoeseni, F. solani, F. moniliforme* var. *intermedium* and *T. paradoxa* were frequently recorded. *Pestalotiopsis palmarum* was isolated from older leaves only (Srinivasan *et al.*, 1995). Isolations from LRD affected spindles with different disease grades has also brought out the predominant species of fungi irrespective of the degree of symptoms and *C. gloeosporioides* was more frequently observed under various grades (Table 31) (Srinivasan and Gunasekaran, 1996b). From several attempts of *in vitro* isolations, presence of fungi singly or co-occurrence in individual palms was noticed.

Table 31. Fungi isolated from leaf rot affected coconut spindles under different grades of disease symptom

Fungi isolated (independently or in combination)	No. of leaflet pieces which developed fungal growth (out of 50 pieces per grade)*					
	August			December		
	Grades of symptom			Grades of symptom		
	I	II	III	I	II	III
Colletorichum gloeosporioides	9(16)	10(14)	7(13)	12(20)	10(17)	7(15)
Exserohilum rostratum	4(9)	2(4)	2(6)	6(9)	4(9)	3(6)
Fusarium spp.	3(5)	3(5)	2(4)	5(12)	8(14)	8(13)
Gliocladium vermoeseni	2(2)	3(3)	1(1)	3(3)	4(4)	2(2)
Thielaviopsis paradoxa	-	2	1	-	-	2
Mortierella elongata	-	-	1	-	-	-
Rhizoctonia solani	-	-	-	-	1	2
Acremonium sp.	-	-	1	-	-	-
Cylindrocladium scoparium	-	-	-	-	-	1
C. gloeosporioides + *Fusarium* spp.	5	2	4	2	3	3
C. gloeosporioides + *Fusarium* spp.	2	2	2	6	4	5
E. rostratum + *Fusarium* spp.	-	-	-	1	2	-
Total No. of leaflet pieces which developed fungal growth	25	24	21	35	36	33
Percentage of fungal recovery	50	48	42	70	72	66

Chi-square value 2.23 (Not significant) 1.29 (Not significant)

* Figures within the parantheses are pooled No. of leaflet pieces for concerned perdominant fungi subjected for chi-square analysis.

Table 32. ***In vitro*** **interaction among the likely incitants of LRD**

Characters of fungal colony	Fungal combinations	Status of inhibiton reaction
Colonies merged	Cg x Er, Cg x Tp, Cg x Cs, Cg x Rs, Cg x Me, Er x Fx, Er x Fm, Er x Cs, Er x Rs, Er x Me, Gv x Tp, Fs x Cs, Fm x Cs, Tp x Me, Cs x Me	No inhibition discerned between colonies under dual cultures of the fungal combinations
Colonies close	Cg x Gv, Cg x Fs, Cg x Fm, Er x Gv, Er x Tp, Gv x Fs, Gv x Fm, Gv x Cs, Gv x Fs, Gv x Me, fs x Fm, Fs x Rs, Fm x Rs, Tp x Rs, Cs x Rs	Extremely mild, mild or moderate inhibition only noticed in specific combinations; in Fs x Rs and Fm x Rs dual cultures moderate inhibition of Rs by Fs/Fm evident; similarly Gv by Cs
Colonies apart	Fs x Tp, Fs x me, Fm x Tp, Fm x Me, Tp x Cs, Rs x Me	Strong inhibitions detected; Tp and Me strongly inhibited by Fs and Fm; similarly Tp by Cs and Me by Rs

In vitro interaction among selected nine species of fungi of LRD was studied by dual culturing. The interaction varied in respect of colony merger, over growing capacity and inhibition zone (Table 32). The behaviour of the predominant fungi was seen to be synergistic than antagonistic, implying etiological significance of disease complex (Srinivasan and Gunasekaran, 1995b).

In certain RWD endemic areas, leaves of middle whorl of palms suddenly become yellow evenwhile the central shoot (spindle) and leaves of outerwhorl appear normal. Lesions/spots appear in such leaves which coalesce leading to severe blighting of lamina. *C. gloeosporioides* was found to be the most commonly associated fungus (90%) and it is likely that the fungus gains access to yellowed leaves of RWD affected palms (Srinivasan and Gunasekaran, unpublished).

Pathogenic behaviour of predominant species of LRD was further elucidated in potted seedlings and field palms, through artificial inoculations. In healthy palms (free from RWD), LRD fungi induced only restricted lesions as compared to severe rotting in RWD palms (Srinivasan and Gunasekaran, 1996d). Based on the frequency of occurrence, seasonal relationship, pathogenicity etc. *C. gloeosporioides* and *E. rostratum* are considered as the main pathogens of LRD (Fig. 21 & 22). The disease symptoms were consistently reproduced in the spindles of RWD affected seedlings in field with the main pathogens, individually and in combination. The inoculations with pathogens developed early lesions which subsequently resulted in the severe rotting of tissues. The disease lesions were observed on lamina, leaf petioles, and mid-veins.

Fig 21. ***E. rostratum***

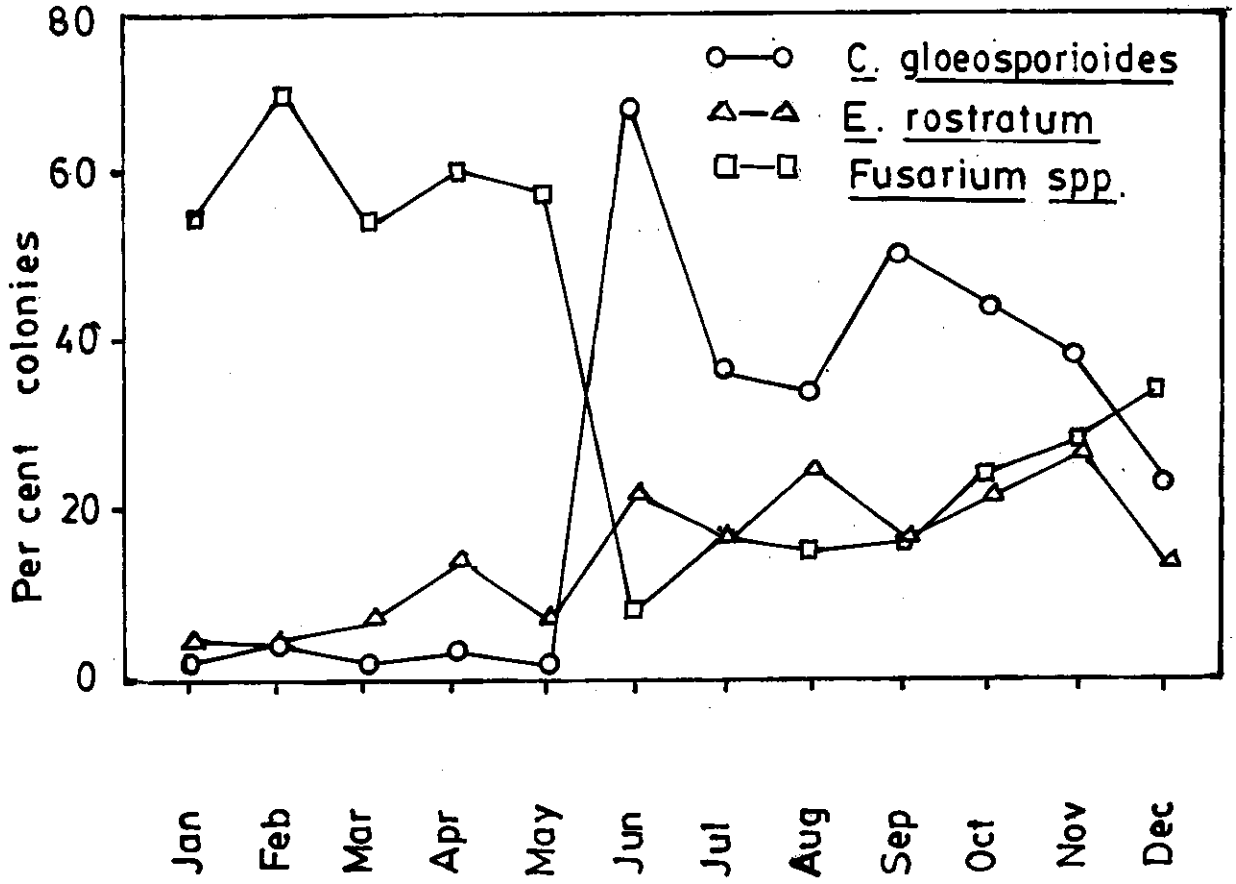

Fig 22. Seasonal variation in the isolation of major fungi from leaf rot affected samples

Symptoms were also noticed on spindles which emerged subsequent to the ones inoculated and thus the disease recurrence in successively emerging spindles was accomplished. Although LRD initially appears as a spot in spindle irrespective of the pathogen, later it results in rotting of tissues. A number of foliar diseases of coconut palms recorded world-wide causing leaf blight/leaf rot could be compared with LRD.

Epidemiology

Knowledge of dynamics of pathogen(s) is essential in view of the complex nature of the LRD. Leaf rot infection was found to be more severe during the seasons when atmospheric humidity was at its maximum (Menon and Nair 1951). Severity of leaf infection with *H. halodes, Gloeosporium* sp. and *G. roseum* was found correlated with high humidity and low temperatures prevalent during the monsoon period (Radha *et al.*, 1961). Monthly records of observations for 3 years revealed that period of high atmospheric humidity and low temperatures

are favourable for natural development of leaf rot disease (Radha and Lal 1968). The population dynamics of LRD pathogens in relation to weather variables was recorded by sequential monthly isolations from diseased palms (spindles) (Srinivasan and Gunasekaran, 1996c). The incidence of *C. gloeosporioides* was conspicuously higher in frequency and population during monsoons with a peak in June/July (Fig. 23). Its incidence was most strongly correlated with rainfall and relative humidity and negatively with maximum temperature and hours of sunshine. It was this fungus isolated from early lesions more frequently than advanced lesions (Fig. 24). Therefore, *C. gloeosporioides* was implicated as the principal pathogen of LRD during monsoons.

Incidence of *E. rostratum* was less frequent, erratic and its population was less strongly/consistently correlated with weather. In winter with low humidity and temperature, *C. gloeosporioides* incidence was subdued while that of *E. rostratum* was moderate. *Fusarium* spp. were isolated most commonly during the dry season of January - May (Fig. 22). Dry conditions

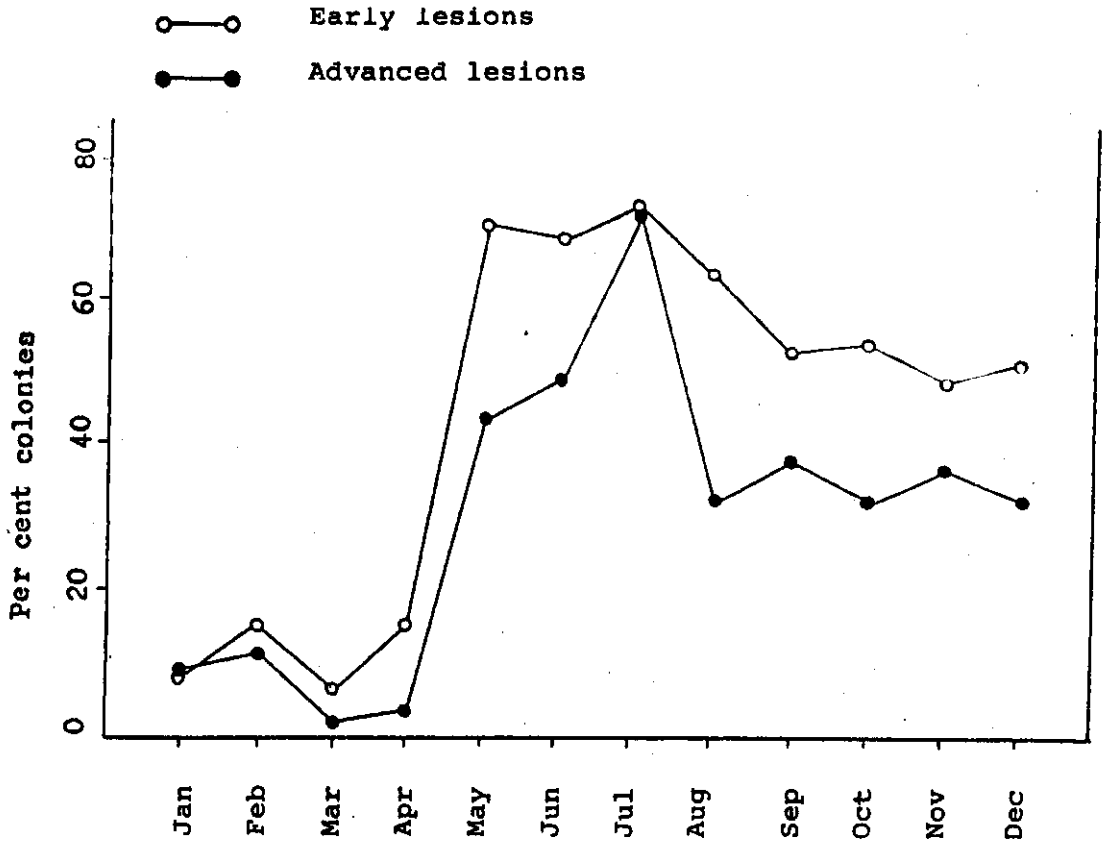

Fig 23. Isolation *C. gloesporioides* from early and advanced lesions during different seasons

generally favoured *R. solani* also. Fusarial presence throughout the year and their predominance in the dry period point out that such fungi could be potential pathogens, co-occurring with other fungi and perpetuating the disease in the dry period. They may also play a role in predisposing the palms to subsequent infection by other pathogens in favourable conditions. The incidence of other fungi was not influenced by specific weather conditions, but they may play some role in the disease in certain circumstances. The low incidence of *C. gloeosporioides* in dry season might be due to its quiescent phase. Aggressive re-emergence of the fungus with the onset of South West monsoon (with favourable weather) was linked to the severity of LRD during monsoons.

From LRD infected young palms also the incidence of *C. gloeosporioides* during the period (June-December) was consistent and significantly higher than *E. rostratum*. Hence aggressiveness of *C. gloeosporioides* in LRD during monsoon months (principal pathogen) was further ascertained (Anon, 1996). Though *E. rostratum* can vigorously induce LRD upon artificial inoculation, in nature *C. gloeosporioides* is distinctly active in the disease during wet season.

Disease Management

Control of LRD is considered important in view of its destructive potential to the palms. *In vitro* screening of fungicides against the pathogens of LRD and control studies were undertaken.

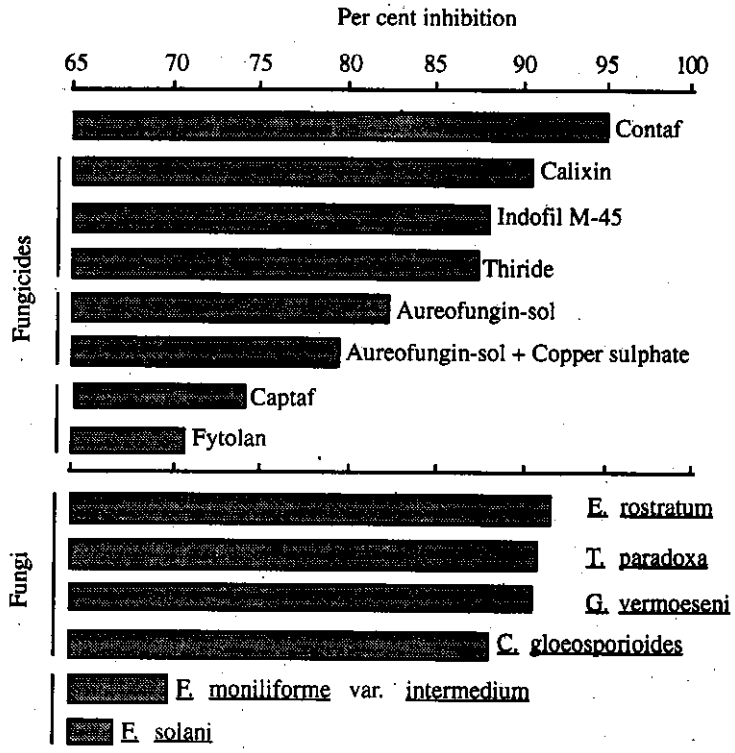

Fig 24. *In vitro* inhibition of major leaf rot fungi by different fungicides

In vitro assay

In vitro studies on the effect of different copper fungicides on the mycelial growth and spore germination of *H. halodes* showed that Bordeaux mixture (0.3%) completely inhibited the mycelial growth. (Prasannakumari *et al.* 1960). Assay of a few contact (Indofil M-45, Fytolan, Captan and Thiram) and systemic fungicides (Contaf, Calixin, Aureofungin -Sol) was done on solid medium against *C. gloeosporioides, E. rostratum* etc. by poisoned food technique (Srinivasan and Gunasekaran, 1998b). Varying degrees of inhibition of fungi by fungicides was observed. Contaf (hexaconazole) exhibited a broad-spectrum activity inhibiting all the pathogens of LRD (Fig. 24).

Field control

Attempts at controlling leaf rot disease were started as early as in 1951. Menon and Nair (1952) conducted spraying in the field using 1.0% and 0.5% of bordeaux mixture against leaf rot. Half strength of bordeaux mixture was almost as good as full strength. In a fungicial field trial using copper fungicides it was observed that Bordeaux mixture reduced the intensity by 74.5% followed by Kirti copper 65.6% and Fytolan 59.6% (Anon., 1963). Gregory (1960) felt that aerial spraying of oil based copper fungicide in Vadayar and Malankara estates was useful in controlling leaf rot. However, Samraj *et al.* (1966) observed that spray fluid did not adequately protect the spindle leaf. George and Samraj (1966). reported that coconut palms affected by leaf rot responded favourably by foliar application of boron, suggesting boron deficiency as the factor responsible for development of disease. Prophylactic basal application of systemic fungicides (Actidione, Bavistin, Benlate and MBC) at the rate of 4g per healthy palm twice a year failed to prevent incidence of leaf rot. (Anon., 1983). Sequential spraying of Bordeaux mixture 1%, Fytolan, 0.3% and Dithane M-45 0.3% on leaf rot affected palms in farmer's gardens resulted in control of the disease (Anon. 1985).

A field control trial on 20 year old infected palms was conducted. Treatments were imposed thrice a year by contact (Indofil M-45 0.3%, Fytolan 0.5%) and systemic fungicides (Calixin 1%). This was continued for three years. The method involved a simple method of pouring the chemical into axil around the spindle, besides spraying on to the crown and root feeding (Srinivasan and Gunasekaran, 1998a). Pouring of calixin and spraying of Indofil M-45 were beneficial as the LRD intensity reduced in newly emerged leaves (Fig. 25). Although the overall impact of the treatments in reducing the disease indices was moderate, the results pointed out the necessity of using a broadspectrum potential fungicide in LRD management and protecting the tender leaves, specially the spindles from infection.

Another field trial initiated in 1986 revealed higher level of disease suppression by Phystosanitation plus pouring of Contaf into leaf axil.

Biocontrol

Studies on biological control of LRD was also initiated. Antagonistic activity of an isolate of *Pseudomonas fluorescens* (TNAU, Coimbatore isolate) against *C.*

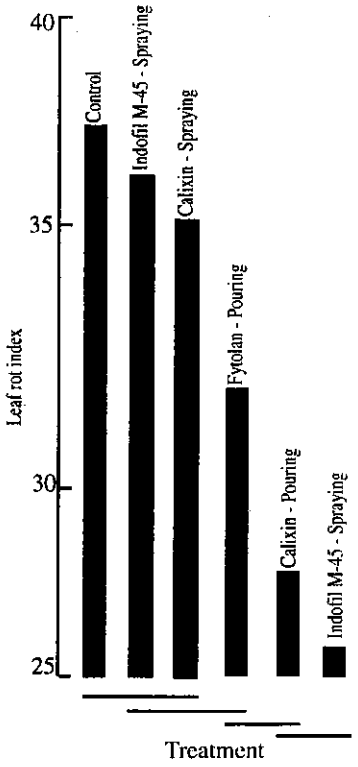

Fig 25. Effect of different fungicides on leaf rot disease

gloeosporioides E. rostratum etc. was tested *in vitro*. The growing bacterial culture as well as its culture filtrate were found to be inhibitory to the growth of all the fungi at different levels. Application of this bacterium with the inoculum of the main pathogens of LRD under *in vivo* conditions reduced the leaf rot onset/lesions development.

In order to search for native antagonistic bacteria, isolations were made from coconut phylloplane and rhizophere and 96 and 21 isolates from these zones respectively were purified and screened against the main pathogens of LRD (Anon, 1997). Assessment of these isolates lead to the detection of two isolates each from phylloplane and rhizosphere as effective native antagonists against both the pathogens.

Radha (1961) observed that Andaman Ordinary and New Guinea were more resistant than other varieties tested against leaf rot disease.

Occurrence of LRD restricted to the RWD endemic region, points to the interrelationship of these diseases. The control of LRD becomes significant because of vulnerability of RWD infected palms to fungal infection. A management system integrating phytosanitation (judicious pruning of infected spindle and a few leaves close to it from the initial stage of disease onset itself), and use of broad-spectrum systemic fungicide, biological agents etc. are of paramount importance (Srinivasan and Gunasekaran, 1998c).

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9

VARIETAL RESISTANCE

P.M. JACOB, R.V. NAIR and T.S.S. RAWTHER

Evidences for the involvement of Phytoplasma in the etiology of the root (wilt)disease of coconut have been presented in Chapter 3.5 of this publication. As the disease cannot be controlled by conventional plant protection measures, development of resistant/tolerant genotypes is an ideal solution to this malady.

Studies on identifying coconut genotypes resistant/tolerant to root (wilt) disease were initiated by Varghese in 1934. He surveyed about 10 sq. km in and around Kayangulam, a highly root (wilt) affected area and could not locate genotypes resistant to root (wilt) disease.

Trials conducted at CPCRI Kayangulam during 1951 to 1968 have shown that open-pollinated progenies of healthy palms from disease endemic areas had higher yields and lesser disease incidence than the progenies from disease affected palms (Menon *et al.*,1981).

Attempts to screen the available coconut cultivars were made as early as 1961 at CPCRI Kayangulam. The cultivars tested were Andaman Ordinary, Andaman Giant, Cochin China, Ceylon tall, Laccadive Dwarf, Laccadive Ordinary, New Guinea, Philippines, Strait Settlement Apricot, Strait Settlement Green, St. Vincent and Spicata. All these cultivars evaluated in the field developed typical symptoms of root (wilt) disease (Menon *et al.*, 1981).

Rawther and Pillai (1972) reported that natural cross dwarf coconuts (NCD hybrids) exhibited higher tolerance to the disease as compared to West Coast Tall (WCT) cultivar. These hybrids also gave higher yield in spite of contracting the disease. However, the survey had some limitations on account of insufficient number of hybrid population in the field.

Large scale field experiments were undertaken during 1972 in CPCRI Kayangulam and also in cultivators' gardens in different soil types in 63 villages of Alappuzha, Kollam and Kottayam districts. Under this programme, one year old open pollinated progenies of 36 cultivars and 43 hybrid combinations were planted in gardens that had 40 to 70 percent incidence of root (wilt) disease. The data collected from these experiments revealed that all the cultivars and hybrids contracted the disease irrespective of the soil type. However, the percentage of incidence was comparatively high in alluvial and reclaimed soil types (Table 33).

Analysis of the data collected for ten years from 21 cultivars and 15 hybrid combinations along with WCT as control, planted in CPCRI Kayangulam in a replicated trial during 1972, revealed that none of the cultivars and hybrids has tolerance to root

Table 33. Percentage of incidence of root (wilt) disease in cultivars/ hybrids planted in different soil types during 1972

Sl. No.	Cultivar/ hybrid	Sandy loam	Laterite	Alluvial	Reclaimed
1.	WCT	34.0	22.0	50.0	52.0
2.	COD	22.0	9.5	26.0	47.5
3.	CODxWCT	38.7	31.0	45.5	51.7
4.	WCTx COD	27.2	36.0	53.1	71.0
5.	WCTx Gangabondam	30.5	25.0	60.0	53.6

(wilt) disease. The percentage of disease incidence ranged from 33.3 to 100 (Table 34). The average annual yield was maximum in WCT x COD hybrid (52.5 nuts per palm) though the disease incidence was as high as 91.6%, followed by Java Giant x KGD with 43 nuts and 83.3% disease incidence and Laccadive Ordinary with 40 nuts and 58.3% disease incidence in the sixteenth year of planting.

Information gathered from the screening experiments laid out during 1972 in cultivators'' gardens (sandy loam soil) indicated that the hybrid combination CODx WCT had the average yield of 80.1 nuts/palm/year with 50% disease incidence, followed by WCT x COD with 70.3 nuts and 94% disease incidence and WCTx Gangabondam with 52.5 nuts and 87.5% disease incidence compared to 70 nuts and 37.5 % disease incidence in West Coast Tall.

New trials involving 27 cultivars, 10 hybrid combinations, F_2 (OP) of DxT and TxD, progenies of elite palm, high yielding WCT and prepotent WCT, were laid out in cultivators', gardens during 1982. Among these, 27 cultivars were planted in 5 locations and the remaining in 8 locations. Data collected from the above trials have shown that among the 27 cultivars, all except Kenthali have taken up the disease and the disease index ranged from 7.1% to 55.6%. All the ten hybrid combinations, F_2 (OP) of TxD and DxT, progenies of elite palm, prepotent and high yielding WCT were affected by the disease ranging from 12.5 percent to 66.7 percent at the fifth year of planting. Of the two hybrid combinations and the three cultivars planted during 1983, MYD contracted the maximum disease incidence (48.3%), followed by Laccadive Ordinary (33.3%) and the minimum by COD x WCT (2.6%) as compared to WCT (6.9%) at the fourth year of planting.

Twenty four exotic accessions collected in 1981 from South Pacific Ocean islands have been planted at the World Coconut Germplasm Centre at Sipighat in Andamans, with the main objectives of producing *inter se* and selfed seed nuts; and also making crosses using pollen collected from them on disease free palms located in the hotspot areas in South Kerala. These seed nuts will

Table 34. Reaction of coconut cultivars and hybrids to root (wilt) disease

Trial No.1 Year of planting: 1972; Location: CPCRI Kayangulam

Sl. No.		No. of palms	Percentage of disease incidence	Annual yield of nuts
a)	**Cultivars**			
1.	Andaman Giant	12	100.0	4.5
2.	Andaman Ordinary	12	33.3	18.4
3.	B.S.I.	12	100.0	9.7
4.	Car Nicobar	12	92.0	22.1
5.	Chowghat Orange Dwarf	12	66.6	30.7
6.	Cochin China	12	50.0	14.4
7.	F.M.S.	12	33.3	18.7
8.	Gangabondam	12	92.0	13.3
9.	Java Giant	12	92.0	25.4
10.	Jamaica	12	66.6	24.1
11.	Kulasekharam Green Dwarf	12	66.6	22.0
12.	Kulasekharam Orange Dwarf	12	66.6	24.1
13.	Laccadive Micro	12	75.0	36.7
14.	Laccadive Ordinary	12	58.3	40.0
15.	M2 irradiated	12	100.0	28.2
16.	Malayan Green Dwarf	12	83.0	31.4
17.	Malayan Orange Dwarf	12	66.6	15.4
18.	Malayan Yellow Dwarf	12	100.0	29.2
19.	Philippines Ordinary	12	41.6	21.9
20.	SS Apricot	12	92.0	19.9
21.	SS Green	12	83.3	14.1
22.	West Coast Tall	36	61.0	29.0
b)	**Hybrids**			
1.	COD x WCT	12	100.0	32.8
2.	Fiji x Gangabondam	12	66.6	16.4
3.	Fiji x SS Green	12	75.0	31.1
4.	Jamaica x Gangabondam	12	75.0	12.2
5.	Java Giant x KGD	12	83.3	43.0
6.	Java Giant x KOD	12	83.3	29.5
7.	Java Giant x KOD	12	33.3	38.2
8.	KGD x Java Giant	12	50.0	35.0
9.	KOD x Java Giant	12	75.0	37.0
10.	KYD x Java Giant	12	83.3	35.5
11.	Laccadive Ordinary x San Ramon	12	66.6	11.7
12.	San Ramon x Gangabondam	12	66.6	13.1
13.	WCT x COD	12	91.6	52.5
14.	WCT x Gangabondam	12	91.6	24.4
15.	WCT x MYD	12	66.6	13.1

be planted in 'hot spot' areas for screening for resistance/tolerance to root (wilt) disease.

A large scale screening trial with three replications was initiated in 1985, in the Kayangulam Kayal farm, 16 km from CPCRI involving 33 Talls, 6 Dwarfs and 9 hybrid combinations with plot size of 16 seedlings have been laid out. The cultivars and hybrids were monitored regularly for their reaction to root (wilt) disease. The first symptom was noticed 30 months after planting. Of the 17 cultivars and five hybrids planted during 1985, twelve cultivars and all the five hybrids have been affected by the disease with the disease incidence ranging from 2.3 to 11.6%, the maximum being recorded in WCTx Gangabondam hybrid. Among the 10 cultivars and four hybrids planted during 1986, Markham Tall and Fiji Tall contracted the disease with an index of 2.1 and 2.3 percent, respectively. The trial had to be unfortunately discontinued.

The elite palms identified in the diseased tract (Iyer *et al.*, 1979) were monitored for their yield and reaction to root (wilt) disease (see back cover). Among the 9 elite palms under observation, three have been affected by root (wilt) disease [Thazhava, Champakulam and Krishnapuram (Kappil)] and their yield level ranged from 96 to 156 nuts per palm per year (Anon., 1988). All the open pollinated progenies of these elite palms planted in CPCRI Kayangulam during 1980, have contracted the disease ranging from 40% to 100%. However, the progenies of Thazhava palm showed their superiority over the others in yield with an annual average yield of 100 nuts in the eighth year of planting.

As an initial step towards breeding for resistance/ tolerance to root (wilt) disease, an intensive survey covering the heavily root (wilt) affected areas (hot spots) of Kottayam, Alappuzha, Pathanamthitta and Kollam districts of Kerala, was initiated during 1985 to identify disease free and high yielding palms (Anon., 1986). Occurence of high yielding palms among the heavily diseased palms was reported as early as 1953 by Davis. The survey was further intensified during 1988. Chowghat Green Dwarf (CGD) which was reported to show maximum field tolerance (over 90%) to root (wilt) disease (Anon, 1972) was also included in the survey. The criteria for the selection of West Coast Tall (WCT) and Chowghat Green Dwarf (CGD) mother palms were as follows :

WEST COAT TALL (WCT)

1. They should yield 80 or more nuts per palm per year
2. They should be regular bearers, the bunches being well supported by the petioles
3. They should be free from all diseases and pests
4. They should show negative reaction to root (wilt) disease in the sero-diagnostic tests
5. They should be 35 or more years of age
6. They should be surrounded by 80% or more root (wilt) affected palms preferably in the advanced stages of disease

7. They should have all typical characters of WCT confirmed by the progeny test to make sure that no hybrid palm is selected as mother palm

CHOWGHAT GREEN DWARF (CGD)

1. Annual yield should be over 100 nuts per palm per year
2. Age of the palm should be 20 years or more
3. Should be serologically negative to root (wilt) disease
4. Should be free from all diseases and pests
5. Should posses all the typical characters of CGD with regard to stem, crown, leaf, nut and inflorescence
6. Should be surrounded by 80% or more root (wilt) affected palms preferably in the advanced stages of disease

A total number of 187 mother palms were selected and employed in the crossing programme (Table 35).

Hybridization involving disease-free elite WCT and CGD mother palms in hot spots of Kottayam, Alappuzha, Pathanamthitta and Kollam districts was carried out in the following cross combinations for generating progenies to study their reaction to root (wilt) disease (Nair *et al.*, 1996).

1. WCT x WCT (*inter se*)
2. WCT x WCT (Mixed pollen)
3. WCT selfed
4. WCT x CGD (TxD)
5. CGD x CGD (*inter se*/selfing)
6. CGD x WCT (DxT)

Artificially pollinated seedling progenies of different cross combinations mentioned above were underplanted since 1991 in different blocks at CPCRI Regional Station, Kayangulam, where more than 80% of the existing palms were affected by root (wilt) disease. A total of 2423 artificially pollinated progenies raised by crossing the disease free elite WCT and CGD mother palms in hotspots have been planted so far, from 1991 to 1997 as shown in Table 36.

Observations on the first batch of 31 CGDxWCT hybrid progenies planted during 1991 at CPCRI Regional Station at Kayangulam revealed that the palms have an yield potential of over 100 nuts per palm per year in the fifth year of planting and came to flowering in 30 to 40 months after planting. The copra content was 215 g/nut. Characteristic symptoms of the root (wilt) disease were noticed only in 5 palms in the eighth year of planting (16%).

On the other hand, the performance of 50 CGDxWCT hybrid progenies brought from a disease-free area (Kasaragod) and planted in 1990 at Kayangulam revealed that the percentage of disease incidence was 60 in the fifth year of planting itself (Fig. 26). This has highlighted the scope of developing the CGDxWCT hybrid from the disease-free CGD and WCT palms in disease endemic areas, as the ideal planting material.

The *inter se* crossed and selfed progenies of WCT from disease-free elite mother palms in hotspots are also being evaluated. The first batch of progenies planted during 1992 are in the initial stages of flowering. Preliminary indications show that the root (wilt) disease incidence is nearly 30% as compared to 50% in the progenies raised from open pollinated seednuts from the mother palms under crossing. This clearly

Fig. 26. CGD x WCT hybrid

Table 35 : Yearwise details of mother palms under artificial pollination in hotspots

	No. of mother palms employed in the crossing programme		
	WCT	CGD	Total
1988-89	25	–	25
1989-90	30	6	36
1990-91	32	–	32
1991-92	40	8	48
1992-93	67	10	77
1993-94	104	5	109
1994-95	95	30	125
1995-96	85	20	105
1996-97	76	111	187

Table 36 : Details of seedlings planted at CPCRI farms at Kayangulam for screening

Year	Block	WCTxWCT			WCTxCGD	WCT	CGDxWCT	CGDxCGD	Total
		B.P.	M.P.	Self	TxD	O.P.	DxT	DxD	
1991	4	74	-	-	-	-	31	-	105
1992	1,4	286	172	79	-	20	14	20	591
1993	4, 6, 7	86	206	46	-	124	-	24	486
1994	3,5,6,11	105	390	53	-	47	88	24	707
1995	3,4,5	257	-	15	23	40	15	-	350
1996	2,3	37	-	77	26	36	44	-	220
1997	6,7	166	-	-	65	-	-	-	231
	Total	1011	768	270	114	267	192	68	2690

B.P. - Biparental, M.P. - Mixed pollen, O.P. - Open pollinated

demonstrated the better performance of the artificially pollinated progenies over the open pollinated progenies in the disease endemic areas.

Seed gardens one each at CPCRI Research Centre, Kannara (Thrissur district) and Coconut Development Board Farm at Neriamangalam (Ernakulam district) have been established for large scale production of disease tolerant planting materials such as WCT x WCT, WCT x CGD and CGD x WCT hybrids for distribution to farmers in the disease endemic districts in Kerala State. Details of the planting are given in Table 37.

Breeding for resistance/ tolerance to coconut root (wilt) disease initiated in 1987-88 is showing encouraging results and indicates the possibility of evolving resistant/ tolerant high yielding CGDxWCT and WCTxWCT hybrids for combating the root (wilt) disease problem.

Table 37 : Details of seedlings planted for establishing seed gardens

Location	Year of start	No. of seedlings planted			Total
		WCT X WCT	WCT selfed	CGD X CGD *inter se*/selfed	
CPCRI (RC) Farm, Kannara	1995	350	200	250	800
CDB Farm, Neriamangalam	1996	250	_	100	350
Grand Total		600	200	350	1150

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10

MANAGEMENT

A. MURALIDHARAN, N.P. JAYASANKAR, K.J. ANTONY and P. RETHINAM

Root (wilt) disease of coconut caused by Phytoplasma has no definite control measures. However, the bearing palms which are in initial to middle stage of disease intensity respond well to the management practices. Experiments carried out in the past have clearly brought out results in favour of formulating strategies for containing the disease in its present geographical limits (Anon., 1986) and managing the disease for sustained productivity of the palms (Sahasranaman *et al.*, 1964; Menon and Nayar, 1978; Muralidharan *et al.*, 1986; Rajagopal *et al.*, 1987).

The importance of plant nutrition in reducing the incidence and severity of pathogenic diseases has been recognised for many years (Huber, 1981). Considerable work was done to study the role of macro and micronutrients on the incidence of root (wilt) disease and on the yield of the disease affected palms as reviewed under Chapter 5. Based on these studies the CPCRI (Anon., 1986) recommended a balanced dose of 500 g MgO per palm per year for the root (Wilt) affected areas to maintain the productivity of the palms.

Eradication of disease affected palms to contain the disease within the contiguously infected geographic limits has been very successful. Uprooting and burning of these diseased palms which occurred in an isolated garden in Vellum village, Shencottai in Tamil Nadu during 1971 prevented recurrence of the disease in the area (Anon., 1986). Based on this encouraging result an attempt was made to create a buffer zone in the sparsely affected areas north of Karuvannoor river in Thrissur District by eradicating nearly 28,000 diseased palms. A garden to garden survey to locate diseased palms was conducted (Rethinam *et al.*, 1982) in 10 villages adjoining north of the buffer Zone during 1979-82. A total of 730 palms in 341 gardens were confirmed to be diseased. These diseased palms were sprayed with 0.05 percent carbaryl to suppress the vector *Stephanitis typica*, before eradication. The bole and roots of the eradicated palms were burnt *in situ*. Replanting was done with elite seedlings. Eradication of diseased palms was continued as and when noticed in succeeding years. These operations have brought down the recurrence of the disease to just 21 palms spread over in 15 gardens of 3 villages in 1989-90 (Table 38).

In an integrated approach to eradicate root (wilt) disease on a large area basis and to increase productivity of the coconut, the Central Plantation Crops Research Institute in collaboration with the Kerala Agricultural University, Department of Agriculture, Kerala State, the Fertilizers and Chemicals Travancore Ltd., (FACT) Alwaye and Indian Overseas Bank,

Table 38. Effect of eradication on disease recurrence in Thrissur District

Year	No. of Villages	No. of gardens	No. of diseased palms
1979-82	10	341	730 *
1983	8	18	21
1984	1	5	8
1985	6	17	21
1986	4	19	22
1987	3	12	25
1988	7	21	39
1989-90	3	15	21

* Total number of diseased palms eradicated in the beginning.

Varandarapally Branch, (a local financing agency) implemented a scheme in 1980-81 in Varandarapally village in Thrissur District, (Anon., 1983; Anon., 1990). The area of operation comprised 50 ha with a total of 5739 palms having an average yield of 28 nuts per palm per year. The schemes involved identification of all diseased palms, spraying them with 0.05% carbaryl, cutting and removing the diseased palms with the bole and roots, and burning them *in situ*. Quality coconut seedlings were used for replanting and a compensation of Rs. 75/- was given for one diseased tree eradicated. All the coconuts in the area were fertilized as per recommended practices. Loans were given for providing irrigation facility wherever necessary. Surveillance of the disease affected gardens for disease recurrence was carried out periodically. An evaluation of the scheme conducted after three years showed that the disease incidence was 79 in 1980-81 prior to implementation of the scheme, with a recurrence of the disease in 20 palms in 1982 and 2 palms in 1985. The productivity increased to 51 nuts per palm in diseased gardens (83% increase) compared to 28 nuts per palm prior to implementation of the scheme. Thus, coordinated and cooperative efforts of all agencies concerned helped to check the root (wilt) disease and increase productivity through scientific management over a large area.

Experiments conducted in farmers fields (Muralidharan *et al.*, 1986) have shown that disease advanced palms never respond to any kind of management practices. Juvenile palms that contract the disease before the onset of flowering seldom yield (Ramadasan *et al.*,1971). These observations necessitate removal of all infected juvenile palms and bearing palms in advanced stages of the disease.

Multiple cropping and mixed farming systems in root (wilt) affected coconut gardens have indicated to exert ameliorating effect on the disease affected palms. Menon and Nayar (1978) reported that intercropping with cassava, elephant foot yam and greater yam for five years in a 16 year old coconut garden planted at 7.5 m x 7.5 m, gave an over all increase in nut yield of root (wilt) affected palms to the extent of 5, 15 and 8 percent respectively. They also reported that in plots intercropped with cassava the intensity of root (wilt) disease was on the increase (indices 32.1 to 37.3) whereas in other plots intercropped with elephant foot yam and greater yam there was a decline (37.0 to 33.3). Antony (1983) studied the effect of intercropping with

cassava, elephant foot yam, colocasia, greater yam, ginger and turmeric and found that there was no significant change in disease indices and yield in affected palms under rainfed condition.

Amma *et al.* (1983) reported that yield of coconut in a root (wilt) affected garden has increased by 27 to 35 per cent as a result of mixed cropping with cocoa. The increase was maximum under double hedge systems of cocoa planting. The superiority of double hedge system in increasing the yield of coconut was also reported earlier by Nair *et al.* (1975). However, data collected from the same experiment after 10 years have shown that the yield of coconut was more under single hedge system of cocoa (Anon.,1988).

The effect of mixed farming on disease amelioration, yield of root (wilt) affected palms and soil fertility was investigated in detail (Sahasranaman *et al.*, 1983). Considerable reduction in foliar yellowing was observed due to mixed farming but flaccidity and necrosis increased indicating that the practice had no curative effect on the disease. Beneficial effects like rejuvenation of roots of disease affected palms, increase in soil organic carbon, available P and exchangeable Ca, Mg and K in the sub-soil were also reported. A net increase of 28.1 per cent in the yield of root (wilt) affected palms was also reported (Table 39). The response was high in disease early palms and low in disease advanced palms (Figs. 27 and 28).

A field experiment was conducted in a farmers garden in laterite soil type with the palms in the early and middle stages of disease to study the influence of basin management with green manure legumes along with recommended dose of fertilizers and 500g MgO on root(wilt) disease. The biomass generated by *Pueraria phaseoloides, Mimosa invisa* and *Calopogonium mucunoides* in a growth period of 130 days during the monsoon period in coconut basin was 24.6, 27.9 and 24.9 kg/basin and the legume biomass was incorporated in respective basins.

There was no significant change in the disease condition of the palms due to legume treatment. But the yield of palms increased as a result of legume treatment, (Table 40) fertilizer application and adoption of plant protection measures (Thomas *et al.*, 1993).

Studies on the regeneration of the root system of disease affected palms using growth hormones (IBA and NAA), thiamine (amino acids, vitamins, glutamic acid) and phenols (chlorogenic acid, caffeic acid, gallic acid and coumarin) showed that the application of 500 ppm IBA+ 400 ppm phenols produced 51 fresh roots compared to two roots in the control and reduction in disease index from 35 to 26 within a period of one year (Amma and Patil, 1982). Dwivedi *et al.* (1980) reported that ascorbic acid (400 ppm fed through cut root) not only reduced disease symptoms but also improved the yield of palms. However, studies made by Rajagopal *et al.*, (1986b) ruled out the role of ascorbic acid in reducing the disease indices or increasing the yield.

Rajagopal *et. al.* (1986a) found that

Fig. 27. Root (wilt) disease managed garden in farmer's field

Fig. 28. Farmers having discussion with the scientists in the root (wilt) disease management demonstration plot

Table 39. Effect of mixed farming on nut yield of root (wilt) palms

Disease index	Pre-treatment yield		Post-treatment yield		Increase over pre-treatment (%)		Estimated net response due to mixed farming (%)
	Control	Treat.	Control	Treat.	Control	Treat.	
0-10	58.3	50.3	62.4	68.4	7.0	36.0	29.0
11-25	31.1	33.7	29.2	42.3	-6.1	25.5	31.6
26-50	20.9	22.5	18.1	24.5	-13.4	8.9	22.3
> 51	19.5	19.0	21.4	23.7	9.8	24.7	14.9
Overall	32.5	31.4	29.8	39.6	-2.0	26.1	28.1

Table 40 : Effect of basin management with green manure legumes on root (wilt) disease and yield of coconut palms

Treatment	Root (wilt disease index		Nut yield/palm/year	
	Pre-treatment	Post-treatment	Pre-treatment	Post-treatment
M. invisa	18.00	17.91	32.32	54.46
P. phaseoloides	12.38	16.58	39.51	63.84
C. mucunoides	21.12	20.91	42.96	52.21
Control	19.38	24.18	26.50	38.93
LSD (P=0.05)		NS		17.12

the flaccidity on leaflets of root (wilt) affected coconut palms was associated with impaired stomatal mechanism leading to increased rate of transpiration. Summer irrigation increased water up-take and reduced transpiration rate of root (wilt) affected palms, thus bringing about balanced water economy to the disease affected palms (Rajagopal *et al.*, 1986 b). From a field experiment, Rajagopal *et al.*, (1987) reported significant decline in disease index, due to summer irrigation at the rate of 250 l water per palm per week. The irrigation and fertilizer managed palms showed 69.7% improvement while there was 78.5% deterioration when both these inputs were absent (Table 39). There was significant reduction to the extent of 51, 60, 40, 60 and 45 per cent respectively in flaccidity, yellowing, leaf rot, senescence and necrosis due to irrigation over the pre-treatment. There was an overall increase of 53.3 per cent of the photosynthetic area with an increase in yield of 10 nuts per palm over the pretreatment (Fig. 29).

Fig. 29. A bearing palm under well managed farmer's garden

Average yield of palms in a root (wilt) disease affected garden increased from 17.6 to 46.0 nuts/palm/year due to combined adoption of irrigation, plant protection measures, mixed cropping and fertilizer application (Anon., 1982). Nambiar (1984) reported that inspite of an increase in the disease index from 23.7 to 34.2, the yield level of palms was maintained over a period of six years under good management practices in a heavily infected area. A large scale demonstration on the eradication of root (wilt) disease affected palms in a mildly infected area of Thrissur district (Anon., 1986) revealed an increase of yield from 20.2 to 49.2 nuts/palm due to the adoption of improved management practices over a period of four years from 1981 to 1985.

Muralidharan *et al.* (1986) reported beneficial effect of integrated management practices on yield and disease condition of palms. An average increase of 24.3 nuts/palm/year (Table 42) was observed under rainfed conditions. This response was mainly derived from the apparently healthy and disease early palms. Increase in yield by adopting management practices was also observed under irrigation (Table 43).

Strategies have been evolved for managing the disease in the mildly affected border districts and severely affected contiguous areas (Anon., 1982, 1986). Based on the available experimental findings, the following management practices are recommended.

1. Application of balanced doses of fertilizers (500g N, 300 g P_2O_5, 100 g

Table 41. Effect of summer irrigation on the condition of root (wilt) diseased coconut palms.

Treatments	No. of palms observed	No. of palms showing		
		Improvement	Deterioration	No change
Irrigation + Fertilizers	33	23 (69.7)*	5(15.1)	5(15.1)
Fertilizer alone	46	14(30.4)	20(43.4)	12(26.2)
No fertilizer, no irrigation	14	2(14.3)	11(78.5)	1(17.2)

* Values in paranthesis indicate percentage on number of palms observed.

Table 42. Effect of integrated management practices on yield of root (wilt) affected palms (rainfed)

Disease index	Yield (nuts/palm/year)				Increase in nut yield over control	
	Pre-expt.		Post-expt.			
	Control	Treat.	Control	Treat.	Number	%
0 - 10	40.8	43.5	51.3	80.9	29.6	57.7
11 - 25	29.9	26.4	35.5	64.3	28.8	81.1
26 - 50	18.7	17.7	21.2	29.6	8.4	39.6
>51	9.7	8.0	9.4	11.4	2.0	17.5
Mean	33.4	32.6	41.0	65.3	24.3	59.3

Table 43. Effect of integrated management practices on yield of root (wilt) affected palms (irrigated)

Disease index	Yield (nuts/palm/year)				Increase in nut yield over control	
	Pre-expt.		Post-expt.			
	Control	Treat.	Control	Treat.	Number	%
0 - 10	52.4	54.4	58.2	72.1	14.0	24.1
11 - 25	27.5	32.6	35.5	61.5	26.0	73.0
26 - 50	13.3	18.6	21.5	25.1	3.0	17.0
>51	9.8	10.2	10.4	7.8	-2.6	-25.0
Mean	35.8	43.6	45.1	62.3	17.2	36.0

K_2O and 500g MgO/palm/year in two splits. One-third dose to be applied during April-May and two-thirds during September-October for rainfed palms and in four splits during January, April, July and October for irrigated palms.

2. Control of leaf rot disease and rhinoceros beetle are given below:

a. Cut and remove rotten portions of only spindle and two top most fully opened leaves.

b. Mix Hexaconazole (Contaf 5 EC) @ 2ml per palm and Dithane M-45/ Indofil M-45 @ 3 gm per palm in 300 ml water. Pour around the base of spindle leaf.

c. Apply Furadan around the base of the spindle.

d. Apply Sevidol (8g @ 50 gm/palm) mixed with 200 CCC river sand around the base of the spindle.

(The above treatment, including labour charges, costs around Rs. 20/palm.)

3. Application of 50 kg farm yard manure per palm per year.

4. Irrigating palms during summer months atleast @ 250 l/palm/week.

5. Application of 50 kg farm yard manure per palm per year.
6. Provision of proper drainage wherever found necessary.
7. Growing green manure crops, preferably *Peuraria phaseoloides* in basins during the period April-May to September-October and incorporation.
8. Raising intercrops in rotation/ adopting mixed cropping/mixed farming with recycling of organic matter.
9. Removal of all disease advanced and uneconomic palms and replanting with healthy elite seedlings if the planting density is less than 175/ha.
10. Removal of juvenile palms showing symptoms of root (wilt) disease irrespective of its intensity.
11. In mildly infected areas all disease affected palms, irrespective of the intensity of disease and yield of palms, should be removed to eliminate the foci of infection.
12. Restructuring the canopy of other perennial tree crops to provide maximum light for the coconut palms.

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EPILOGUE

Since the first record of the coconut root (wilt) disease a century ago in three pockets around Kollam and Kottayam, it has spread in a contiguous manner to eight southern districts of Kerala from Thrissur to Thiruvananthapuram. The loss caused by this disease is estimated to be about 968 million nuts in Kerala. The disease occurs either contiguously or scattered in patches in all soil types, and palms of all ages are susceptible. Studies conducted so far have clearly shown that the earliest visual symptom of the disease is the flaccidity of the leaflets. Serological and physiological tests have been standardized for disease detection prior to the expression of visual symptoms. Every aspect of crop production and management from soil factors, physiological processes and pathological aspects have been looked into.

Systematic investigations carried out over a period of more than three decades have ruled out the role of any physiological/ nutritional disorders as the primary cause of the malady. The effect of Rare Earth Elements occurring in the monazite belt needs to be investigated carefully. Several biotic agents like fungi, bacteria and nematodes reported earlier to be associated with the disease could not reproduce symptoms characteristic of the disease. Even though a viral etiology was presumed in the sixties, further studies have ruled out the association of any virus or virus-like particles with this malady.

In this context, the consistent presence of phytoplasma in the tissues of a large number of diseased palms as revealed by serum diagnosis as well as electron microscopy and its total absence in the disease-free palms assumes significance. Successful transmission of the disease by lace bug *(Stephanitis typica)* and plant hopper *(Proutista moesta)* under insect-proof conditions has established the phytoplasmal etiology unequivocally. Partial remission of symptoms observed consequent to the application of oxytetracycline has lent additional credance to phytoplasmal etiology.

The earliest attempt to demonstrate the possibility of preventing the recurrence of the disease in a mildly affected area around Shencottah in Tamil Nadu, was made when three diseased palms located there were eradicated in 1971 and the absence of recurrence was observed. Against this backdrop, a massive effort was made by CPCRI in Thrissur district to contain the disease with the natural boundaries of a river (Karivannur) and to prevent its further spread to newer areas by creating a buffer zone of disease free belt. When the foci of infection were removed by the eradication of all diseased palms, results were very promising. This strategy had been welcomed by the developmental agencies as well, leading to joint efforts by CPCRI, Coconut Development Board, Agriculture Department of Kerala and the Kerala Agricultural University for removal of diseased and senile palms.

Adoption of recommended cultivation practices of fertilizer and organic matter application, irrigation, intercropping as well as pest and disease control makes it possible to obtain sustainable yield. Formulation of an effective and comparatively cheaper control measure for leaf rot is a step in the right direction. The palms in the advanced stages of the disease do not respond to management and juvenile palms that contract the disease before the onset of flowering do not give economic yields. The strategy developed to combat the disease in the heavily affected contiguous tract, therefore, consists of eradication of highly diseased and unproductive palms as also young palms that have taken up the disease prior to flowering. Refinement of the existing management technology to make it affordable and to obtain a higher benefit/cost ratio, is needed.

Use of satellite imaging would aid in the detection of the extent of spread of the disease and also aid in understanding the incidence and intensity of disease in other areas.

The sero-diagnostic test developed for the detection of coconut root (wilt) disease before the onset of visual symptoms needs further refinement to make it fool proof. The possible application of ELISA and PCR techniques for the rapid screening of large number of samples for diagnosis needs to be explored. Monoclonal Antibody Technique by which phytoplasma specific clones are exclusively selected and propagated, is worth exploiting.

Host plant resistance seems to be the most promising and ideal solution. Elite disease free, high yielding adult palms are often encountered in heavily diseased gardens. *Inter se* mating/selfing of these palms are being effected to generate resistant/field tolerant progenies for further evaluation and selection. Field tolerance observed in CGD X WCT hybrids generated by crossing disease free parents located in hot spots, is very encouraging. Seed gardens are being established for the large scale production of such hybrids in the hot spot areas. Germplasm collections from Indonesia and Pacific and Indian Ocean Islands can be expected to offer further sources of resistance, thus broadening the genetic base for breeding newer combinations. Apart from the experiments currently in vogue, the possibility of perfecting an *in vitro* screening technique is also under consideration.

Application of DNA finger printing techniques for screening germplasm, hybrids/cultivars which exhibit resistance/tolerance to the disease; and development of molecular probes to identify and characterize the pathogen for molecular diagnosis should receive priority in research.

Studies to identify the environmental and edaphic factors responsible for the geographical delineation of the root (wilt) disease and evolving management practices to modify the crop environment to make it

less favourable for the pathogen or its vector, are areas that merit attention.

It is high time that we fully exploit the most modern tools of research available at our hand and effectively use them for tackling the menace of root (wilt). Adequate funds must be made available for the most modern tools of research to be employed in root (wilt) research and to find a lasting solution to this scourge of Kerala.

Considering all the leads available, it would be of utmost importance that we develop a comprehensive view of this complex disease whose primary incitant is the MLO which is transmitted by winged vector and the decline of the palm is further aggrevated by soil and climatical factors, and the leaf rot organisms leading to total loss of the palms. Comparisons have been made with the more deadly MLO of the Lethal Yellowing Disease of Carribian region, which kills the palm within 6 months, whereas in RWC, MLO is a slow killer, causing a gradual decline in yields. In fact, the name "Coconut Decline" had therefore, been suggested to replace the long established name "Root (wilt) disease".

AUTHOR INDEX

ERRATA

Page	Mistake	Should read as
25	phenamiphos 2 10ga.i./palm	phenamiphos @ 10g.a.i/palm
66	4.20 to 4.35 mg $cm^{-2}Sec^{-1}$ 9.15 to 10.50 mg $cm^{-2}Sec^{-1}$	4.20 tp 4.35 µg cm^{-2} Sec^{-1} 9.15 to 10.50 µg $cm^{-2}Sec^{-1}$
75 (Fig 15)	2 50 CM DEPTH	25-50 CM DEPTH
76 (Fig 16 Scale on Y-axis for 50-100 CM DEPTH)	0, 40, 80, 120, 160, 200, 240	0, 10, 20, 30, 40, 50, 60
83	LRD infected so palm	LRD infected palms
88	(Fig 21 and 22)	(Table-31 and Fig. 22)
90	(Fig. 24)	(Fig. 23)
92	Fytolan 0.3%	Fytolan 0.5%
93 (Fig. 25 Treatment No. 2 X-axis	Indofil M-45 spraying	Indofil M-45 pouring
105	dose of 500g MgO	dose of 500g N, 300g P_2O_5, 1000g K_2O and 500 g MgO
110	500N, 300g P_2O5, 100g K_2O	500N, 300g P_2O_5, 1000g K_2O