COCONUT ROOT (WILT) DISEASE

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13-1 INTRODUCTION

Considerable data have accrued from the investigations carried out over a period of time on this palm disease. Citations have been restricted after careful scrutiny, and references have been made only to consistent observations. Butler[1] referred to this malady as root disease, probably on the basis of his observations on the rotting of roots. But Nagaraj and Menon[2] felt that the name "wilt" was a more apt term because of the foliar symptoms. At present, it is known as coconut root (wilt) disease.[3,4]

The disease is not lethal but debilitating, occurs in all soil types, and has a geographical delineation. Symptoms are well defined and the nature of the causal agent fairly well understood. Diagnostic measures are available for the detection of the disease before the onset of visual symptoms. It causes an annual loss of around 968 million nuts, and absolute control of the disease with the use of any chemical may be a difficult proposition.

Apparently healthy palms and palms in the early stage of the disease respond to care and management. The canopy and the pattern of land utilization of the crop enable cultivation in the interspaces, which will increase income from unit area. The current strategies that have crystalized are to "live with the disease" in the heavily affected contiguous core and to contain the disease by preventing its spread to newer areas. Coconut root (wilt) disease has been dealt with in detail by Menon[5] and reviewed since then by Lal,[6,7] Shanta and Radha,[8] Jayasankar,[9] Nayar and Jayasankar,[10] and Jayasankar and Bavappa.[11]

13-2 OCCURRENCE AND DISTRIBUTION

Coconut root (wilt) disease was recorded around 1874 from Erattupetta in the erstwhile state of Travancore (now Kerala), India. It became significantly noticeable after the floods of 1882.[12] Around 1897 the disease was independently reported from Kaviyoor-Kallooppara and Karunagappally, approximately 50 km away from each other (Figure 13-1). The disease subsequently started spreading to adjoining areas from these foci. Several attempts have been made in the past to determine the spread of the disease.[1,13,14] Indications are that the disease occurs in sporadic isolated pockets and is spreading in all directions and in all soil types. Palms of all ages are susceptible; disease-free gardens

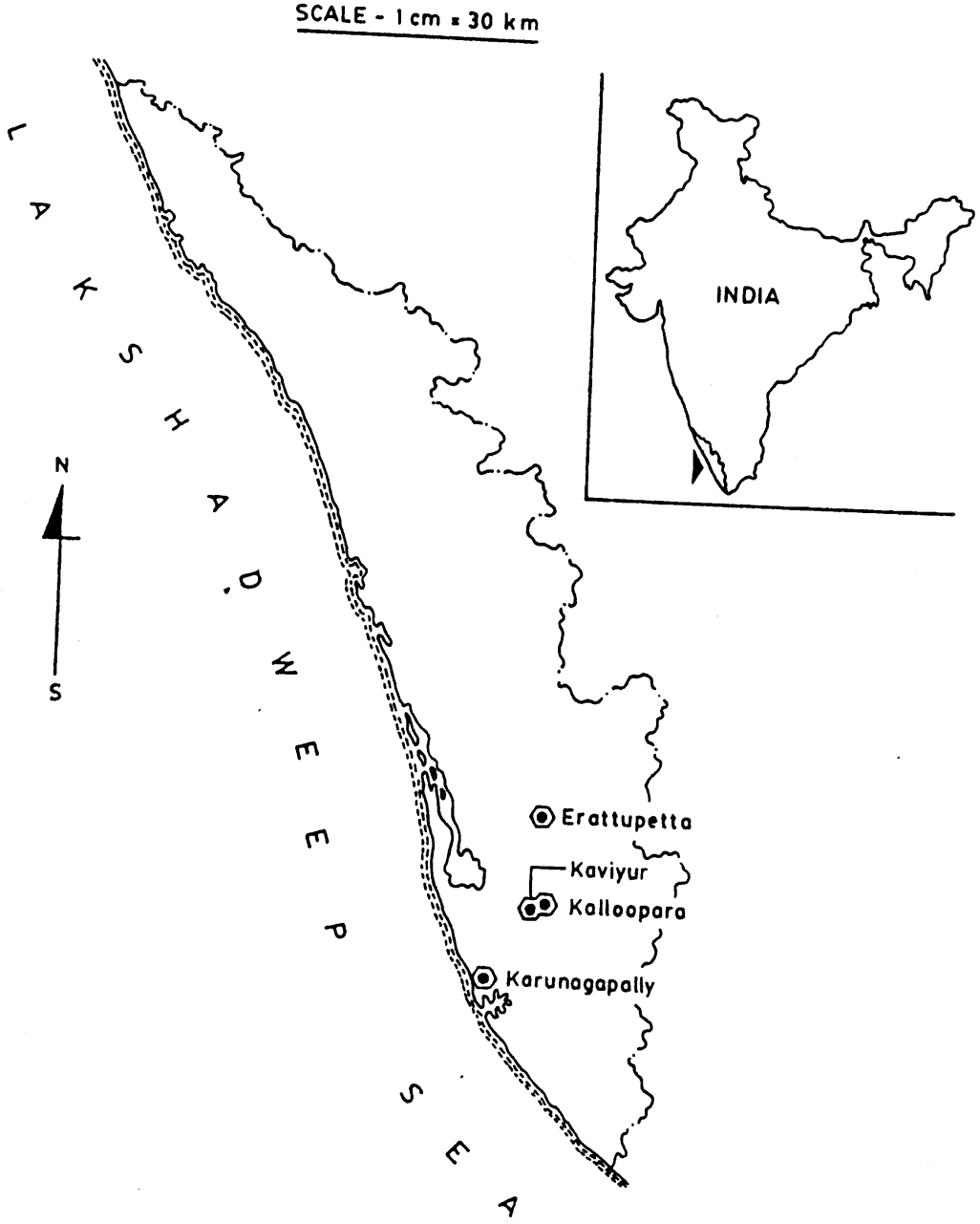

Figure 13-1 Origin and distribution of coconut root (wilt) disease.

occur rarely in the midst of heavily diseased areas, and apparently healthy palms in heavily infected gardens.

A comprehensive survey undertaken in recent years reveals that the disease occurs in a contiguous manner in 4.1 million ha affecting an estimated population of 59.2 million bearing and 32.4 million nonbearing palms.[15] The disease tract has a contiguous core in the southern districts of Kerala state (Figure 13-2). It has also been observed in a sparse manner in isolated pockets in the remaining districts in northern Kerala and in the adjoining districts of Kanyakumari and Coimbatore in the state of Tamil Nadu (Figure 13-2). The disease does not occur in any other parts of the country.

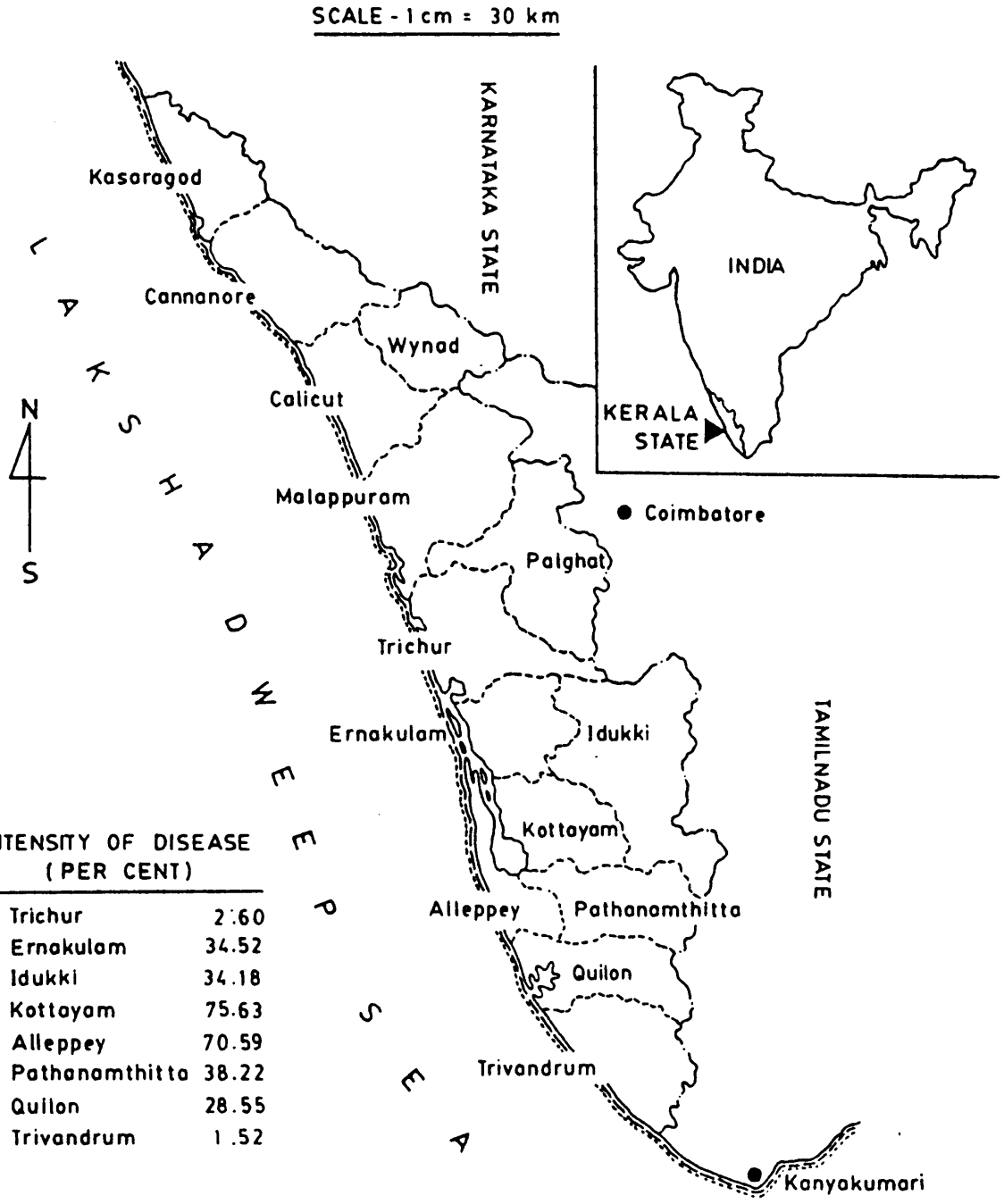

Figure 13-2 Intensity and production loss consequent to coconut root (wilt) disease.

Within the contiguous tract the intensity of the disease varies. The highest intensity of 75.63% is noticed in the Kottayam district, followed by the Alleppey district with an intensity of 70.69%. The intensity tapers toward Trichur in the north (2.60%) and Trivandrum in the south (1.52%). The survey has also revealed an annual loss of 968.09 million nuts consequent to the development of the disease. The districtwise intensity of disease and production loss are furnished in Table 13-1.

13-3 SYMPTOMS

Wilting and drooping of the leaves, flaccidity, paling and yellowing, and necrosis of leaflets were considered to be the typical foliar symptoms of the disease.[12,13] However, a clear understanding of the symptom picture has emerged on the basis of the observations of the foliar symptoms of 7,000 palms of varying age groups.[16] Accordingly, the characteristic bending and ribbing of the leaflets of the leaves in the central and outer whorls is the earliest consistent visual symptom (Figure 13-3). Foliar yellowing and marginal necrosis, the other two symptoms, set in with the advancement of the age of the palms and intensity of the disease (Figure 13-4). Flaccidity is considered to be the most consistent symptom in palms of all ages in all soil types.[16]

An indexing method has been worked out for quantitatively scoring the intensity of the disease based on grade points assigned separately for flaccidity, yellowing, and necrosis. Based on the scores, it is possible to classify the diseased palms as early, middle, and advanced. Subsequently, this method has been simplified for ease of operation by restricting the rating of the three major symptoms in the leaves of any five spirals.[18]

Extensive root damage has been reported in diseased palms,[13,19] and the rotting of roots has been considered to be one of the symptoms of the disease.[1,12,20] On the other hand, significant differences in the root decay between healthy palms and those in the early stage of the disease could be traced by many workers.[2,7,21,22] These variations can be attributed to differences in sampling and the techniques adopted in the excavation of the root system, location, the depth of zone studied, and the age of the palms.

Anatomical studies of roots revealed degenerated phloem, disorganized trachaeal elements, and tylosis in the metaxylem in diseased palms.[23,24] The occurrence of phloem anomalies in both the roots and the pinnae of palms affected with the disease was reported by Govindankutty.[25] The observation of the internal browning in the stelar tissues with

TABLE 13-1: INTENSITY AND PRODUCTION LOSS IN THE CONTIGUOUS CORE

Serial No.		Intensity (percentage)	Production Loss (millions of nuts)
1	Trivandrum	1.52	11.34
2	Quilon	28.55	110.56
3	Pathanamthitta	38.22	99.39
4	Alleppey	70.69	271.02
5	Kottayam	75.63	254.39
6	Idukki	34.18	31.11
7	Ernakulam	34.52	177.13
8	Trichur	2.60	12.63

Figure 13-3 Flaccidity: Earliest consistent foliar symptom of the disease.

vascular discoloration in the roots of diseased coconut palms[23] was reconsidered by Dwivedi et al.[26] It was shown that vascular discoloration is not observed in the presence of an antioxidant.

13-4 DIAGNOSIS

Several attempts have been made to develop diagnostic tests to detect the disease before the onset of visual symptoms. A color test based on succinic dehydrogenase activity was developed[27] that was neither consistent nor conclusive. An accumulation of amino acids, particularly arginine, was observed in paper chromatograms,[28] but its elution and estimation of intensity were not successful enough to employ the technique as a tool for diagnosis. Infrared aerial photography was attempted to detect the disease,[29,30] but has not proved successful. Adequate ground level data have not been gathered in these studies. A rapid biochemical test was devised by Dwivedi et al.[31] on the basis of colorimetric estimation of the constituents extractable in ethylene diamine tetra acetic acid (EDTA), but the results were inconsistent.[32]

However, a serodiagnostic test developed by Solomon et al.[33] is a successful diagnostic aid to detect root (wilt) disease irrespective of the age of the palms and of the type of soil in which they grow. The method was further refined with a saving in time to the extent of 50%,[34] and it was also possible to determine the severity of the disease based on the intensity of the reaction. Likewise, a diagnostic test on the basis of the changes in stomatal regulation has been successfully formulated.[35]

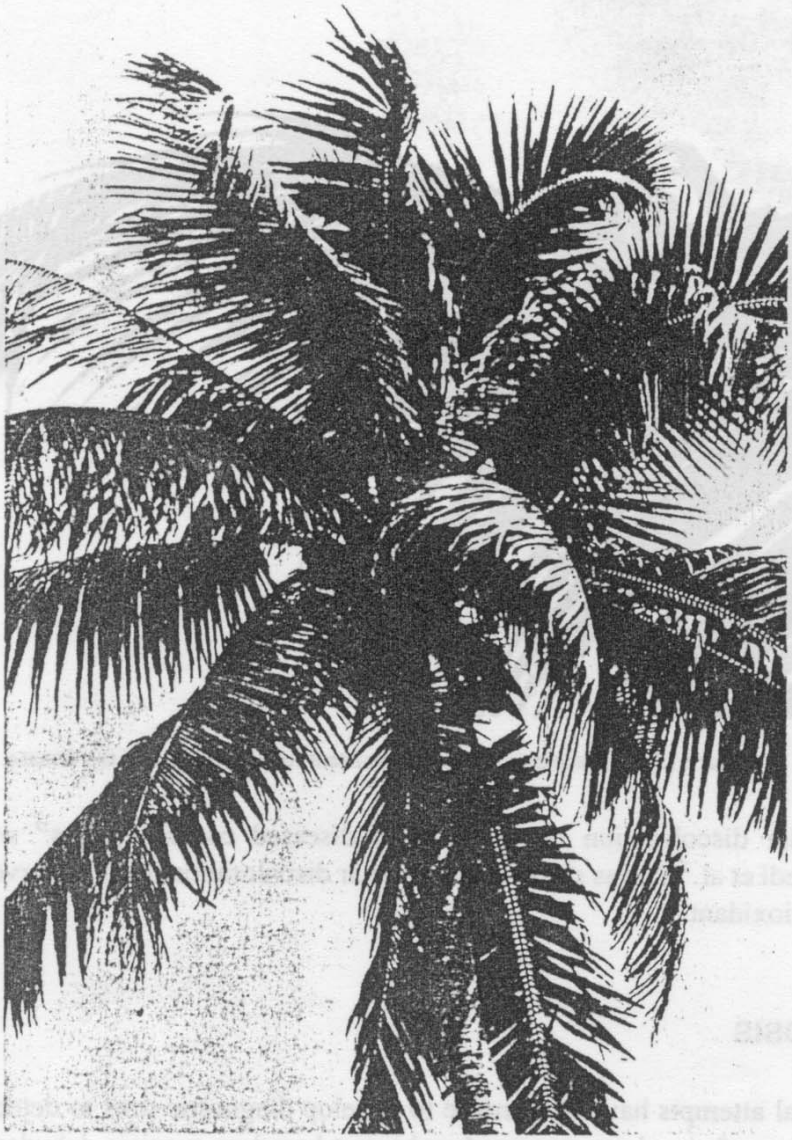

Figure 13-4 A palm in the advanced stage of coconut root (wilt) disease.

A comparative study of the serodiagnostic test and the physiological test was made by Rajagopal et al.[32] It is clear that the root (wilt) disease is characterized by a serologically positive reaction and a relatively low stomatal resistance. Besides, these tests could detect incipient infection of coconut palms by the root (wilt) pathogen 6 to 20 months before the manifestation of the visual symptoms.[32]

13-5 SOILS AND NUTRITION

Soil sickness characterized by low pH and microbial activity, nutrient imbalance together with mineral deficiencies, and inadequate drainage and poor aeration has been reported as a factor responsible for the incidence of root (wilt) disease.[5–7,36–38] Likewise, it was re-

ported that the symptoms of the disease were more pronounced in areas with poor soil aeration and in soils with poor moisture-retentive capacity, high water table, shallow depth, and poor drainage.[13,20,39] Earlier studies on the major soil groups[37,38,40–42] showed that soils in diseased areas had low available potash, exchangeable calcium and magnesium, iron, and total exchangeable bases. It was also indicated that waterlogging could be the prime reason, predisposing the palms to root (wilt) disease.

An accumulation of major nutrients and silica in the leaves of diseased palms was recorded by Verghese et al.,[43] and Cecil[44] observed a deficiency of calcium and magnesium. A systematic detailed investigation on the soils and leaves from healthy and disease-affected tracts covering the major coconut-growing soils of Kerala State has ruled out the possibility of the direct involvement of major nutrients in the incidence of the disease.[45] An imbalance of cationic-to-anionic ratios, like K : Mg, P : S, and N : S, was observed in diseased palms.[44,45] It can be concluded on the basis of the mineral nutrition of the palms that the major nutrients are not involved in the incidence of the disease. The disease occurs in all the soil types of Kerala under varying ecological conditions ranging from foothills to coastal plains.

The application of secondary nutrients at different levels and through different sources did not cure the disease or prevent fresh incidence. In a micronutrient fertilizer trial[46] fresh incidence of disease was not prevented, but sustained economic yield was obtained. No relationship has been observed by Khan et al.[47] between the micronutrient profiles of diseased palms and the intensity of the disease compared with healthy palms.

Scanning electron X-ray microprobe analysis indicated high deposition of Al, Mn, Cu, and Co in the roots of diseased palms and Cr, Ti, Pb, and Ga in the cabbage tissues of diseased palms compared to healthy ones.[48,49] The investigations of Wahid et al.[50] on soils, roots, and leaf samples of diseased and healthy palms employing the energy dispersive X-ray fluorescence technique indicated the presence of nickel and strontium in higher concentrations in the roots of diseased palms.

Before concluding, it should be pointed out that the observations of Cecil[51] and Cecil et al.,[52] over a period of 12 years on the beneficial impact of the correction of a magnesium deficiency, are significant. It reduces the prebearing age of the palms by up to 9 months and increases nut yield, and the response to magnesium was more pronounced in diseased palms compared with healthy ones.

13-6 PHYSIOLOGY

Investigations on the physiological aspects of the disease have shown an increased rate of respiration.[53] The rate of respiration of the leaves from the apparently healthy palms in the diseased tract and the healthy palms from the disease-free tracts differed substantially, with a lower rate in the latter category. The rate of CO_2 fixation and chlorophyll content were significantly greater in the apparently healthy palms compared with the diseased palms.[54] There was a deranged translocation and distribution of sugars[55] and an altered nitrogen metabolism[56] associated with the disease. The concentration of total, reducing, and nonreducing sugars was higher in the leaves of diseased palms, but lower in the roots of such palms. A considerable increase in the nonprotein nitrogen content coupled with a sharp decrease in the water-soluble nitrogen protein and nitrogen fractions was observed

in the diseased tissues.[56] The accumulation of certain free amino acids was noticed by Pillai and Shanta[28] in the leaves of wilt-affected palms. The concentration of amino acids in the leaves increased with the advancement of the disease, with the accumulation of arginine in the diseased palm tissues.

A significant decrease in the total phenol content in the roots of coconut palms with an increase in the intensity of the disease was observed by Joseph and Jayasankar.[57] Likewise the levels of polyphenol oxidase and peroxidase were also found to increase with intensity. The correlation between these phenol-oxidizing enzymes and the coconut root (wilt) index[17] formulated on the basis of foliar symptoms was positive.[58] Levels of phenyl analine ammonia lyase were higher in the root (wilt) -affected palms, compared with healthy palms.[59] These observations suggest an accelerated phenol metabolism characteristic of root (wilt) disease.

The permeability of the root tissues of diseased palms was observed to have been altered, and an imbalance in the water mechanism subsequent to disease development was indicated.[60–62] The root sap collected from the diseased palms differed in quality. The sap from apparently healthy palms was acidic, odorless, and clear with high K_2O and MgO contents, whereas the root sap of diseased palms was neutral to alkaline and foul smelling with low K_2O and MgO.[63] Tomato seedlings grown in the root sap of diseased palms developed epinasty and bending of leaves, but such symptoms were not observed with sap collected from healthy palms.[60]

Differences in the levels of protein values, carbonic anhydrase, cellulase, and pectinolyase were observed subsequent to the incidence of the disease. A comparison of the protein values in the leaves of healthy and diseased palms was suggestive of decelerated protein synthesis and accelerated protein breakdown.[64] The alkali-extractable, water-extractable, and ethanol-extractable protein values were higher in the diseased palms compared with the healthy palms.

A reduction in the availability of biologically active zinc has been indicated on the basis of the comparatively low levels of carbonic anhydrase activity observed in the leaves of diseased palms.[31] Significantly higher activity of cellulase was noticed in the decayed roots of diseased palms, while no such activity was observed in the nondecayed roots of both healthy and diseased palms. The pectin-lyase activity in the root tissues was six times higher in the diseased palms compared with the healthy ones.[65]

The absorption of water by roots[63] and the uptake and transport in the trunk[61] were considerably less in the wilt-affected palms compared with the healthy ones. Soil moisture depletion in irrigated plots was observed in the diseased palms.[35] An abnormal opening of the stomata in the diseased palms, with an altered regulation leading to excessive loss of water, was also noticed in the diseased palms.[35] The rate of transpiration increased with an increase in the intensity of the disease.[66] Likewise the root (wilt) -affected palms had a low leaf-water potential and leaf-turgor potential compared with the healthy palms.[67]

13-7 ETIOLOGY

The spreading nature of root (wilt) disease suggests the involvement of a pathogen, and the results of the physiological investigations lend support to a pathological condition.

This hypothesis is supported by the observations of symptoms in the field and controlled conditions in transmission studies.[68-72] Concerted efforts have been made to elucidate the causal agent of the disease from different angles.

13-7-1 Fungi

A "fungoid" concept was proposed in 1906 by Bourdillon,[73] and a *Botryodiplodia* sp. was isolated by Butler,[1] perhaps from rotted roots, a factor responsible for its absence in most of the subsequent investigations. Menon and Nair[13] have suggested a possible association of not only *Botryodiplodia theobromae* but also *Rhizoctonia solani* Kunn. and *R. bataticola* (Taub.) Butler with the rotted root system of diseased palms. These isolates could not produce the foliar symptoms in young experimental palms in spite of their ability to infect root tips,[13] a situation contrary to the suggestion of Butler[1] that the root rot alone can pave the way for the disease. Similarly, Radha and Menon[74] observed that the survival of *R. solani* in soil with adequate moisture was poor in young experimental palms. Further attempts yielded *Fusarium equiseti* (Corda) Sacc. and *Cylindrocarpon effusum* Bugn. from apparently healthy roots of diseased palms.[75] Sosamma and Koshy[76] isolated *Cylindrocarpon lucidum* Booth from nematode-induced root lesions. Pathogenicity trials conducted in field tanks using these fungal isolates, *Fusarium equiseti* and *Cylindrocarpon*, failed to reproduce the symptoms of the root (wilt) disease when inoculated singly or in combination with the other associated biotic agents[34] (Figure 13-5). Any primary role of fungi in inciting the disease has been ruled out.

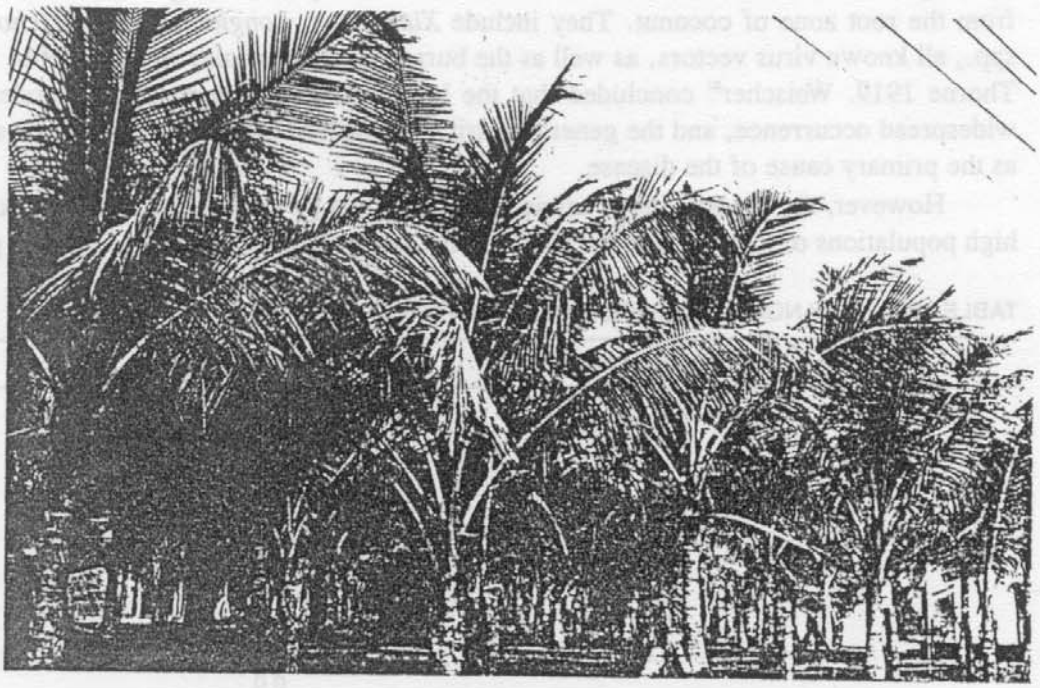

Figure 13-5 Pathogenicity trial in microplots initiated in 1982.

13-7-2 Bacteria

The observation of Menon and Nair[13] of the presence of two species of bacteria did not gain momentum until the discovery of vascular streaming movement characteristic of bacteria in the roots of disease-affected palms. The isolates were identified as species of *Pseudomonas*. But persistent efforts since then have revealed the association of only *Enterobacter cloacae* (Jordan) Hornachae and Edwards.[77] The bacterium does not belong to conventional plant pathogenic genera; however, species of *Enterobacter* have been implicated in plant diseases.[78,79] The coconut *E. cloacae* isolate elaborated wilt-inducing toxic principles in growing culture filtrates.[77] The toxin has been purified and characterized, and the fraction was antigenic, showing a serological reaction to extracts from diseased materials.[80] The isolates were sensitive to streptomycin and oxytetracycline. A field experiment initiated in 1977 employing a commercial oxytetracycline (OTC) tree formulation indicated an ameliorating effect on the disease condition of the experimental palms[80] (Table 13-2). The palms were in the age group of 10 to 20 years.

The bacterium was inoculated alone and in combination with other associated biotic agents in 1-year-old seedlings in microplots. Symptoms characteristic of the disease were not produced in the experimental seedlings, and the involvement of the bacterium in coconut root (wilt) disease stands ruled out.

13-7-3 Nematodes

Investigations of the involvement of nematodes[81–84] have yielded 35 genera of nematodes from the root zone of coconut. They include *Xiphinema*, *Longidorus*, and *Trichodorus* spp., all known virus vectors, as well as the burrowing *Radopholus similis* (Cobb. 1892) Thorne 1919. Weischer[81] concluded that the low population density of nematodes, the widespread occurrence, and the general distribution pattern could exclude the nematodes as the primary cause of the disease.

However, the investigations carried out since then by Koshy, et al.[85,86] showed very high populations of *R. similis* in the roots of root (wilt) -affected as well as healthy palms

TABLE 13-2: CHANGE IN DISEASE INDEX (1977–1981)[a]

OTC-Treated Palms[b]	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

[a]The intensity of the disease was indexed by the method of George and Radha.
[b]One experimental palm was destroyed by pest infestation.
t value of 3.81, significant at 1% level OTC.

in the diseased tracts. Further, *R. similis* infestation produces small, elongate, orange-colored lesions on tender creamy white roots, which enlarge and coalesce to cause extensive rotting of roots. Besides, the pathogenic potential of the nematode has been clearly established.[87-89] However, in a large-scale pathogenicity trial initiated in 1982 in microplots, the production of typical root (wilt) disease symptoms, even after 6 years, on young palms inoculated with 1 million nematodes was absent. As such, *R. similis* is not involved as an incitant in the etiology of root (wilt) disease.

13-7-4 Virus

Virological investigations of root (wilt) disease have gained accent since 1952 with the successful transmission of the disease in the field by mechanical transmission and by the banana lacewing bug, *Stephanitis typica* (Distant).[70] Flaccidity, paling, and slight stunting of younger leaves were noticed in five out of six seedlings inoculated with leaf extracts of diseased palms and in one of the six seedlings inoculated with *S. typica*.[3,71] The presence of a sap transmissible agent in the diseased tissue has been demonstrated by the production of transmissible symptoms on cowpea.[90] In view of the peculiar nature of the symptoms on cowpea, Holmes[91] and Holmes et al.[92] suggested that the sap transmissible agent might be a viruslike spirochete or a protozoan. But, due to reasons unknown, the symptom production on cowpea was erratic in subsequent studies.[93]

Summanwar et al.[94,95] attributed disease to the presence of tobacco mosaic virus (TMV), and Maramorosch and Kondo[96] reported the presence of two types of submicroscopic particles identified as phytoferritin and icosahedral virus in diseased palm tissues. The association of the TMV strain has not been confirmed in separate investigations employing serology and electronmicroscopy.[97-99] The icosahedral particles reported earlier[96] have since been identified as plasmodesmata, sectioned in tangential plane.[100] The inability to detect virus particles in diseased palm tissues with electron microscopy and the inconsistent response of cowpea rule out a viral etiology of root (wilt) disease.

13-7-5 Mycoplasmslike Organisms

Against the background of the investigations carried out from different angles on the etiology of the disease, the observation of Solomon et al.[101]—of mycoplasmalike organisms (MLOs) under electron microscope in ultrathin sections of developing leaves, unopened inflorescences, root tips, and terminal bud tissues (in the sieve tubes of phloem) in coconut palms affected by the (wilt) disease, and their conspicuous absence in samples from healthy palms—assumes significance, as no viruslike particles or other submicroscopic organisms other than MLOs have been made out in the ultrathin preparations. The constant association of MLOs has subsequently been established in a large number of diseased palm tissues (Figure 13-6). Histochemical staining reactions using Diene's staining[102] and fluorescence staining of DAPI[103] suggest the accumulation of DNA in extra nuclear sites, indicating the presence of MLOs.[104]

While intensifying the search for insect vectors in this context, besides the lace bug *S. typica*, the longtime suspected vector in early studies,[70,71] a leafhopper, *Sophonia greeni*

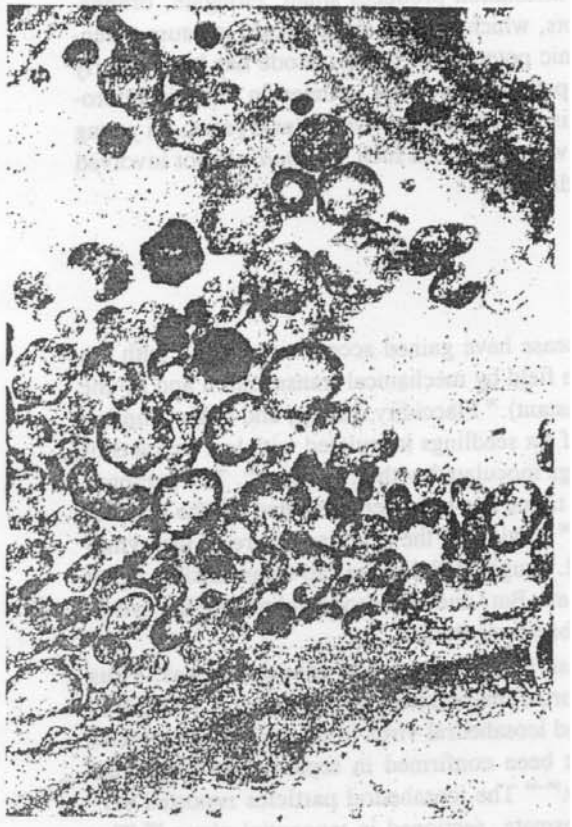

Figure 13-6 MLOs in sieve tubes of diseased palm tissues.

(Distant), and a plant hopper, *Proutista moesta* (Westwood), were recorded.[105,106] Electron microscopic observations of the lace bugs that had a sufficient acquisition-plus-incubation period on diseased coconut palms (18 to 23 days) revealed the presence of structures resembling MLOs in the salivary glands and brain tissues (Figure 13-7), which were not observed in the bugs collected from disease-free areas and also not in those with an acquisition-plus-incubation period less than 18 days.[107] MLOs are normally transmitted by leafhoppers or plant hoppers.

The lace bug *S. typica* is not a conventional phloem feeder, but the insect feeds through the stomata and its stylets reach the phloem of palm leaves.[108] Referring to the hoppers, electron-microscopic studies located the presence of MLOs in the salivary glands of *P. moesta* with an acquisition-plus-incubation period above 40 days.[109] As was already indicated, the transmission of the disease from coconut to coconut through lace bugs has been reported earlier during investigations on possible viral etiology. Renewed attempts were made in this direction employing refined techniques under controlled conditions (Figure 13-8); MLOs were observed in all the four experimental seedlings, and in two seedlings flaccidity of leaflets was observed by the 17th month,[110] unlike in the control seedlings. The vector role of the plant hopper is yet to be assessed.

The parasitic angiosperm *Cassytha filiformis* Linn., when established on a 4-year-old diseased palm bridged with the periwinkle *Catharanthus roseus* (Linn.) G. Don (a

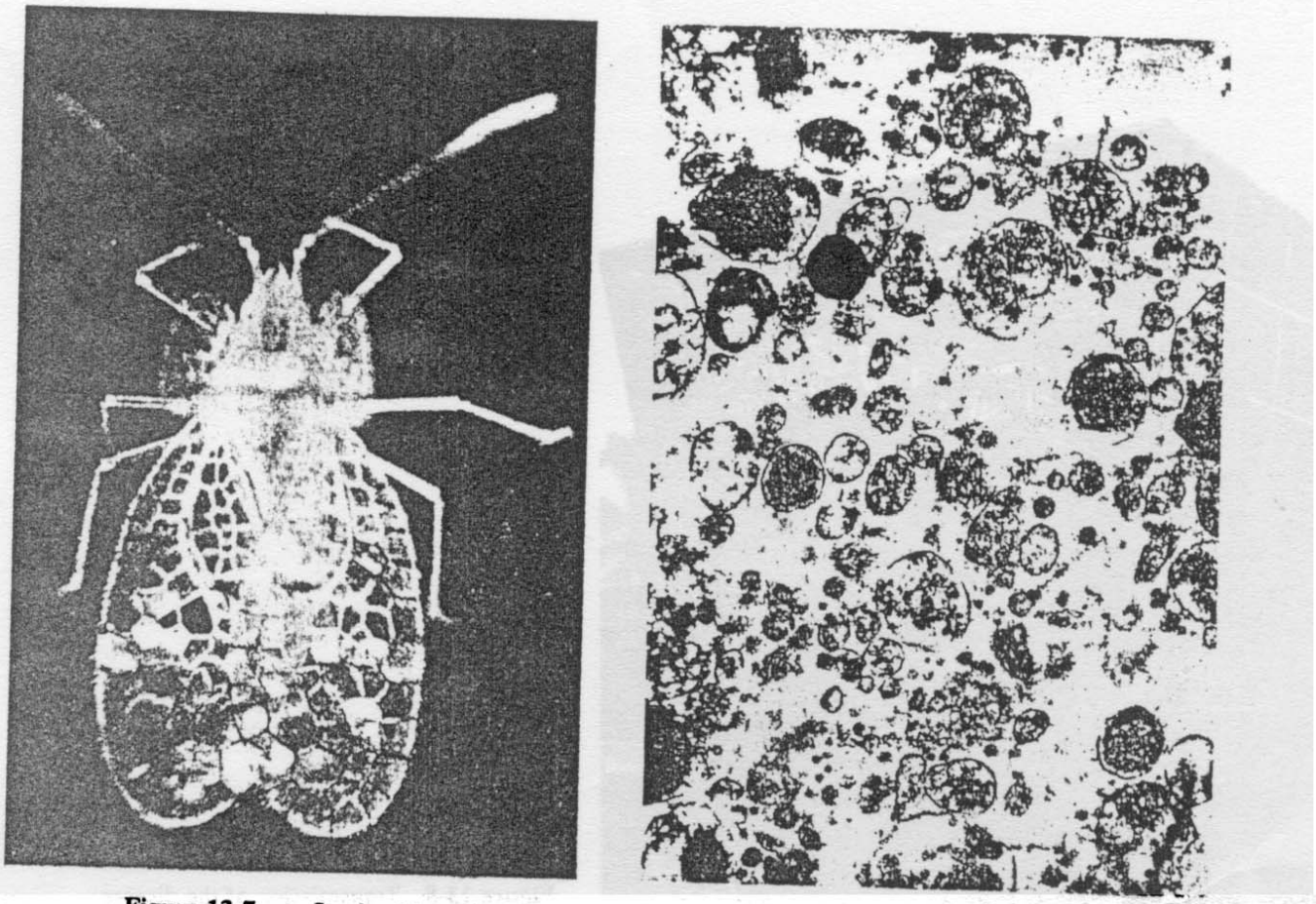

Figure 13-7 *a*, *Stephanitis typica*, the lace wing bug; *b*, structures resembling MLOs in salivary glands of *Stephanitis typica*.

known mycoplasma indicator host) and maintained under insect proof conditions, developed the characteristic interveinal yellowing suggestive of infection by MLOs (Figure 13-9). Mycoplasmalike organisms were observed in the vegetative vector and test plants. Serial transmission was successful when primary infected periwinkle plants were bridged to a secondary set of healthy plants.[111]

The role of MLOs in the etiology of coconut root (wilt) disease gains additional support from the response of the disease to OTC. It was observed, even prior to the implication of MLOs with the disease as dealt with earlier, that the application of OTC prevented the deterioration of the diseased condition of experimental palms, while the untreated control palms showed deterioration.[80] The field trial started in 1984 with OTC, neomycin, penicillin, and distilled water control indicated after a span of 3 years that the fresh set of leaves had remission of symptoms in 53% of the palms treated with high concentrations of OTC (3 g and 6 g a.i.). On the other hand, palms treated with distilled water and penicillin deteriorated significantly compared with the control palms.[112]

To sum up, the constant association of MLOs in diseased palm tissues combined with their total absence in healthy palms, the establishment of the vector role of *S. typica* through transmission trials, and the positive response of diseased palms to OTC suggest a mycoplasmal etiology of coconut root (wilt) disease.

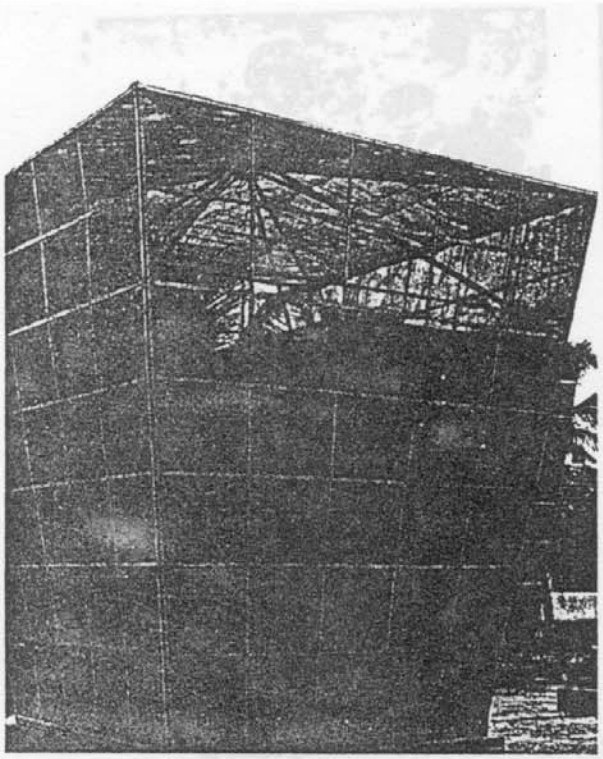

Figure 13-8 Transmission of the disease by *Stephanitis typica* in insect-proof cages.

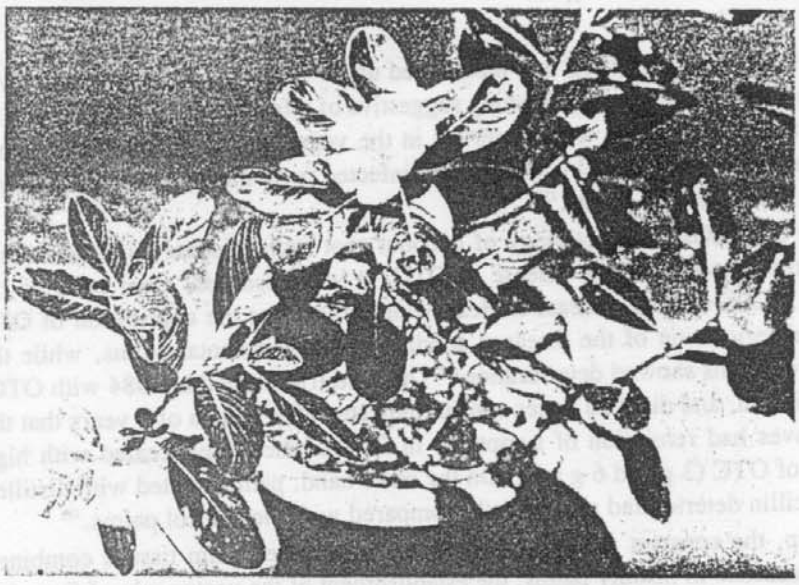

Figure 13-9 Transmission through vegetative vector: Symptoms on periwinkle.

13-8 ATTEMPTS AT CONTROL

A number of beneficial effects have emerged in the attempts made to sustain productivity in root (wilt) -affected coconut palms. A canopy occupation for the absorption of solar energy by a middle-aged palm will be a small area compared with the available solar energy penetrating to the land area.[113] The active root zone of the coconut palm extends laterally only to an area of 2 m around the palm. This indicates that only about 25% of the total soil mass is being utilized effectively by coconut roots.[114] The situation is ideal for growing compatible crops in the interspaces, which will not only sustain the productivity of the palms but also increase income for unit area.

The cultivation of fodder crops in the interspaces of a diseased coconut plantation and the maintenance of milch cows to enable the recycling of cattle manure and other wastes in the plantation has resulted in an increase in nuts of 26.1% over a period of 5 years, irrespective of the disease condition of the palms.[115] A follow-up experiment during the next five years confirmed the profitability of such a farming system (Figure 13-10). In addition, there was a significant increase in soil fertility, as evidenced by higher values of soil organic carbon, exchangeable calcium, magnesium, and potash, with enhanced soil microbial activity.[116,117]

Mixed cropping with cacao increased the yield of coconut to the extent of 29% to 35% without any lessening of the disease intensity of the palms, under rain-fed conditions.[118] In a similar experiment conducted in a farmer's field for a period of 5 years under irrigated conditions with recommended doses of fertilizers to both the crops, the yield of coconuts increased from an average of 17.6 to 46.0 nuts/palm/year. Increased production of palms, irrespective of the disease condition, under crop mixing with cacao in the double-hedge system was also reported by Nair et al.[113] However, after a lapse of 10 years better productivity of coconuts was observed in the single-hedge system of cacao.[112]

Figure 13-10 Mixed farming with fodder crops and milch cows in coconut plantations.

Cultivation of cassava, elephant's-foot yam, and yam for a period of 3 years in the interspaces of palms in disease-affected gardens increased the nut yield by 4.96%, 15.57%, and 8.07%, respectively[119] (Figure 13-11). In plots intercropped with elephant's-foot yam and yam, a slight improvement in the foliar condition of the diseased palms was also noticed. Detailed studies have been carried out in farmers' fields on the effect of intercropping tubers and rhizomes (viz., cassava, greater yam, elephant's-foot yam, colocasia, ginger, and turmeric) on soil fertility, disease intensity, and the productivity of palms.[120] Studies revealed enhanced soil fertility without any adverse effects either on the disease index or on the yield of palms.

The observations of Bavappa et al.[121] in a coconut-based, high-density multispecies cropping system indicated considerable increases in the total and beneficial microbial population and soil microbial biomass in the coconut basins as well as in the interspaces.

Referring to aspects other than inter-, mixed-, or multispecies cropping systems, nine species of leguminous green manure crops were raised in coconut basins in a farmer's field in a heavily diseased tract in sandy loam soil.[122] Among the lot, *Peuraria phaseoloides*, *Mimosa invisa*, and *Calopogonium mucunoides* yielded 18.43, 17.00, and 14.71 kg green matter per basin, respectively, in a period of 4 months (Table 13-3). There was a significant increase in the microbial populations and soil enzymatic activities. Green manuring augments mycorrhizal symbiosis in coconut palms.[123]

The water requirement of the root (wilt) -affected coconut palms was suggestive of the necessity of irrigation during summer months for the maintenance of optimum growth.[66,67] Summer irrigation increased the water uptake with a reduction in the rate of transpiration under diseased conditions.[66] It was also observed that summer irrigation decreased the intensity of the disease and increased the productivity of the palms.[67]

Figure 13-11 Intercropping with tubers.

TABLE 13-3: GROWTH AND NODULATION OF GREEN MANURE CROPS IN COCONUT BASINS

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	17.0	153.19	125	1.860
Pueraria phaseoloides	19.43	121.29	132	1.128
Leucaena leucocephala	2.95	16.55	0	0.0
Sesbania aegiptica	1.30	6.98	56	0.535
Macroptilium atropurpureum	9.10	66.64	60	0.108
Glycine wightii	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

The continuous application of NPK fertilizers enhanced the yield of diseased palms.[124] The beneficial effect of magnesium sulfate in reducing the prebearing age of palms and increasing productivity in root (wilt) -affected tracts has already been indicated.[51] The hybrid Chowghat Dwarf Orange (CDO) x West Coast Tall (WCT) under good management in heavily diseased sandy soils yielded a higher number of nuts with a lower incidence of disease compared with WCT palms of identical age in the early years of production. The application of magnesium increased the cumulative yield of the CDO x WCT hybrid from 463.5 to 658.8 nuts/palm up to 12 years of planting.[118]

According to the observations of Nambiar,[125] the yield of palms was constant over a period of 6 years in a heavily infected area under rain-fed management practices. During this time the intensity of the disease increased nearly 50%.

A program for assessing the yield potential and resistance and tolerance to root (wilt) disease has been in progress since 1961. Since then 63 cultivars and 32 hybrids have been screened and all of them found to be susceptible.[126] High-yielding elite palms were identified by Iyer et al.[127] in the contiguous disease-affected tracts. Open-pollinated progenies of these elite palms, however, have contracted the disease, ranging from 40% to 100%.[109]

Leaf rot disease of coconut palms is normally found superimposed on root (wilt) -affected palms, causing considerable loss in yield[12,21,16] (Figure 13-12). Regular spraying with fungicides significantly reduced the incidence of leaf rot disease. In sequential spraying with Bordeaux mixture (1.0%), Dithane M-45 (0.3%), and Fytolan (0.5%) carried out on 1,610 leaf rot–affected palms, the incidence of disease could be brought down to 200 palms within a period of 3 years.[128]

The beneficial effect of integrated management practices was brought out by Muralidharan et al.[129] on the basis of systematic and replicated field experiments. The observation that young coconut palms that take up root (wilt) disease before the onset of flowering may not flower at all or will have delayed flowering as well as low productivity merits consideration. In the replicated trials[129] all diseased young palms and severely affected uneconomic adult palms were eradicated and balanced doses of fertilizers were applied with basin cropping with *Pueraria*. Farmyard manure at the rate of 50 kg/palm/year was applied, leaf rot disease was checked by sequential spraying with Bordeaux mixture,

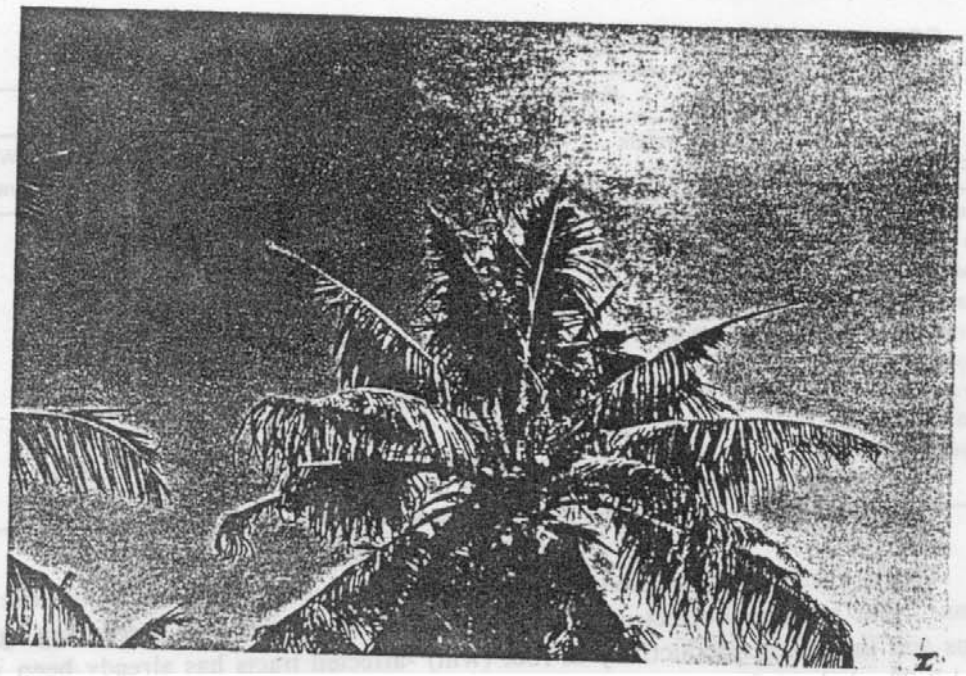

Figure 13-12 Leaf rot disease of coconut superimposed on root (wilt) disease.

Dithane M-45, and Fytolan, and prophylactic measures were undertaken against the major pests of coconut by leaf axil filling with BHC-sand mixture. Yams in rotation were cultivated in the interspaces, and other perennial crop species were restructured. The effect of such an integrated approach as observed over a period of 5 years was promising, with an average increase of 23 nuts/palm/year (Table 13-4).

13-9 OPERATIONAL STRATEGIES

Complex maladies, particularly of perennial crop species, have been and are still controlled by management devices. Definite strategies have crystalized on the basis of scien-

TABLE 13-4: EFFECT OF MANAGEMENT PRACTICES ON THE YIELD OF PALMS IN ROOT (WILT) -AFFECTED GARDENS

Disease Index	Preexperiment		Postexperiment		Increase over Control	
	Contr.	Treat.	Contr.	Treat.	Number	Percent
AH (0/10)	40.8	43.5	51.3	80.9	29.6	57.7
DE (11–25)	29.9	26.4	35.5	64.3	28.8	81.1
DM (26–50)	18.7	17.7	21.2	29.6	8.4	37.6
DA (>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

AH = apparently health
DE = disease early
DM = disease middle
DA = disease advanced

tific investigations by Muralidharan et al.[129] to manage coconut root (wilt) disease in the heavily affected contiguous area and in the isolated pockets of sparse infection in the mildly affected tracts. In the heavily diseased tract all diseased juvenile palms, irrespective of the intensity of the disease, and all adult palms in the advanced stage of the disease need eradication. By a process of systematic replanting and underplanting, the number of palms in a unit area is restricted to the optimum level of 175 palms/ha. Fertilization (consisting of 0.5 kg N, 0.3 kg P_2O_5, 1.0 kg K_2O, and 0.5 kg MgO in two split doses) with farmyard manure and compost at the rate of 50 kg/palm/year and the raising of green manure crops like *Pueraria* in the basins and incorporation are essential. Intercropping with the least competing crops (yams, ginger, colocasia) in rotation or mixed farming with fodder crops and milch cows in diseased coconut gardens with the recycling of organic matter will be beneficial. The control of leaf rot disease and pests, the prevention of waterlogging and the lack of aeration in the soil, and the regulation of excessive shading are the other required factors in the package.

The earliest attempt to prevent the recurrence of the disease in mildly affected areas by the removal of the foci of infection was in 1971.[130] The uprooting and burning of three diseased palms prevented recurrence. Efforts since then made to contain root (wilt) disease by the repeated removal of disease-affected palms in the border areas north of the Karuvannur River in the Trichur district (Figure 13-13) have clearly brought out the possibility of containing the disease in the contiguous core and preventing the spread to newer areas. A total of 730 palms in 341 gardens in 10 villages[130] were eradicated and the boles and roots burned in situ during 1979 to 1982. The recurrence of disease has been limited to 21 palms spread over 15 gardens in 3 villages (Table 13-5). This essentially forms the basis of the strategy recommended for the sparsely affected isolated pockets away from the contiguous core. All diseased palms, irrespective of the intensity of the disease and yield, need to be eradicated followed by insecticidal spraying to prevent the spread of insect vectors carrying the pathogen. Irrigation is an assured component to increase productivity in both the tracts.

13-10 FUTURE THRUST

The suppression of insect vector(s) can perhaps result in economic control of the disease. The possible application of the ELISA and western blot techniques for rapid diagnosis need attention in the light of a positive serodiagnostic test. The exclusive selection and propagation of MLO-specific clones by the application of the monoclonal-antibody technique are worth elucidation.

Environmental studies to specify the factors contributing to the geographical delineation of root (wilt) disease and to render the crop less favorable for the pathogen or vector(s) by appropriate management are worthwhile. There is a need to develop a suitable strategy for ill-drained low lands and coastal soils, and the existing management devices can be improved by reducing the cost to the greatest possible extent.

Host-plant resistance will be the best solution to the malady. High-yielding mature palms are often located in heavily diseased coconut gardens that are tolerant to wilt disease. Improved progenies for evaluation, selection, and hybridization can be generated. DNA hybridization techniques to locate MLOs in plants and insects can be ideal for

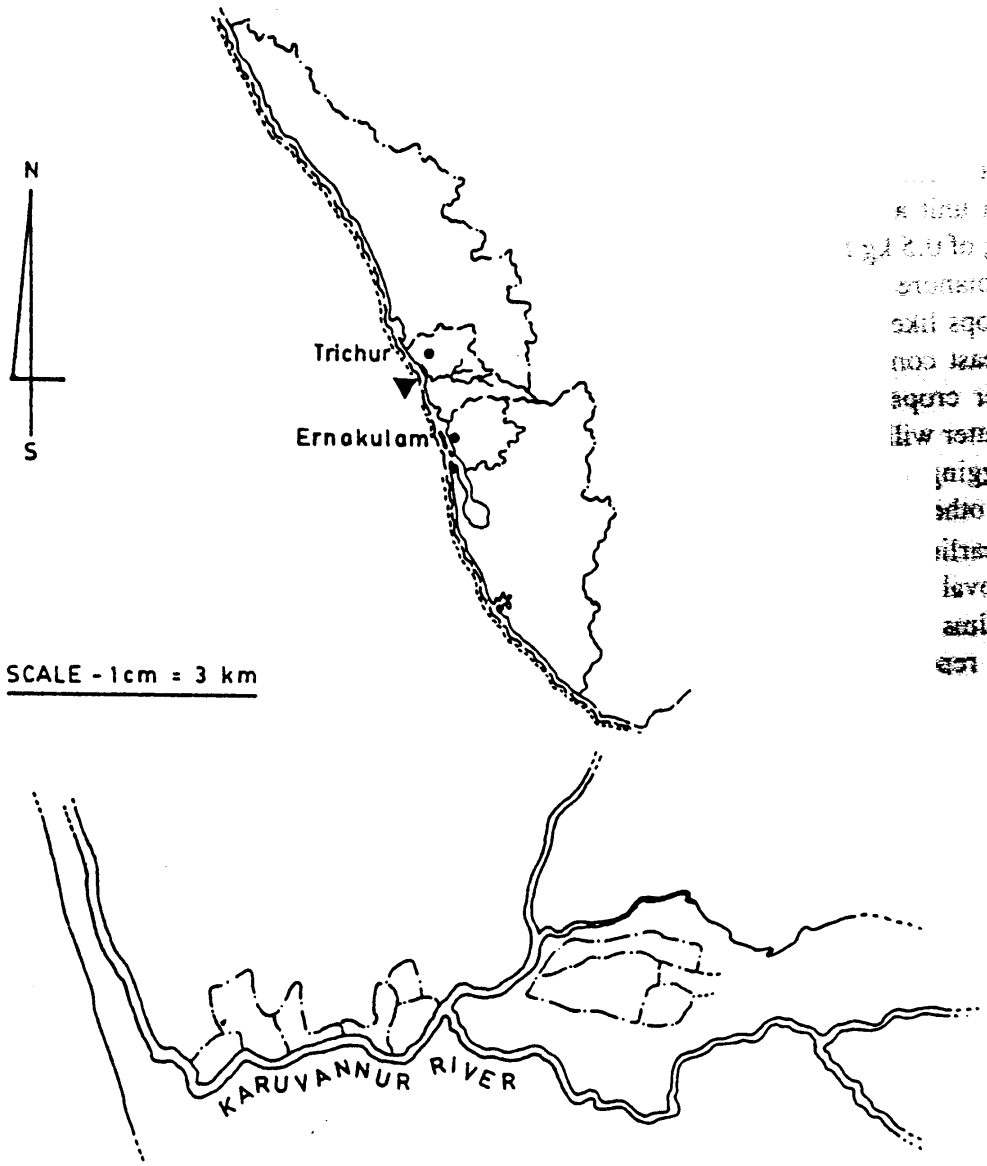

Figure 13-13 Area of operation in the mildly affected tracts north of the Trichur district.

TABLE 13-5: EFFECT OF ERADICATION ON DISEASE RECURRENCE

Year	No. of Villages	No. of Gardens	No. of Palms
1979–1982	10	341	730
1983	8	18	21
1984	1	15	8
1985	6	17	21
1986	4	19	22
1987	3	12	25
1988	7	21	39
1989–1990	3	15	21

screening varieties for disease resistance and tolerance. Screening against the possible insect vector(s) should be initiated.

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