

**Phytoplasma diseases of coconut in India -
root (wilt) and tatipaka diseases**

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Abstract

Root (wilt) and Tatipaka diseases in India are caused by phytoplasma. Lethal yellowing disease in Caribbean region, Lethal yellowing like disease in west and east Africa and Coconut stem necrosis in Sumatra and Peninsular Malaysia are phytoplasma induced diseases of coconut in other continents. Root wilt disease in India was first reported from Kerala in 1874. Tatipaka disease was first noticed in Andhra Pradesh after the 1949 cyclone. The progress made on various aspects including history, nature of spread, description of external & internal symptoms, etiology, abiotic factors, biotic agents implicated in transmission of disease, culturing, chemotherapy, screening of coconut germplasm, breeding for disease resistance/tolerance and disease management is detailed.

Key words: Root (wilt) disease, Tatipaka disease, phytoplasma, India

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Introduction

Coconut palm (*Cocos nucifera* L.) is grown in over 93 countries in the humid tropics. It is affected by a number of maladies-some of them are lethal and others of debilitating nature. In the past thirty years, the causes of several coconut diseases of previously uncertain etiology have been determined. Phytoplasma, virus, viroids and protozoan flagellates have been implicated as the cause of some of these diseases.

Phytoplasmas earlier referred as mycoplasma-like organisms (MLOs) are plant pathogenic mycoplasmas that are non-helical, non-culturable and transmitted by arthropod insect vectors. Lethal yellowing disease in Caribbean region, Lethal yellowing like disease in west and east Africa and Coconut stem necrosis in Sumatra and Peninsular Malaysia are phytoplasma induced diseases of coconut in other continents. Root (wilt) and Tatipaka diseases in India are confirmed to be caused by phytoplasma.

Root (wilt) disease

History of the disease

Root (wilt) disease of coconut was first noticed in the erstwhile princely State of Travancore around 1874 and became very much evident after the great floods of 1882. The disease was initially reported from three independent locations - Erattupetta area of Meenachil taluk, Kaviyoor and Kalloopara of Thiruvalla taluk and Kayangulam of Karthikappally taluk each at a distance of about 50 km (Butler, 1908; Pillai, 1911; Varghese, 1934). Since then the disease has spread from the original foci of infection and according to a survey made during 1984/85 it occurs in a contiguous manner in an area of 4,10,000 ha in eight out of the fourteen districts of the Kerala State. It is also noticed in isolated pockets in the remaining six northern districts of the state and in the bordering districts of Tamil Nadu (Fig.1). The disease intensity in the contiguous disease tract ranged between 1.5% in Thiruvananthapuram district to 75.6% in



Fig.1. Distribution of root (wilt) disease

Kottayam district (CPCRI, 1985). Extent of disease incidence in eight southern districts was updated in 1996 by a sample survey (Department of Agriculture, Government of Kerala, 1997) and it ranged from 2.1% in Thiruvananthapuram district to 48% in Alapuzha district (Table 1). There is an overall reduction in disease incidence since 1984 from 32.37 to 24.05. This is mainly attributed to the removal of about 59 lakh diseased palms, replanting with quality seedlings, adoption of disease management practices and crop conversion from coconut to rubber. The annual crop loss due to the disease was estimated to be about 968 million nuts (CPCRI, 1985). The total estimated monetary loss in terms of loss in husk, copra yield and leaf number and quality of leaves on the basis of 1984 price index for coconut was of the order of about Rs.3000 million.

Table 1. Survey of coconut wilt in Kerala

Districts	Disease incidence (%)	
	1984-85	1996-97
Thirissur	2.60	6.19
Ernakulam	34.52	33.01
Idukki	34.18	33.56
Kottayam	75.63	36.56
Alapphuza	70.59	48.03
Pathanamthitta	38.22	37.80
Kollam	28.55	25.97
Thiruvananthapuram	1.52	2.09
Overall	32.27	24.05

Nature and spread

The disease is non-lethal but debilitating and palms of all age groups from seedlings to adult palms are affected. Disease contraction in the pre-bearing age delayed flowering and affected the vitality of the palm. The leaf rot disease occurs superimposed on about 65% of the root (wilt) diseased palms (Srinivasan, 1991) and contributed to the rapid decline and reduction in yield of the affected palms. *Colletotrichum gloeosporioides* and *Exserohilum rostratum* are the main pathogens associated with the disease (Srinivasan and Gunasekharan, 1994).

The root (wilt) disease occurs in all major soil types but the spread is faster in sandy, sandy loam, alluvial and in heavy textured soil than in laterite. Relatively higher incidence is recorded in waterlogged low lying areas adjacent to rivers and canals. The pattern of spread is erratic, occurs in jumps or leaps, characteristic of insect transmission. The rate of spread is reported to be about 1-4 km over a period of three years (Pillai, 1981).

Symptoms

External symptoms

The most obvious and diagnostic symptom of the disease is the abnormal inward bending of the leaflets termed 'ribbing' or 'flaccidity'. Foliar

yellowing of the outer whorl of leaves and marginal necrosis are the other associated symptoms (Radha and Lal, 1972) (Fig.2).

**Fig. 2 Palm with root (wilt) symptom**

In seedlings and juvenile palms yellowing of foliage is virtually absent and 'flaccidity' is the only symptom evident. Instances of diseased palms having their outer leaves quite healthy but with yellowing of leaves in some of the inner whorls is also noticed (Menon, 1938). The root system in the early stage of disease is apparently normal and the percentages of rotten roots are few. However, with the progress of the disease, extensive rotting of roots is observed (Menon and Pandalai, 1958). The main roots and rootlets start drying from tip backwards (Menon and Nair, 1951). The capacity of the palms to regenerate fresh roots is drastically reduced (Michael, 1964).

Shedding of immature nuts if the tree is bearing at the time of disease contraction is yet another symptom observed in some cases (Menon, 1938). Drying up of spathes and necrosis of spikelets from tip downward in unopened inflorescence is noticed in certain cases. A higher percentage of pollen produced is either sterile or with low viability (Varkey and Davis, 1960). Meiotic irregularities are also observed (Nambiar and Prasannakumari, 1964).

The vitality of the diseased palms is so adversely affected that they produce small spathes with fewer female flowers. In the advanced stage the crown gets very much reduced in size and ceases to produce flower bunches. Radha *et al.* (1962) reported reduction in yield of nuts upto 80% in palms in the advanced stages of disease.

The nuts from diseased palms have thinner husk and fibers are definitely weaker and less firm (Varghese, 1934). The kernel is thinner and never dries up into hard brittle copra but remains soft and flexible. Although no significant changes in the biochemical constituents of the coconut meal due to the disease were observed but the quality of coconut water was affected (Sosamma and Chandrasekaran, 1993). The tender coconut water of diseased palm is insipid. The loss of copra /oil per nut is 9.0/11.3 % (CPCRI, 1985) and the oil loses its flavour as well (Menon, 1938).

An indexing method for quantifying the disease intensity giving due weight age to the three predominant foliar symptoms has been developed (George and Radha, 1973; Nambiar and Pillai, 1985).

Internal symptoms

Comparative anatomical studies of the tender unopened leaves of healthy and root (wilt) affected palms revealed general stunting of epidermal cells, reduction in thickness of the cuticle on the adaxial side and differential rate of division of the upper epidermis in longitudinal and transverse plains in leaves of diseased palms. Reduction in wall thickness of cells, sclerenchymatous fibers and bundle sheath is also observed. These anatomical changes may be contributing to the downward curling of leaflets (Joseph and Shanta, 1964).

The distribution of stomata per unit area is found to be more in leaflets of diseased palms (Joseph and Shanta, 1964). The stomatal regulation is adversely affected due to the disease irrespective of the age of the palm, time of the day and season. The stomata in diseased palms failed to close in response to soil and atmospheric drought. The excessive transpirational loss in diseased palm due to impaired stomatal mechanism is not replenished adequately as the

root system is damaged (Rajagopal *et al.*, 1986 a, b). The overall disturbance in water relation caused to the two facets of water transport namely absorption and transpiration may also be contributing to the flaccidity symptoms.

Gross structural changes are also very much evident in vascular tissues of diseased palms. Wall thickness of metaxylem elements is reduced resulting in uneven shape of the xylem component. The uptake and translocation of solutes is also impeded with the formation of tyloses in xylem vessels, gummosis and necrotic obliteration of phloem in the roots of diseased palms (Govindankutty, 1981). Leaching of metabolites due to permeability changes also aggravates the disease syndrome (CPCRI, 1977).

Etiology

A number of biotic agents and abiotic factors were implicated as the cause of the disease.

Abiotic factors

Physiological and biochemical changes observed in diseased palms such as derangement in uptake and translocation of solutes, higher respiration and transpiration rate, altered nitrogen metabolism and an accelerated phenol metabolism are indicative of a pathogen mediated aberrant host metabolism than of a physiological disorder (Mathew *et al.*, 1991).

Soil sickness as a contributing factor of the disease was also excluded based on field fertility trials and analysis of tissue and soil (Cecil and Amma, 1991).

Biotic agents

The sporadic occurrence and spreading nature of the disease implied the involvement of a pathogen as the cause of the disease. A number of biological agents such as fungi, bacteria and nematodes were reported to be associated with the disease. However, pathogenicity experiments with these organisms singly and in combination failed to reproduce the symptoms of the disease thereby, indicating their non-involvement in the disease causation (Joseph and Lilly, 1991;

Jayasankar and George, 1991; Sosamma and Koshy, 1991). Nagaraj *et al.* (1954) based on the systemic nature of the disease and resemblance of symptoms to other known plant virus induced diseases (yellows type) suggested it to be a virus disease. Association of a virus with the disease was also indicated through transmission studies (Nagaraj and Menon, 1956; Shanta *et al.*, 1964). Summanwar *et al.* (1969) reported the isolation of a virus from *Chenopodium amaranticolor*. The virus was also identified as a strain of TMV based on its reaction to TMV antiserum (Summanwar *et al.*, 1971). However, subsequent systematic serological and electron microscopic studies have conclusively ruled out the association of any virus with the disease (Shanta *et al.*, 1975). Similarly, Maramorosch and Kondo (1977) reported the presence of icosahedral particles in epidermis and ground parenchyma cells of root (wilt) diseased palms. These particles were later identified as plasmodesmata sectioned in tangential plane (Parthasarathy, 1978). Polyacrylamide gel electrophoretic analysis of isolated nucleic acids from diseased palms also failed to subscribe to a viroid etiology (Randles and Hatta, 1980).

Phytoplasma

Structural alterations observed such as disorganisation and disintegration of vascular tissues, increased chromophily and necrotic obliteration of phloem tissues suggested the involvement of a vascular bound pathogen.

Electron microscopy

Electron microscopic examination of juvenile tissues like sub-meristem, petiole of developing leaves, rachilla of unopened inflorescence and root tips of diseased palms revealed the presence of phytoplasma (Solomon *et al.*, 1983). Phytoplasmas earlier referred as mycoplasma-like organisms (MLOs) are plant pathogenic mycoplasmas that are non-helical, non culturable and transmitted by arthropod insect vectors (Tully, 1993). The prokaryotes are phloem bound and observed only in sieve tubes. The mollicutes are delimited by a trilamellar unit membrane and contained DNA strands and ribosomes (Solomon, 1991).

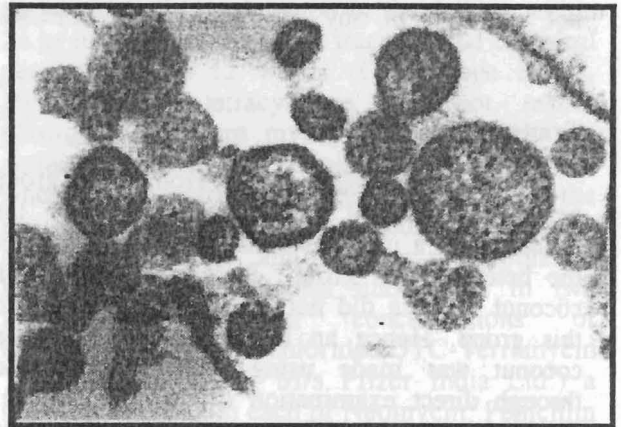


Fig. 3. Phytoplasma in sieve tube of tender rachilla of root (wilt) disease

Pleomorphic forms varying from circular to oval and occasionally beaded or filamentous ones are observed. The size of the coccoid forms ranged from 250-400 nm (Fig.3).

In sieve tubes the phytoplasma generally occupied a parietal position close to the sieve tube wall or seen in proximity to sieve plates (Solomon, 1997). Distribution of the phytoplasma within the vascular bundle is rather sparse and not all sieve elements in a patch contained them. Some of the bundles are totally free of the organism. In the older leaves moribund forms lacking internal contents only were observed (Solomon and Geetha, 1993). The apparent absence of phytoplasma in mature parts and their occurrence in tender tissues suggest the movement of the organism in the phloem assimilate to the "sink region".

Constant association of phytoplasma with the disease has been conclusively established with the finding of the organism in more than eighty diseased palms and their absence in seventy-five healthy palms studied. The palms examined are of various age groups, intensities of disease and from different locations. None of the other biological agents reported earlier to be associated with the disease could be observed in the vascular tissues (Solomon *et al.*, 1999). The possible association of trypanosomatid flagellates was also studied by examining rachilla of inflorescence of six palms each in the early, middle and advanced stages of disease. The protozoan flagellate was

not observed in any of the samples (CPCRI, 1991).

Transmission

Insects belonging to the Homoptera group generally transmit Phytoplasmas with the exception of a member of the Pentatomorpha of the heteropterous group. Record of insects on coconut in India did not enlist any belonging to this group. Hence an inventory of insects on coconut, was made using various traps and through direct examination of coconut foliage. This led to the identification of a leaf hopper *Sophonia greeni*, a plant hopper, *Proutista moesta*, both belonging to the Homopteran group, besides lace wing bug, *Stephanitis typica* (Tingidae) already known putative vector (Rajan and Mathen, 1984; 1985). A rapid survey of representative gardens in the eight southern districts where the disease is prevalent in a contiguous manner confirmed the presence of all the three insects.

Acquisition of phytoplasma by these putative insect vectors while feeding on diseased palm was studied. Phytoplasma was observed in the salivary glands of lace bug 18-23 days after feeding on diseased palms (Mathen *et al.*, 1987) and also in plant hoppers fed on diseased palms for over 30 days (CPCRI, 1991; Rajan *et al.*, 2002). The vector role of lace wing bug and plant hopper have been conclusively established in transmission experiments (Mathen *et al.*, 1990; CPCRI, 1996).

Field experiments were conducted to assess the control of aerial insects on fresh incidence of disease. Even fortnightly spraying of insecticide with higher concentration of both systemic and contact insecticide could not protect the seedlings from disease contraction. The perennial nature of the crop, the presence of insect vector throughout the year, the persistent mode of transmission of phytoplasma and the reinfestation of sprayed plants within a short period may be the contributing factors for the failure to control the disease. Large scale chemical treatments, at frequent intervals also would be costly and can pose environmental hazards (Solomon *et al.*, 1999).

The phytoplasma also could be experimentally transmitted from coconut to periwinkle, a mycoplasmal indicator host through dodder laurel (*Cassytha filiformis*). Periwinkle grown in insect proof cages bridged to diseased coconut palms through dodder laurel developed chlorotic spots in the interveinal areas and at vein endings of fully opened leaves in three to four weeks of haustorial establishment. Detection of phytoplasma in periwinkle, dodder laurel and the source palm confirmed transmission of the disease (Sasikala *et al.*, 1988).

Culturing

Although the phytoplasma associated with root (wilt) disease had been transmitted through insect vector to coconut seedlings and to periwinkle through the dodder laurel, culturing of the organism in cell-free medium and assessing its pathogenicity is one of the basic tenet of Koch's postulate. Phytoplasmas as a group have not been cultured so far in cell-free medium. Nevertheless concerted effort was made to culture the organism from rachilla of unopened inflorescence, spear leaf, mid vein and vascular sap from root (wilt) diseased palms, petiole and mid vein of symptomatic periwinkle and infective lace bugs in about 40 different media (CPCRI, 1988, 1989a; Solomon *et al.*, 1999).

Phloem sap as such or supplemented with serum is found to be an ideal medium for culturing fastidious organisms such as *Acholeplasma laidlawii*, *Mycoplasma fermentans*, *Spiroplasma citri* and *Phytomonas davidii* (Eden-Green and Waters, 1982; Mc Coy, 1976, 1977, 1978). A method for aseptic collection of vascular sap in unmodified form, from inflorescence of coconut palms in ice packed vacuum flasks was standardised and the biochemical constituents of the sap from apparently healthy and diseased palms analysed (Rajagopal *et al.*, 1988; Chempakam and Rajagopal, 1989). The data provided a rationale for formulation of new media. Freshly collected vascular sap from apparently healthy palms as such or supplemented with nutrients was tried as potential medium for culturing. Coconut water from nuts of healthy palms was also used as a basal constituent of certain media. However the intraphloemic phytoplasma was found not

amenable to culturing in any of the medium (CPCRI, 1989b).

Attempts were made to culture the root (wilt) phytoplasma in chick embryos by employing different routes of inoculation and varying cultural conditions. Although in an identical study the mycoplasma associated with phyllody disease in sesamum could be cultured in chick embryos, the root (wilt) organism had defied culturing in this potential medium also, confirming its non-adaptability to environment outside the phloem tissue (Srinivasan and Solomon, 1992).

Similarly, attempt was also made to maintain the organism in insect tissues through organ culture. Excised salivary glands and midgut segments of infective lacewing bug and plant hopper were inoculated to insect tissue culture medium. Phytoplasma could not be maintained in the insect tissues in culture.

Another line of investigation pursued at present is on maintenance of phytoplasmas in plant tissues micropropagated in vitro in tissue culture media. Phytoplasma with internal contents could be observed in the roots in culture up to nine months and thereafter only moribund forms were evident (Solomon *et al.*, 1999).

Chemotherapy

Phytoplasmas being non-cultivable in cell-free media posed a problem in testing its pathogenicity and fulfilling the Koch's postulates mandate. Characteristically the prokaryotes lacking a cell wall and delimited by cell membrane are insensitive to penicillin and are sensitive to tetracycline group of antibiotics. This differential chemotherapeutic response is accepted and used as an adjunct to provide circumstantial evidence on the etiology of phytoplasma induced diseases. To assess the efficacy of any antibiotic on the organism it should be ensured that the chemical reaches the target site in unaltered state within a reasonable period. Pillai and Raju (1985) had fabricated a pneumatic pressure injector for administering the antibiotic. The uptake and translocation of the antibiotic administered was studied employing a tracking dye and through bioassay using *Bacillus cereus* sub sp. *mycoides* as the test organism. The

antibiotic could be detected in the foliage within 24 hr of its application and traces of the chemical persisted up to 12 weeks (Chowdappa *et al.*, 1989). Since tetracyclines are not mycoplasmacidal but are mycoplasmastatic only the antibiotic needs to be administered to the plant when the concentration petered out. Based on the results of the bioassay studies, quarterly application of the antibiotics was stipulated. Results of the field trial undertaken in two locations with four concentrations of Oxytetracycline hydrochloride (OTC-Terramycin Tree Formulation of M/s Pfizer India Ltd.) a single concentration each of Neomycin, Penicillin and distilled water control revealed remission of symptoms in 53% of the palms treated with 3 and 6g a.i. of OTC. Palms treated with Penicillin and distilled water control deteriorated over the pre-treatment condition thus providing further evidence to the mycoplasmal etiology of the disease (Pillai *et al.*, 1991). The field application of OTC however cannot be recommended either as a prophylactic or curative measure as the antibiotic needs to be given repetitively and temporary remission of symptom only is obtained. Prohibitive cost of the antibiotic and the caution against its indiscriminate use for treating any plant disease are the other factors, which should weigh against it being recommended as a treatment.

Screening of coconut germplasm

Field evaluation of 63 cultivars and 32 hybrid combinations since 1972 indicated that none of the cultivars/hybrids manifest resistance /tolerance to the disease (Jacob *et al.*, 1991). However the hybrid, Chowghat Orange Dwarf (COD) x West Coast Tall (WCT) under ideal management yielded higher number of nuts compared to WCT palms of identical age in the early years of production (Cecil and Amma, 1998). Germplasm collection (24 exotic accessions) made from South Pacific Ocean Islands in 1981 have been planted at the World Coconut Germplasm Center at Sipighat in Andamans with a view to screening their progenies and different cross combinations against the disease (Jacob *et al.*, 1998).

Breeding for disease resistance / tolerance

An intensive survey was conducted in the heavily diseased hot spot areas to locate high yielding apparently healthy WCT palms of over 35 years. About 200 palms have been so far identified in hot spots of Alappuzha, Pathanamthitta, Kollam and Kottayam Districts of Kerala State. Chowghat Green Dwarf (CGD) showed field tolerance of over 90 per cent to root (wilt) disease (CPCRI, 1972). About 110 CGD and 75 WCT disease free palms identified in hot spots are being used in the crossing programme in various combinations. About 6000 seedling progenies raised from the crosses involving disease free WCT and CGD mother palms in hot spots have so far been planted in five locations to establish nucleus seed gardens at Thrissur, Kottayam, Alappuzha and Kollam Districts. These plants when come to bearing will partly meet the planting requirements of these districts (CPCRI, 2003).

Disease management

One of the significant features of the disease is that it is not lethal but a debilitating malady, which responds to ideal management. Two strategies, one for the heavily diseased contiguous area, and another for the mildly affected area have been formulated. In the heavily diseased area, the yield of palms can be sustained or even improved through adoption of integrated management practices-removal of disease advanced and juvenile palms, balanced fertiliser application, addition of organic manures, raising of green manure crops in the basins and incorporation, irrigation during summer months, practicing plant protection methods and adopting inter and mixed cropping (CPCRI, 1986). Mixed farming in the diseased gardens involving the raising of fodder crops in the inter spaces, maintaining milch cows and recycling of organic waste has aided in increasing the yield of palms by 28.1% (Sahasranaman *et al.*, 1983). Mixed cropping with cocoa increased the yield by 27-35% and slowed down the decline of palms (Amma *et al.*, 1982). Similarly, cultivation of tuber crops such as tapioca, elephant foot yam and yams in the interspaces of palms in the disease affected gardens for a period of three

years increased the nut yield by 4.96 to 15.57 and 8.07% respectively (Menon and Nair, 1978).

The strategy for the mildly affected area is to contain the disease by removing all the diseased palms. Following the eradication of a few diseased palms at Shenkotta (Tamil Nadu) and at Nadathara (Kerala) in 1971, recurrence was not noticed subsequently (CPCRI, 1986). With the above encouraging results an attempt was made to arrest the spread of the disease beyond Karuvannoor River in Thrissur district and to contain it within the geographical limits. With the removal of 730 diseased palms from 341 gardens in ten villages in Thrissur district during 1979 to 1982, the disease could be brought to just 21 palms spread over in 15 gardens in three villages in 1989-1990 (Muralidharan *et al.*, 1998).

Since there are no curative or prophylactic measures available to combat the disease the long-term option is to breed for disease resistance. Concurrently adoption of recommended management practices could yield economic returns per unit area. Systematic rouging of diseased palms in the mildly affected area could prevent the further spread of the disease.

Tatipaka disease

Occurrence, distribution and crop loss

Tatipaka disease was first noticed in Andhra Pradesh after the 1949 cyclone and named as 'Tatipaka' disease after the village in which it was first noticed. (Rao *et al.*, 1956). The disease is endemic to East and West Godavari, Srikakulam, Nellore, Krishna and Guntur districts (Subbiah and Rao, 1963; Rethinam *et al.* 1989). It generally occurs in heavy black deltaic soils than in sandy, sandy loam and red loam soils. The disease is observed in well-managed and neglected gardens. A survey made during 1985-1990 in the central delta of the river Godavari "Konaseema" which account for 60% of the area under coconut in the state indicated that the disease is prevalent in 85 out of 201 villages (Narasimhachari *et al.*, 1991). About 8179 palms were identified as Tatipaka diseased (Rajamannar *et al.*, 1993).

The disease is non-lethal but of a debilitating nature, generally affecting palms in the age groups of 20-60 years. Palms below 20 years are very rarely affected (Rethinam *et al.*, 1989). The spread of the disease is not contiguous but sporadic at a slow pace of 3.5% over a period of five years.

Symptoms

The disease-affected palms generally bear profusely for 2-3 years before the expression of foliar symptoms and more number of dark leaves often fasciated appears in the crown. With the onset of disease there is a reduction in both number and size of leaves (Fig. 4). The leaves exhibit characteristic chlorotic water soaked spots and the fronds bend abnormally sometimes twisting in loops. In the advanced stage with the narrowing of leaflets and reduction in size of crown, the affected palm looks like a Date palm in appearance (Fig. 5). The spathes produced are very small with very few rachilla. The bunches contain a mixture of normal and atrophied nuts (Fig. 6). The atrophied nuts are barren with thinner spongy mesocarp with or without shell, copra and nut water. The undersized nuts show longitudinal cracks with occasional gumming. In the advanced stage of the disease the stem tapers and produces smaller spathes and inflorescences, which ultimately do not bear any fruit (Rao, 1966; Ramapandu and Rajamannar, 1981).



Fig.4. Symptoms of Tatipaka disease

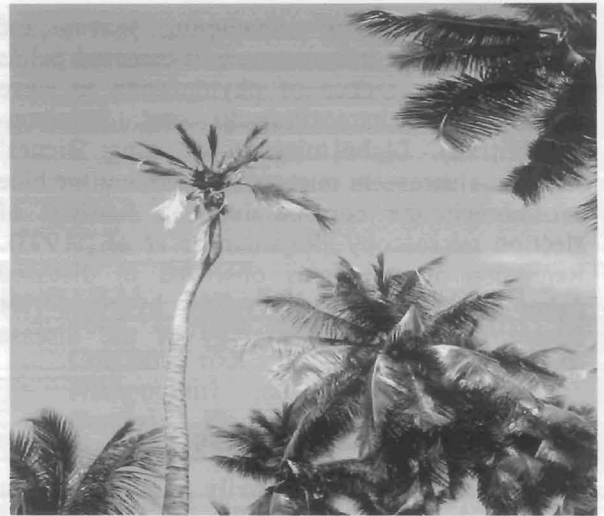


Fig.5. Tatipaka diseased palm

Etiology

The disease was considered as one of uncertain etiology till recently. The involvement of fungi and bacteria in the incidence of the disease was conclusively ruled out, as these microbes could not be consistently isolated from diseased palms. Sap transmission and purification studies have also ruled out virus as the etiological agent of the disease (Ramapandu and Rajamannar, 1981; Rajamannar *et al.*, 1984). Polyacrylamide gel electrophoretic analysis of isolated nucleic acid from Tatipaka diseased palms did not identify any viroid type pathogen either (Randles and Hatta, 1980). Electron microscopic examination of tender roots, sub-

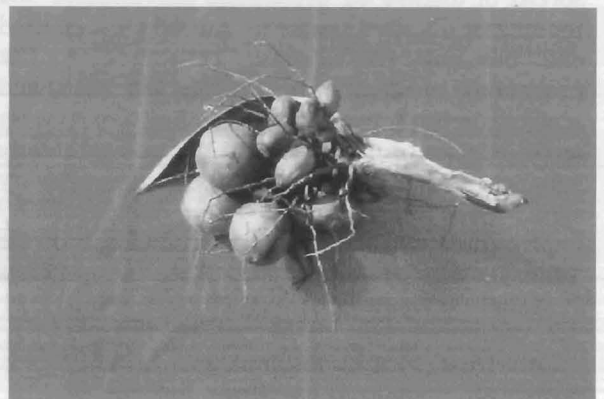


Fig.6. Normal and atrophied nuts in a bunch

meristem, petiole of developing leaves and rachilla of tender inflorescence of diseased palms revealed the presence of phytoplasma in sieve tubes (Rajamannar, Prasadji and Solomon-unpublished). Light microscopy using Dienes' stain and fluorescent microscopy with aniline blue as fluorochrome corroborated the findings of electron microscopy (Rajamannar *et al.*, 1993). Remission of symptoms observed in diseased palms treated with tetracycline hydrochloride further confirmed the etiology of the disease (Rajamannar *et al.*, 1993).

Screening for disease resistance

Survey of the heavily diseased area indicated the cultivar; Gangabondam to be free of disease and the incidence was high on the East Coast Tall. This observation has to be confirmed so that this cultivar could be used as a parent in the future breeding programme for evolving disease resistant genotypes.

Disease management

Attempts to control the disease by foliar and soil application of various chemicals, hormones and nutrients did not yield any encouraging results. Root regeneration of affected palms also could not ameliorate the condition of palms (Rajamannar *et al.* 1993).

Since there are no prophylactic or curative measures available for treating phytoplasmal diseased plants, the option left are to arrest the spread of disease by systematic surveillance and roguing of diseased palms as and when identified and breeding for disease resistance. The programme of identifying the diseased palms and eradication is a more practical step in this disease as the number of diseased palms is comparatively very low and the spread is reported to be very slow. The programme if systematically implemented will help in total eradication of the Tatipaka disease from the State of Andhra Pradesh.

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