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DISEASES AND DISORDERS OF COCONUT AND THEIR MANAGEMENT

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COCONUT (*Cocos nucifera* L.) is one of the most useful plants in the world. The coconut palm, described as 'Kalpavriksha', 'Tree of Life', etc. is a powerful and enduring symbol with multiplicity of uses. This plantation crop is a source of life, food, shelter and rural income in the developing world, especially in the humid tropical regions. A blessing of the crop is that it is a long term, though low-input, crop and it can tolerate harsh conditions and low inputs. However, coconut cultivation is threatened by the impact of various diseases in different parts of the world and serious epidemics had devastated millions of palms in the past. Epidemics are active in different continents and certain diseases are endemic.

Coconut palm is affected by many diseases, due to various pathological agents, namely, phytoplasmas, fungi, viruses, viroids, flagellates, bacteria and nematodes. Different parts of coconut—bud, leaf, stem, root, fruit, etc. – are affected and the diseases may be systemic (involving the whole palm) or non-systemic (localized to a specific part) depending upon the pathogen involved or the nature of the disease as the case may be. Coconut palm is also affected by certain disorders (non-pathological) due to imbalance nutrients, etc. Among all groups of diseases, the phytoplasmal and fungal diseases seem to be most widespread and devastating. In this chapter the diseases and disorders of coconut are described in parts A-D. The phytoplasmal and fungal diseases in general and such diseases with special reference to India are dealt with in detail.

PART – A: PHYTOPLASMAL DISEASES

HISTORY

Phytoplasma (previously referred to as mycoplasma-like organisms, MLOs) has been associated with over 300 plant diseases, ever since the first report of association of such

an organism with plant disease (Doi *et al.*, 1967). Under the aegis of the Food and Agriculture Organization (FAO) of the United Nations, Maramorosch (1964) reviewed in a worldwide survey, the diseases of uncertain etiology of coconut and listed 11 diseases as of unknown etiology. In the past three decades, several coconut diseases of previously uncertain or unknown etiology have been determined and association of phytoplasma with several of such cases has been reported.

Phytoplasmas are wall-less prokaryotes, found in the phloem sieve tubes of (Yellows) affected plants. These agents invariably induce systemic symptoms, transmissible and the remission of symptoms could be achieved by antibiotic therapy. Unlike Spiroplasmas (the other group of plant mycoplasma), Phytoplasmas have never been cultured. Animal (cultivable) and plant mycoplasmas (non-cultivable) were shown unrelated by DNA hybridization. Molecular techniques, however, showed relationships amongst the plant MLOs, and hence a new classification of this group of organism has been proposed under the name 'Phytoplasma' (Albanese *et al*, 1994). Evidence from analysis of 16S rRNA gene showed the close relationship of Phytoplasmas with Acholeplasmataceae. Kirkpatrick *et al*, (1987) cloned Phytoplasma DNA fragments from the insect vector of Western-X disease that began a new age of plants Photoplasmal investigations. Considerable advances in scientific knowledge of coconut Phytoplasmal diseases have been made. The current world distribution of Phytoplasma associated with coconut diseases is presented in Table 1.

LETHAL YELLOWING DISEASE (LYD) AND RELATED DISEASES

Background

LYD was first reported about 100 years ago from Jamaica and subsequently it became established in several other countries (Cuba, Cayman Islands, Haiti, Bahama Islands, etc.). It also established itself in Florida (USA) in the early 1970s; spread to Yucatan Peninsula of Mexico in the late 1970s and to Belize in 1993. Lethal diseases of coconut with symptoms similar to those of LYD (of Caribbean, etc. region) were reported in the early 1900s form the East (Kenya, Mozambique and Tanzania) and the West (Nigeria, Ghana, Togo, Cameroon) African countries. Among these coconut diseases, Phytoplasma was first discovered in the LYD infected palms in 1972 in three independent laboratories (Beakbane *et al*, 1972; Heinze *et al*, 1972; Plavsic-Banjac *et al*, 1972). Within a considerable period of discovery of Phytoplasma in Caribbean LYD, these agents were shown to be present in coconut diseases with similar symptoms in both East and West Africa also (Schuiling *et al*, 1981; Schuiling and Forstel-Neuhaus, 1992).

Symptoms

LYD and similar diseases cause the rapid death of palms and susceptible varieties usually die within 3-7 months of the first appearance of symptoms. The early symptoms of

TABLE 1

Phytoplasmal Diseases of Coconut Distributed in Different Parts of the World

No.	Name of Disease	Geographical Distribution	Insect Vector
1.	Lethal yellowing disease	Caribbean Islands (Bahamas, Cayman Islands, Cuba, Dominican Republic, Haiti, Jamaica) USA (South Florida and South Texas) Mexico Belize	*Myndus crudus*
2.	Blast, Dry bud rot	Ivory Coast (West Africa) (Also suspected in South American countries and Indonesia)	*Recilia mica*
3.	Cape St.Paul wilt disease	Ghana (West Africa)	*Myndus adiopodumensis*
4.	Kaincope	Togo (West Africa)	Not known
5.	Kribi disease	Cameroons (West Africa)	Not known
6.	Awka wilt/Bronze leaf wilt	Nigeria (West Africa)	Not known
7.	Lethal diseases	East African countries, namely, Tanzania, Kenya and Mozambique	Not known
8.	Coconut stem necrosis*	Malaysia Sumatra (Indonesia) (South East Asia)	Not known
9.	Root wilt disease	India (Kerala and Tamil Nadu States) (South Asia)	*Stephanitis typica* and *Proutista moesta*
10.	Tatipaka disease	India (Andhra Pradesh State) (South Asia)	Not known

* Current evidence (Harries, 1998) points to the possible association of Phytoplasma in Natuna wilt and Kalimantan wilt diseases also in Indonesia

the disease include shedding of immature nuts, blackening of immature inflorescence, necrosis of spear (spindle) leaves and death of actively growing root tips. Discolouration and necrosis of leaves start usually with the oldest and progress to the younger ones, but occasionally mid-whorl leaves are affected at an early stage. With the progress of foliar discolouration, the spear leaves cease to expand and become necrotic. A foul smell emanates from the internal tissues of the spear and the growing point is affected. In Jamaica and Ghana seasonal effect on symptoms has been observed.

Pathogen

The Phytoplasma is non-cultivable and the disease is spread by insect vector. Severe disturbances occur in phloem transport; several physiological and biochemical changes occur in diseased palms (Dabek, 1974; Eskafi *et al*, 1986; Oropeza *et al*, 1995). Concentration of Phytoplasma in coconut seems to be particularly low in comparison with other plant diseases. The distribution of phytoplasma is that the highest densities are found at phloem sink sites such as base of spear leaf, developing inflorescence and root tips.

Phytoplasmas of Caribbean LYD have been detected by molecular diagnostic techniques like DNA probes (Harrison *et al*, 1992) and Polymerase chain reaction-PCR (Harrison et al, 1994a), and such techniques have been applied for detection of East and West African LYD-like diseases.

Although the diseases both in the Caribbean and West and East African countries are associated with Phytoplasmas with extensive similarities in symptomatology, they appear to involved genetically different strains of the pathogen (Eden-Green, 1994a). Using PCR analysis, Phytoplasmas of Tanzania and Kenya (East Africa) were shown similar (probably identical) but they were found to differ from the Phytoplasmas of Cape St. Paul wilt and Awka diseases (West Africa). Both groups of African coconut Phytoplasmas differed from the Caribbean Phytoplasmas, which in turn were more closely related to East African than West African Phytoplasma. Primers based on cloned random DNA were thought to provide fragments better technique to distinguish between these Phytoplasmas (Lee *et al*, 1993). Sequence analysis of coconut 16s rDNA has shown that the Phytoplasmas found in Ghana and Nigeria are possibly the same as their sequences had more that 99 per cent similarity; whereas similarities between the Phytoplasmas of East and West Africa was 95.3 per cent; differences between the African and Caribbean Phytoplasmas were between 94 and 97 per cent (Tymon and Jones, 1997). Jones and Tymon (1997) by using the PCR reaction amplified the 16S rRNA genes and the 16S-23S spacer regions of Phytoplasmas of LYD-like diseases in Africa. Based on sequencing of the rDNA products, two primers (specific for East and West African diseases) were designed. Neither of these primers, when paired with a universal primer, produced PCR amplification products from healthy coconut DNA, infected coconut DNA from the Caribbean or DNA from a variety of periwinkle-maintained Phytoplasmas. Interestingly, phylogenetic analysis confirmed that these coconut Phytoplasmas form a primary

cluster (within the Phytoplasma clade) and the Phytoplasma of coconut diseases in West Africa formed a new sub-clade within this cluster. Specific amplification of Phytoplasma rDNA from highly diluted extracts of coconut tissues was further standardized (Rhode *et al*, 1993) and molecular analysis established the existence of genetic relationships between Phytoplasmas associated with lethal decline disease in the Caribbean and Africa but indicated that the pathogens of the diseases, although very similar, are not genetically identical (Harrison *et al*, 1994b); genetical relationship between pathogens of West and East Africa are also that they are not identical (Tymon *et al*, 1997).

Control

Control of LYD and similar diseases depends mostly on integrated management practices with a thrust on exploitation of host genetical resistance to the disease. Since hardly much time is left from the onset of disease symptoms to the death of the palms and the nature of the disease itself, no conventional plant protection measures are known for control or cure of infected palms. Rehabilitation programmes have been adopted in diseased regions. For example, in Jamaica, Malayan Yellow Dwarf was found resistant to the disease; in Ghana, Sri Lanka Green Dwarf and Vanuatu Tall varieties were found to have low susceptibility to the disease (Harries, 1997; Dery *et al*, 1997). Long-term national strategies are evolved, assisted by international fora for management of LYD and like diseases with national-international perspectives (Eden-Green and Ofori, 1997). Prevention of the disease from spreading to newer areas is of paramount importance and strict quarantine measures, while exchange of breeding material, is imposed. FAO has issued guidelines for the safe-movement of coconut germplasma material in order to strictly avoid the introduction of LYD-like diseases to unrecorded countries.

COCONUT DISEASES OF RELATED, YET UNCERTAIN ETIOLOGY

A number of coconut diseases of uncertain etiology have emerged. The cause(s) of such diseases have still not been conclusively established. These unresolved problems of coconut are: Leaf scorch decline (Sri Lanka); Rapid decline diseases like Malaysian wilt (Peninsular Malaysia, Sarawak, etc.); Wilt syndromes in Indonesia (Natuna, Riau, Sumatra, Kalimantan, Sulawesi) and certain diseases in Madagascar.

The area of such diseases of greatest uncertainty lies in Southeast Asia. Symptoms of these diseases, however show close similarities, and possibility of a common cause for these diseases is suspected; possible involvement of a phloem limited prokaryote is very much considered (Eden-Green, 1997b). About 20 years earlier, it was predicted that LYD-type of diseases would prevail in the Far East (Harries, 1978; Chiarappa, 1979). It seems early evidence of association of Phytoplasma with Natuna wilt and Kalimantan wilt (Indonesia) has flown out in support of such speculation (Harries, 1998). It may be hoped that causes of all such diseases are resolved in the near future with the advent of rapid

detection models available to researchers. Satisfactory resolution of these diseases would enhance the management measures.

PHYTOPLASMAL DISEASES OF COCONUT IN INDIA

In India, there are two diseases in coconut, namely, root wilt disease and Tatipaka disease where association of Phytoplasmas has been reported. These diseases are considered economically important in the country and hence are reviewed in detail.

Root Wilt Disease

Root wilt disease (RWD), as popularly referred to by researchers, is often pronounced as Kerala wilt and in the vernacular (Malayalam) language as 'Kattuveezhcha' (meaning the disease carried by wind).

Background: RWD was reported to be present, since 1882, initially at three pockets in the erstwhile princely State of Travancore (Varghese, 1934) and now endemically prevails in eight southern districts of Kerala State (26.44 per cent out of 9.15 crore palms). The disease also has appeared in isolated pockets in the northern districts of the State and in the borders of the adjoining State of Tamil Nadu. The loss due to the disease was estimated as 968 million nuts annually (CPCRI, 1985a). The latest survey shows the incidence of RWD in 24.05 per cent out of 10.20 crore palms in disease endemic districts (Department of Agriculture, Government of Kerala, 1997). In contrast to the LYD in the Caribbean Islands, Africa, etc. RWD is a non-lethal disease but a slow decline, debilitating disease. The disease incidence occur irrespective of the age of the palms and in all major soil types. The spread of the disease is slow.

Symptoms: The consistent and visual diagnostic symptom of RWD has been 'flaccidity', the characteristic bending-ribbing of the leaves in the middle and outer whorls of the crown (Radha and Lal, 1972). Later symptoms are foliar yellowing and marginal necrosis—usually observed in diseased adult palms. In young palms/seedlings only the flaccidity symptom is usually observed. An indexing system based on flaccidity, yellowing and necrosis symptoms, to quantify the intensity of RWD infected palms was evolved (George and Radha, 1973). Softening and whitening of leaflets of the spindle with necrotic spots followed by rotting in diseased palms are also observed; yellowing of leaves in the middle whorl occasionally seen (Dwivedi *et al*, 1979). Butler (1908) considered root rot as the major symptom of the disease. Root regeneration capacity of diseased palms is affected. Inflorescence necrosis is occasionally noticed (Menon and Pandalai, 1958). Pollen sterility/ low viability, yield reduction, depending on disease intensity (Jacob Mathew *et al*, 1993) are the other manifestations of RWD. Female flowers and immature nuts shed abnormally resulting in low yield and if the palms in pre-bearing age contract the disease they remain unproductive. The quality of nuts in the diseased palms is also affected—kernel becomes rubbery, flexible;

husk thinner and weaker; oil content reduced. The quality of coconut water was also affected; though little effect on sugar content, aminoacids increased with advancing infection as did Mg, Ca and P, while ash, N and K contents decreased (Sosamma Cherian and Nair, 1993).

Stunted epidermal cells, reduced cuticle thickness in lower surface of leaves, differential rate of division in upper epidermis, reduced thickness of cell walls, etc. are the anatomical derangements noticed in infected palms (Solomon, 1994). In roots of diseased palms browning of vascular elements, disintegration of vascular tissues (Indira and Ramadasan, 1968), tyloses in xylem and necrosis of phloem are noticed (Govindankutty and Vellaichamy, 1983). Sero-diagnostic (Solomon *et al*, 1983) and physiological (stomatal resistance) (Rajagopal *et al*, 1986, 1988) tests have been evolved for possible pre-visual; diagnosis of the disease. The RWD affected palms are super-infected severely by leaf rot, caused by fungi, which bring about rapid decline of palms.

Etiology: A long history may be traced on the investigation of the etiology of RWD. Biological agents (fungi, bacteria, nematodes, virus), physiological, soil versus nutritional aspects, etc. were viewed earlier. The disease as fungoid nature was proposed by Bourdillon in 1906 (Varghese, 1934). Butler (1908) reported *Botryodiplodia* sp. in root rot of diseased palms that was then considered as the possible cause. In later studies various species of fungi (*Rhizoctonia solani*, *Rhizoctonia bataticola*, *Fusarium equisetii*, *Cylindrocarpon effusum*, *Cylindorcarpon lucidum*, etc.) were isolated from the roots. Isolation of bacteria from the roots of diseased palms was reported by Menon and Nair (1951). Srivastava *et al* (1969) reported a bacterial streaming movement from the roots of diseased palms and isolation of a bacterium which was tentatively identified as *Pseudomonas* sp. George *et al* (1976) reported isolation of *Enterobacter cloacae*. The absence of typical bacterial streaming but only bacterial cloudiness in decayed roots from diseased palms, and dominance of Gram (+)ve bacteria (*Bacillus* sp.) over Gram (-)ve ones in isolation from the roots were observed in due course: possible non-involvement of phytopathogenic bacteria in RWD was pointed out at critical stage (CPRI, 1984). Nematological investigations lead to identification of 35 genera of nematodes from coconut roots/soil around it; *Radopholus similis* was assessed with special emphasis of its role, if any, in RWD. In fact, no correlation was available between the presence of nematode and occurrence of RWD. Weischer (1967) earlier opined with strong scientific facts to exclude the nematodes from consideration as the cause of RWD. In inoculation studies fungi (Joseph and Lily, 1991), bacteria (Jayasankar and George, 1991) and nematodes (Sosamma and Koshy, 1991) singly and in combination did not produce the symptoms of RWD. The role of these organisms in the diseases was ruled out.

Varghese (1934) ruled that the disease was not physiological. Detailed studies on analysis of soil, tissue samples and physiological changes in the diseased palms pointed more towards a pathological derangement in the plant rather than a soil or physiological disorder (Mathew *et al*, 1991; Cecil and Amma, 1991). In the affected palms, stomatal regulation was adversely affected (that is, stomata failed to close) that resulted in excessive water loss (Rajagopal *et al*, 1986; Rajagopal, 1991). Various other physiological/biochemical characters

analysed in the diseased palms did not point out any of these factor(s) as the cause of the disease. Geo-chemical factors were also related in the incidence of RWD. Mathai *et al* (1984) indicated that the deficiency of Ca, Mg or Zn might predispose the palms to infection by RWD pathogen. Higher concentrations of nickel and strontium in the roots of diseased palms were reportedly found by X-ray fluorescence technique (Wahid *et al*, 1983). Biddappa (1985) indicated a high deposition of chromium, titanium, copper, lead, bismuth and gallium (heavy metals) in the cabbage tissues of RWD infected palms whereas in the roots, aluminum, manganese, copper and cobalt were found. A hypothetical role of lanthanides (cerium), in conjunction with magnesium deficiency, in the causation of RWD was proposed (Valiathan *et al*, 1992, 1993; Eapen, 1993). Deficiency of gadolinium in the RWD endemic area (foliar and soil analysis) was also related (Wahid, 1998). All these reports, however, did not deliver conclusive evidence as to such element(s) as the cause or predisposing factor for RWD.

Virus as the probable cause of RWD was also thought of (Nagaraj *et al*, 1954) and several papers appeared on this line. Summanwar *et al* (1969) reported the isolation of TMV from diseased palms. Maramorosch and Kondo (1977) also reported (icosahedral) virus-like particles from the leaves of diseased palms. No virus could, however be conclusively isolated from any of these studies and hence the claim of virus as the cause of the disease did not last. Randles and Hatta (1980) excluded viroid also from the etiology of RWD, by analysis of nucleic acids from diseased palms.

Phytoplasma as the Cause of RWD: A major constraint in the diagnosis of plant mycoplasmal diseases has been that (except the *Spiroplasmas*) the organisms are not cultured in a cell-free media. Hence the association of the sub-microscopic mycoplasmas with various plant diseases was necessarily to be detected by electron microscope, since the first report of Doi *et al*, (1967), even while supplementary microscopical techniques (such as fluorescence, staining, etc.) were in vogue and helped (Dienes *et al*, 1948; Deeley *et al*, 1979; Schuiling *et al*, 1981; Srinivasan, 1982). As the possible involvement of MLOs in RWD was suspected, electron microscopical examination of RWD palms was felt essential. Initially, four RWD affected palms were sampled in 1982 by a group of workers including the author, and the samples examined in Germany by Prof. F. Nienhaus. Electron microscopic examination of various parts of these four RWD infected palms revealed the presence of Phytoplasma (MLOs) in sieve tubes (Solomon *et al*, 1983). Increased number of the organism bodies prevailed in the sieve tubes of sink region (sub-meristem, petiole of spindle, young inflorescence, root-tip, etc.); the pleomorphic forms, bound by a triple layered unit membrane and have DNA stands, ribosomes that ought to resemble such organism as already described in several other plant diseases. Constant association of the organism with RWD (and its absence in healthy) palms has now been established. Structural anomalies (elaborated membrane, large number of cell organelles, presence of paramural bodies, electron dense material, callose, phytoferritin, etc.) were noticed in vascular tissues (Solomon and Geetha, 1993). Later (to initial studies) supplementary staining technique (such as Diene's staining, fluorescence with DAPI) in detection of the diseased palms was reported by an art of sampling (Solomon *et al*, 1987).

The distribution of the organism in the infected palms seems to be non-uniform which is in conformity particularly with the reported low titre/concentration of Phytoplasma in LYD affected coconut palms in the Caribbean Islands, etc. (Thomas, 1979). This phenomenon seems to be unique to coconut and hence, detection of the RWD organism might be critically faced.

Lace bug—*Stephanitis typica* (Distant)—was suspected earlier (Shanta and Menon, 1959) as a probable vector of RWD, that was corroborated (Mathen *et al*, 1976). In addition to lace bug, leaf hopper—*Sophonia greeni* (Distant)—and plant hopper—*Proutista moesta* (Westwood)—were detected as vector candidates in RWD endemic region (Rajan and Mathen, 1984, 1985). The phloem feeding nature of lace bug was ascertained (Mathen *et al*, 1988) and with acquisition and incubation (A+1) period of 18-23 days on diseased palms, the insects acquired and hence the Phytoplasma observed in the acini of the salivary glands (Mathen *et al*, 1987). The Phytoplasma was transmitted from diseased palms to healthy palms (grown under insect proof controlled conditions) by lace bug, offered 18 days of A+1 period; transmission confirmed by electron microscopy, etc. with presence of Phytoplasma in the recipient palms and expression of primary visual symptom (flaccidity) of RWD by them (Mathen *et al*, 1990). In natural conditions, increased colonization of lace bugs in inner leaves of the crown and a direct linear correlation between the insect colonisation and fresh RWD incidence, as established (Mathen, 1985) earlier, are supportive of vector role of the specific insect. The Phytoplasma was detected in plant hoppers also that was given A+1 of over 37 days and vector role of the insect has been established through transmission (CPCRI, 1997). Vector role of leafhopper is yet to be established. The Phytoplasma has been also transmitted from RWD infected seedlings to indicator periwinkle—*Catharanthus roseus*—through dodder laurel—*Cassytha filiformis*—and further serially from periwinkle to periwinkle (Sasikala *et al*,1988). The transmission of the organism by such a vegetative vector is generally in line with other plant diseases of Phytoplasmal etiology.

With the detection of the intraphloemic organism in RWD infected palms, intensive efforts were made during 1983 through 1989, if the organism could be cultured *in vitro*. Over 40 different media with various nutrient combinations, growth factors, vitamins, nucleic acid precursors, animal sera, etc. were tried in different cultural conditions to culture the organism from tissues of RWD infected palms, symptomatic periwinkle and infective lace bugs (Srinivasan and Solomon, 1998a). Phloem sap of coconut as such was found to be a potential medium for culturing of (already cultivable) animal mycoplasmas and plant *Spiroplasmas* (Eden-Green and Waters, 1982) and with that base, nutritional value of phloem sap in context of RWD was also evolved (Chempakam and Rajagopal, 1986; Rajagopal *et al*, 1988). Hence, phloem sap from apparently healthy coconut palms, individually and supplemented with serum, etc. was also utilized in these attempts of *in vitro* culturing. No evidence of the growth of the organism *in vitro* was observed that was confirmed by electron microscopic examinations also. Further in that study, animal mycoplasmas and a plant *Spiroplasma* (from sesamum phyllody) was found to survive and grow in coconut phloem

sap and nut water (developed nuts), derived from healthy palms; the RWD organism did not grow in any of these media as well. (Srinivasan and Solomon, 1998a).

The organism was further subjected to culturing in embryonated hens' eggs through different routes of inoculation. Even then the organism was non-adaptable to the *in vitro* system of the chick eggs and also not cultivable in cell-free media after passage through embryonated eggs. Electron microscopical examination of contents (allantoic and amniotic fluids) of inoculated (incubated) eggs showed no evidence of growth of the organism (Srinivasan and Solomon, 1998b). It must be noted that from the same laboratory, a *Spiroplasma* from phyllody infected sesamum plants was successfully cultured by these authors (Srinivasan and Solomon, 1991) that formed the first record of plant *Spiroplasma* in India. This *Spiroplasma* was soon cultured in embryonated eggs also (Srinivasan and Solomon, 1992). The chick embryo inoculation seems to have been so far not reported (or tried) in any other non-culturable plant mycoplasmas and therefore the results of RWD formed a reference to other relevant diseases of crop plants. The RWD associated organism was thus proved to be filterable-fastidious bodies and non-amenable to grow *in vitro*. In the world scenario, culturing attempts of plant mycoplasmas appears to have been made in limited laboratories only. Efforts made to culture the RWD pathogen may be rated as one of the intensive attempts of culturing.

The use of molecular techniques has currently allowed the detection and classification of plant MLOs. Therefore, on the basis of Phylogenetic studies the term MLOs has been changed to Phytoplasma, as accepted by the International Organization for Mycoplasmology held in Bordeaux, France, between 16-26 July 1994 (Albanese *et al*, 1994). The RWD associated MLOs has fallen within this convention and since named as RWD Phytoplasma. Maintenance of Phytoplasmas in micro-propagated plants for various diseases has been in vogue and similar lines are adopted with RWD Phytoplasma.

Application of the antibiotic—Oxytetracycline Hydrochloride—into the RWD infected palms showed remission of the disease symptoms in 53 per cent of palms among treated ones (Pillai *et al*, 1991). It had been a conventional practice since 1967 to confirm mycoplasmal (Phytoplasmal) diseases of plants by antibiotic mediated symptom remission.

From the foregoing details it must be convincing that RWD is an infectious, systemic disease and the non-culturable biological agent—Phytoplasma—as its cause has received consideration. Detection of RWD infected palms at molecular level, purification of Phytoplasma/analysis of its gene sequences by molecular techniques—PCR, RFLP, etc.—remain to be realized.

Control/Management: RWD as a Phytoplasmal disease is hardly amenable to conventional protection measures. A redeeming feature of the disease is that it is non-lethal (unlike the LYD elsewhere) and hence integrated management measures are advocated. In the disease management, the strategies are eradication of all infected palms in the areas of sporadic occurrence (Trichur district and beyond north in Kerala, pockets in Tamil Nadu); removal of palms in an advanced stage of the disease, diseased pre-bearing palms in the

endemic region, rejuvenation/rehabilitation of plantations with high yielding, quality seedlings and comprehensive adoption of a package of practices as recommended (Jayasankar and Bappa, 1986). Mixed farming, inter-cultivation, irrigation of palms during summer, organic nutrition, balanced fertilization, raising green manure crops in basins, control of leaf rot (a fungal disease severely superimposed on RWD palms that is dealt with later in this chapter), recycling of organic wastes, etc. can render (the early-middle stage infected) bearing palms to sustain the yield substantially (Muralidharan *et al*, 1991). Evolving resistant/tolerant variety against RWD has been the strategy for a long lasting solution. In fact, evaluation of cultivars/hybrids has been going on since long. The hybrid Chowghat Orange Dwarf (COD)X West Coast Tall (WCT) under ideal management reportedly yielded better results (Bavappa *et al*, 1986) than WCT. Healthy high yielding palms identified in the disease endemic region (hot spots) are being utilized in the hybridization programme; Chowghat Green Dwarf (CGD)X WCT hybrids reported as disease tolerant (Nair *et al*, 1996). Seed nuts of exotic accessions from South Pacific Ocean islands were raised in the 1980s at Sipighat (Andamans) for use in screening against RWD. The availability of ideal plant type to farmers may, it is hoped, provide reliable results.

Tatipaka Disease (TD)

Background: The disease was named after a village—Tatipaka (Razole taluk in Konaseema region of East Godavari district, Andhra Pradesh) with its appearance (in 1951-52?) after a severe cyclonic storm hit the region in 1949. However, a survey during 1956-58 at Uddanam area in Srikakulam district pointed out that the disease might have made its appearance 20 years prior to 1949 cyclone (Subbaiah and Rao, 1963). Scientific investigation on the disease probably started in 1959 with the implementation of 'Coconut Diseases Investigation Scheme' to understand its cause and for remedial measures.

TD is found only in Andhra Pradesh. It is a non-lethal, slow debilitating disease like RWD. The survey of the disease in the endemic region and in Konaseema led to the identification of 8,179 infected palms (Rethinam *et al*, 1989). The latest survey also (Narasimhachari *et al*, 1991) revealed widespread occurrence of the disease in Konaseema and the concentrated pockets are Razole, Allavaram and Malkipuram; isolated incidence of the disease is now noticed in Krishna and Guntur districts, implying its spread. Distribution of the disease seems to be more sporadic rather than contiguous but the spread is relatively more in black soils than in sandy or sandy loam soils.

Symptoms: The symptoms of TD are usually observed in palms of 20-60 years age and are rare in young palms. In infected palms, the size and number of leaves are reduced. At an early stage, the lower leaves become light-green and turn to yellowish-green with chlorotic spots and later, all the leaves turn yellowish. Leaflets of some leaves adhere and show fasciation. Leaflets may also develop water-soaked chlorotic spots, which may form streaks. The fronds may appear bow-like due to bending at the middle. Reduction in the

number and size of roots, root rotting—poor regeneration is noticed. Stem tapering below the crown and twisting may also occur. The spathes in diseased palms small with reduced rachilla and bunches are reduced as the years pass. Normal but reduced nuts are produced at an early stage and with disease advancement mixture of normal and atrophied nuts or only atrophied nuts are produced; the trees may even cease to produce nuts and become barren. The nuts in diseased palms become abnormal—change from longitudinal to round, mesocarp turns soft with reduced contents of water and copra and not storable. The nuts ultimately become shrivelled, full of fibre only seen and do not contain shell, water and copra. The symptom parameters are marginally variable among diseased palms. However, in all diseased palms reduced leaf number/size, pale green foliage, rounding of nuts, soft mesocarp, atrophied nuts and decline in crown size are common features. The palms shed leaves and slowly decline. An indexing procedure for the disease was attempted (Ramapandu and Rajamannar, 1983) that was however expected to be modified. To identify the diseased palm, it is desirable to consider a set of symptoms than a single feature (Rajamannar *et al*, 1994).

Etiology: Involvement of fungal or bacterial organism with the TD was ruled out (Rao *et al*, 1966). The possible association of a virus was suspected; however, sap transmission and purification studies (Rajamannar *et al*, 1984) could not yield conclusive results to regard the disease as of viral origin. Randles and Hatta (1980) reported non-association of viroid with the disease; but presence of MLOs, besides virus-like (rod shaped) particles, cytoplasmic crystalline inclusions and rare inclusions in TD samples was pointed out. A need for further electron microscopical examination of the samples from diseased palms was strongly felt, and accordingly association of MLOs with TD was confirmed through electron microscopy (Ramapandu and Rajamannar, 1981). Demonstration of MLOs in phloem of diseased palms was followed later by light microscopic staining and fluorescence methods (Rajamannar *et al*, 1993, 1994).

Leaf hopper (*Sophonia greeni, Idioscopus nigroclypeatus*), lace bug (*Stephanitis typica*) and mealy bug (*Palmiculter* sp.) have been identified as the insects visiting the diseased palms. However, transmission of the disease (MLOs) by any insect vector and/or by vegetative vector (dodder) is yet to be known. There seemed to be no evidence of earlier attempts to culture the organism in a cell-free media, etc. The response of diseased palms to oxytetracycline hydrochloride (in terms of slight symptom remission) has been observed. The TD associated MLOs has been also grouped under Phytoplasma as per the current scenario. Phytoplasma as the cause of TD might be further strengthened and characterization of the organism by molecular techniques, etc. are yet to be determined.

Control/Management: Application of manure (Subbaiah and Rao, 1963) showed no amelioration of the diseased palms. Similarly, fertilizer application did not improve the condition of affected palms, though the vigour became slightly better. Zinc application to soil (Zinc sulphate at the rate of 2 kg/palm twice for one year and then one kg yearly) led to benefits of lesser reduction in the number of leaves, chlorotic spots, faciation, atrophied nuts,

etc. in the diseased palms. Efforts of root regeneration at above bole region and in lower part of trunk by such measures as ringing and filling with sand/silt/farmyard manure showed that except for a check on fast deterioration, no significant improvement in the condition of diseased palms was achieved. Application of various fungicides, antibiotics and other chemicals also proved to be ineffective on the disease control (Rajamannar *et al*, 1984).

As a phytoplasmal disease, no curative measure is available. Hence, a strategic management of TD has become necessary. Eradication of the diseased palms is encouraged and the region needs to be monitored for any fresh incidence of the disease through surveillance. Screening of coconut germplasm against the disease is in progress. The native prestigious cultivar, 'Gangabondam' seems to be free from the TD in the endemic region even while the local tall—East Coast Tall—is already severely infected. Inclusion of dwarfing genes in resistance breeding programmes (as in LYD and RWD diseases) has drawn attention. The development of disease diagnostic techniques and rapid screening of genetic material with reliable results are likely to augment disease management measures.

PART – B: FUNGAL DISEASES

Fungi constitute another important group as pathogens of coconut. A wide spectrum of species of fungi attack different parts of the palm crop leading to diverse kinds of symptoms and various levels of seriousness. The economic importance of such diseases may marginally vary from country to country and, in fact, certain diseases are endemic to a specific locality/ country/region. As such, leaf rot, bud rot, basal stem rot, stem bleeding, gray leaf spot and lethal bole rot may be designated as the major or main diseases of coconut; various types of leaf spots, petiole rots, fruit rots, etc. besides post-harvest problems, and diseases of uncertain etiology are existing in diverse conditions. The list and distribution of various fungal diseases are presented in Table 2.

LEAF ROT DISEASE (LRD)

Background

LRD is widely prevalent in the southern districts of Kerala State, where root wilt disease (RWD) is endemic. The disease probably existed in the erstwhile princely states of Travancore and Cochin since the 1880s (Varghese, 1934; Menon and Nair, 1951) and seemed to have been considered as a part of RWD. Scientific investigations of LRD were initiated in the early 20th century, almost in parallel with the research on RWD. LRD generally appears on palms already affected by RWD and the palms having latent infection of RWD might be also infected by LRD (Radha and Lal, 1968). LRD incidence can be on palms of all ages and the disease flourished in palms below the age of 25 years. In recent years, severe incidence of the disease in young palms, seedling has also been recorded. Destruction of photosynthetic area due to LRD is quite obvious and RWD affected palms draw very clear attention with the

TABLE 2

Fungal Diseases of Coconut Distributed in Different Parts of the World

No.	Name of Disease	Causal Organism	Geographical Distribution
1.	Leaf rot	Fungal complex (Main pathogens: *Colletotrichum gloeosporioides* and *Exserohilum rostratum*)	India (Kerala State)
2.	Bud rot	*Phytophthora palmivora* (*P. katsurae* also implicated)	India, Indonesia Jamaica, Srilanka Phillipines, Ivory Coast, etc.
3.	Basal stem rot	*Ganoderma lucidum* *G. applanatum* *G. boninense*	India, Srilanka. West Indies, Seychelles, Guam, etc.
4.	Stem bleeding	*Thielabiopsis paradoxa*	Srilanka, India, Indonesia Malaysia, Phillipines, Papua New Guinea, Fiji, Trinidad, Ghana, etc.
5.	Grey leaf spot	*Pstalotiopsis palmarum*	Cuba, India, British Guyana, Malaysia, Srilanka, Nigeria, Trinidad, Brazil, New Hebrides, etc.
6.	Lethal bole rot	*Marasmiellus cocophilus* *M. inoderma*	Kenya, Tanzania, Malaysia, Madagascar, Solomon Islands
7.	Leaf spots	Various species implicated: *Alternaria alternata* *A. tenuissima*	India
		Curvularia maculans *Curvularia* sp.	Malaysia, India
		Exosporium durum *E. palmivorum*	Phillipines, India
		Epicoccum cocos	Samoa, Papua New Guinea, Phillipines, Indonesia, etc.
		Thielaviopsis paradoxa	Jamaica, Mauritius, Phillipines, India

Continued ...

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No.	Name of Disease	Causal Organism	Geographical Distribution
		Cercospora apii	India (Andamans)
		Bipolaris incurvata	Papua New Guinea,Brazil, India (Andamans)
		Stemphylium sp.	Oman
		Capitorostrum cocoes	Papua New Guinea
		Mycosphaerella palmicola	
8.	Leaf blights	*Lasiodiplodia theobromae*	Brazil, India
		Catacauma (Phyllachora) torrendiella	Brazil
		Cocostroma palmicolla	
		Botryosphaeria cocogena	
		Pestalosphaeria elaeidis	India
9.	Sooty mould	*Capnodium footi*	Philippines,Malaysia
		Aithaloderma setosum	Solomon Islands, etc
10.	Spear rot/Top rot/ Shoot rot/Tip break	*Fusarium* sp.	Malaysia, Brazil, Myanmar Papua New Guinea, etc.
		F. solani	India
		F. moniliformae var *intermedium*	Malaysia, Srilanka, Papua
11.	White thread blight	*Corticium penicillatum*	New Guinea, Fiji, New Hybrides, etc
12.	Petiole rot	*Anthostomella cylindrospora*	Srilanka, Malaysia, Papua New Guinea, New Caledonia,
		A. fusispora	India, etc.
		Leiosphaerella longiispora	Papua New Guinea, India
		Phytophthora parasitica	Jamaica
		P. palmivora	India
		P. paradoxa	
		Phytophthora sp.	

Continued ...

... *Continued*

No.	Name of Disease	Causal Organism	Geographical Distribution
		Cylindrocladium pteridis	Brazil
13.	Fruit rot	*P. palmivora*	India, Malaysia, Colombia, Indonesia
		P. katsurae	Hawai
		Botryosphaeria rhodina	India
		Colletotrichum gloeosporioides	Brazil, India
		Lasiodiplodia theobromae	Indonesia, Vietnam, India, etc.
		Ceratocystis paradoxa	Sau Paulo
14.	Uncertain problems: Immature nut fall/button shedding	Biotic, abiotic, environmental,. physiological stresses etc	General
	Pencil point	Abiotic factors	India, Srilanka, Myanmar, New Guinea
15.	Post-harvest problems	Various species of fungi attack seed coconuts and copra/ shell of coconuts.	General

onset of LRD. LRD incidence brings about rapid deterioration in the condition of palms and an average of 70 per cent yield loss is felt (Radha *et al*, 1962). Srinivasan (1991) showed general confinement of LRD to RWD affected palms and on an average of 65 per cent RWD affected palms superinfected with LRD; incidence also related to the severity of RWD. A strong relationship of LRD with RWD incidence has been seen and there exists a correlation between them. The susceptibility of young palms with RWD symptoms to ready attack of LRD, irrespective of soil types, was also spelt out. The palms weakened by RWD pathogen (Phytoplasma) might become susceptible to LRD. The occurrence of LRD with RWD is a distinct Phytoplasma-fungal disease complex. The prevalence of LRD restricted to the RWD endemic region points more towards its inter-relationship with RWD. Hence LRD forms a part of RWD.

Symptoms

The earliest mention of the symptoms of LRD was by Butler (1908). Patel (1938) described a shoot rot disease. Subsequently, several reports (for example, Varghese, 1934;

Menon and Nair, 1951; Radha and Lal, 1968: Srinivasan and Gunasekaran, 1992) have described the symptoms

The disease initially appears as minute, water-soaked lesions on the emerging spindle with different shapes and colours, and occasionally on young tender leaves. These lesions enlarge, coalesce leading to extensive rotting especially under high rainfall, r. h. and low maximum temperature. Mould growth is commonly seen on the surface of the affected leaflets (Srinivasan *et al*, 1995). The rotting may extend into the interior of the spindle and the infected spindle gradually decays attracting ants, earwigs, maggots, etc. The tips of rotten spindle leaflets sometimes stick together even while the base of the leaflet is open. The rotten portions turn black and fall off when dried. Tips of leaflets and midribs often become shriveled and blackish. The progress of rotting is slowed with maturity of leaflets. The affected dried tissues break off and leave the basal portions of leaflets in certain palms to remain without symptoms, giving a fan-like appearance of the leaves in the crown. Successive infection of newly emerging spindle does occur, rotting may progress to varying degrees, resulting in expression of the LRD symptoms even in all the leaves of the crown. The quantitative pattern of LRD symptoms in different whorls of infected palms was analysed; 24-43 per cent of palms in different soil types expressed symptoms in entire crown implying the successive infections with linear progression of the disease. The symptoms of the disease on petiole, mid-rib-midveins of leaflets, both in natural conditions and upon artificial inoculations, has also been recently reported (Srinivasan and Gunasekaran, 1992, 1998a). The younger leaves especially the tender unopened spindle leaf play a critical role in LRD incidence. Chronic infection of the disease makes the palm declining steadily and might even succumb. Grading system has been evolved for indexing the disease (Radha *et al*, 1961; Mathai, 1980; Srinivasan and Gunasekaran, 1996a).

Etiology and Epidemiology

The earliest record of isolation of a (*Penicillium*-like) fungus from LRD affected palms was by McRae (1916). The disease was believed to be primarily caused by fungus (Varghese, 1934). Subsequent studies revealed association of *Helminthosporium (Bipolaris), Gloeosporium, Gliocladium, Pestallozia (Pestalotiopsis)* and *Fusarium* with LRD. Rotting of tissues by various fungi (except *Fusarium*) was observed and *Helminthosporium* was considered as the most virulent (Menon and Nair, 1951). The fungus *Helminthosporium halodes* (=*B. halodes*) was given emphasis of its pathogenic role in LRD. Spores of the fungus are airborne. It developed maximum spots on most tender inoculated leaves (Lily, 1963, 1981). Although the fungus was reported to be isolated from coconut leaves in Kasargod, North Kerala (Radha and Lal, 1968) there seem to be no further evidences on such a line. Certain nematodes such as *Panagrolaimus* sp. (Nadakal, 1965), *Aphelenchoides aligarhiensis*, *Rhabditis* sp. and *Panagrolaimus rigidus* (CPCRI, 1997) especially from rotten tissues

were reported. Nematodes seem to have no role in the disease incidence. It may be noted that such decaying tissues (with foul smell) may attract various scavengers.

Although fungal etiology of LRD was evident, still the need for detailed study of the disease etiology was felt. Isolations made from infected spindles revealed the association of a number of fungal species and the disease was attributed to fungal complex (Srinivasan and Gunasekaran, 1993). The fungi were identified as *Colletotrichum gloeosporioides*, *Exserohilum rostratum*, *Gliocladium vermoeseni* (Srinivasan and Gunasekaran, 1994a). *Cylindrocladium scoparium* (Srinivasan and Gunasekaran, 1995a), *Fusarium solani*, *Fusarium moniliforme* var. *intermedium* (Srinivasan and Gunasekaran, 1998b), *Thielaviopsis paradoxa*, *Rhizoctonia solani*, *Mortierella elongata*, *Curvularia* sp., *Acremonium* sp. *Thielavia microspora*, *Thielavia terricola* and *Chaetomium brasiliense* (Srinivasan and Gunasekaran, 1994b). Except the last four species, the others were pathogenic. All the fungi have been appropriately documented. In diseases spindle tissues profuse mycelial growth and spore masses of several among these species were found and *P. palmarum* seen only in older leaves (Srinivasan *et. al*, 1995).

Isolations from spindles with different grades of LRD also brought out the predominant species of fungi. Among various species *Colletotrichum gloeosporioides* was more frequently isolated under various grades (Srinivasan and Gunasekaran, 1996b). From several attempts of *in vitro* isolations, presence of fungi singly or in combinations in individual palms was noticed. Association of more than one fungus with greater number of diseased palms further contended to the fungal complex nature of LRD. The behaviour of predominant fungi, under *in vitro* interactions, was associative than antagonistic, bearing etiological significance of the fungal complex (Srinivasan and Gunasekaran, 1995b). LRD fungi induced only restricted lesions in healthy plants (free from RWD) whereas in RWD infected plants severe rotting was discerned (Srinivasan and Gunasekaran, 1996c). Based on the frequency of occurrence, seasonal relationship, pathogenicity, etc. *Colletotrichum gloeosporioidies* and *E. rostratum* were designated as the main pathogens of LRD. The disease symptoms were reproduced in field planted seedlings with symptoms of RWD by artificial inoculation of the pathogens singly and in combination (Srinivasan and Gunasekaran, 1998a). A number of diseases of coconut palm recorded in other countries cause symptoms like leaf blight/leaf rot which may be comparable to a certain extent with LRD (Ohler, 1984; Nambiar, 1994).

Although so far no correlation between weather factors and incidence of RWD has been found, interestingly, the incidence, severity and fungal species composition, etc. of LRD are influenced by weather. The most severe incidence, of LRD is observed during monsoons with high atmospheric r. h. and low temperature. Free water, leaf wetness (rain/dew), compactness of spindle foliage, etc. conditions in dry months may also be favourable (Menon and Nair, 1951; Radha *et al*, 1961; Radha and Lal, 1968; Mathai, 1980).

Srinivasan and Gunasekaran (1996d) brought out the population dynamics of LRD pathogens in relation to weather variables: *Colletotrichum gloeosporioides* occurred

nuts while on the palm, colonised the husk and infected the embryo and invaded the cavity. Similarly *Marasmiellus semiustus* is an important seed-borne pathogen in Malaysia. *Marasmiellus palmivorus* outbreak was recorded in Malaysia on all cultivated varieties; 20-30 per cent incidence noticed on most germination beds in widely separated localities (Tey and Chan, 1980). Not much other details of disease epidemiology are known (Ohler, 1984; Nambiar and Rawather, 1993).

Control/Management

Strict quarantine measures are necessary to prevent transportation of infected seeds and seedlings to healthy areas and countries. The other measures suggested are dipping seed nuts after partially (top) husking, in 250 ppm phenyl mercury acetate for 15 minutes for control of pre-emergence shoot rot (Jackson and Firman, 1982). In Western Samoa, Niu'ati variety and local tall were thought to be resistant to *Marasmiellus inoderma*.

OTHER FUNGAL DISEASES

LEAF SPOT DISEASES

A number of fungi affects coconut leaves leading to leaf spot types of symptoms. Although these diseases are generally considered as minor diseases, at times such diseases may also pose serious problems under specific circumstances.

***Alternaria* sp.**

Alternaria alternata (Fr.) Keissler develops black, round or oval spots and such spots coalesce, especially in dry conditions (summer) into large patches. This disease is usually seen on up to 3-year-old-seedlings and has been reported front Andhra Pradesh (Rao and Subramaniam, 1975) and also Kerala. Another species recorded from affected leaves was *Alternaria tenuissima* (Prasad *et al*, 1985).

***Curvularia* sp.**

Curvularia maculans develop small, circular, yellow spots in the nursery seedlings (Chan, 1974) as reported from Malaysia. These spots turn brown and gradually dry with a sunken impression. Tey and Chan (1980) observed that when seedlings grow under shade the disease spreads rapidly due to high humidity. Poor nutrition and water stress were also considered as predisposing factors. Malayan Tall is resistant to the disease while Malayan Yellow Dwarf is highly susceptible. Chan (1974) reported promising control of the disease by spraying with Captan, Actidione and Difoltan. *Curvularia* sp. was recorded from India

also, in context of root (wilt)/leaf rot disease (Radha and Menon, 1954; Srinivasan and Gunasekharan, 1994b) and in seedlings (Mishra and Singh, 1987).

***Exosporium* sp.**

Exosporium durum and *Exosporium palmivorum* are the two species reported. The disease produces black tubercular or wart-like fructifications on the surface of the leaves. The disease was not common and occurs only in neglected gardens and reported in the Philippines and India (Ohler, 1984).

Epicoccum cocos

It causes oval leaf spots and is reported from several countries. These spots are dark tan in colour and spores occur in black masses. Infection often occurs through insect induced injuries.

Thielaviopsis paradoxa

This fungus was reported to cause bitten leaf disease in Jamaica and Mauritius (Bain, 1940). A large wedge-shaped spot with broad dark margins running across the folded segments of the young leaves appear. Later when leaves open out, the midribs of the leaflets break at the diseased places, the terminal parts wither/torn away and the crown appears ragged (Ohler, 1984). Almost a similar condition (leaf spot) was reported from the Philippines (Protacio, 1965). The disease is controlled by cutting away the infected shoot below the spotted area followed by fungicide application. In India, *Thielaviopsis paradoxa* was found pathogenically associated with leaf rot, a fungal complex disease (Srinivasan and Gunasekaran, 1994b) which has already been dealt with earlier. The fungus is the causal agent of stem bleeding disease also.

Cercospora apii

Reported to develop leaf spots on seedlings in South Andamans (Rao and Renukumar, 1989).

Bipolaris incurvata

It was recorded in the Andamans (Rao, 1988b). Small yellow spots appear which enlarge and the lesion dries out. As a result, growth of seedlings is retarded. The disease was earlier reported form Papua New Guinea also.

Periconia saraswatipurensis

Purplish brown, circular-elongated spots appear on leaves with fructifications below the epidermis (Gupta and Asha, 1971).

Botryosphaeria rhodina*, *Cladosporium cladosporioides* and *Simatosporium falcatum

Associated with blighted leaves of drought weakened plants (Sulladmath and Ponnappa, 1979).

Other Species

Various other species implicated in leaf spots include *Mycosphaerella palmicola*, *Capitorostrum cocoes*, *Periconiella cocoes*, *Pseudoepicoccum cocoes* in Papua New Guinea, Australia, India, Indonesia (Hyde and Philemon, 1991; Hyde and Frohlich, 1995) and *Stemphylium* sp. in Oman (Hammouda, 1991).

LEAF BLIGHTS

Lower leaves become brown and die prematurely. Leaf browning spreads from the tip to the base. It is caused by *Lasiodiplodia theobromae* and is reported from Brazil (Ram, 1989). In the dry season the lesion size is more than in the wet season (Warwick *et al*, 1993b); the hybrid PB 141 was more tolerant than PB 231 and Brazilian Tall (Warwick *et al*, 1991). In India also, similar symptoms were observed and *Lasiodiplodia theobromae* was isolated from infected leaves (Srinivasan, unpublished). In Brazil, there were two other foliar diseases reported (Warwick *et al*., 1993a): Small verrucosis (*Lixa pequena*)- caused by *Catacauma torrendiella* (*Phyllachora torrendiella* (Subileau *et al*, 1993). The leaflets became necrotic and the whole leaf collapses. Small black, charcoal-like fruiting bodies (stroma) formed in the early stage on the leaflets and midrib; large verrucosis (*Lixa grande*)— the stroma are brownish, larger, more superficial and is caused by *Cocostroma palmicola*. In Brazil, *Botryosphaeria cocogena* has also been implicated in leaf blight (Subileau *et al*, 1994; Warwick *et al*, 1994).

SOOTY MOULD

It is caused by *Capnodium footi* or *Aithaloderma setosum*. A large portion of the coconut leaves is covered with a black layer of fungus-growth which develops on sweet exudation of some insects. Spraying of an emulsion mixture of soap (225 g), Kerosene (9 L) and water (4.5 L) diluted to 1:9 with water is reported to control the disease (Ohler, 1984).

SPEAR ROT

The disease, termed as shoot rot, was reported from India and *Fusarium* sp. was implicated (Sundararaman, 1929); McRae (1929) observed *Gloeosporium* sp. The disease appeared in weakened palms (Patel, 1938). Fusarial top rots have been reported in several countries. Tey and Chan (1980) reported spear rot from Malaysia in nursery seedlings. On

the spear or the neighbouring new leaves dark purplish or brownish stripes appear that rapidly extend downwards without reaching the bud. The base of the spear decays and the spear can be pulled out. Pathogenically tests showed *Fusarium* sp. as the cause of the disease. Sanitary measures, avoiding over-crowding of seedlings and application of a fungicide are suggested for effective control. Disease syndromes akin to Malaysian spear rot were observed in the context of leaf rot disease in India and two species of *Fusarium* (*Fusarium solani* and *Fusarium moniliforme* var. *intermedium*) were implicated (Srinivasan and Gunasekaran, 1998b). Progressive chlorosis and crown rot was recorded in North Kerala and various fungi isolated (Govindan *et al*, 1996).

WHITE THREAD BLIGHT

The causal agent is *Corticium penicillatum* and the disease is recorded from Papua New Guinea, Sri Lanka, Fiji, etc. It usually occurs on older leaves. Large circular necrotic patches appear on the upper surface. On the undersurface of the leaves, white fungal threads appear along the midrib that spreads towards the tip of the leaflets. The leaf tissue is killed by the fungus. In Malaysia a similar disease (leaf blotch) by *Corticium* sp. was reported (Tey and Chan, 1980). Avoiding over-crowing of plants, sanitation of affected leaves and spraying Bordeaux mixture are suggested for the disease management.

PETIOLE ROT

The disease has been recorded in various countries (Table 2). In India, Sulladmath and Ponnappa (1978) reported it from Karnataka. Chocolate brown or dark irregularly circular to elongated lesions appear on the adaxial surface of leaf petiole. These lesions coalesce, involve large areas and spread to the entire petiole. Irregularly raised patches, blisters may also appear. The infected petioles break at the points of lesions due to weakening of the tissues and the broken fronds hang down. The disease was reported to be caused by two species of *Anthostomella* [*Anthostomella cylindrospora* (Booth and Shaw) and *Anthostomella fusispora* (Booth and Shaw)]. In infected petioles black stroma of the fungus is produced below the surface. *Leiosphaerella longispora* in collapsed petiole due to drought in Papua New Guinea (Sivanesan *et al*, 1976) and India (Kamanna and Ponnappa, 1993); *Phytophthora parasitica* from lesions on the petiole with leaf stalk rot in Jamaica (Ohler, 1984) and India (Kurian, 1992); *Phytophthora palmivora*, *T. paradoxa* from affected petioles in Kerala and Karnataka (India) (Nambiar and Rawther, (1993); *Cylindrocladium pteridis* in Brazil (Ponte and Silveria-Filho, 1997) were also implicated. Sanitation of affected petioles and fungicidal spray are useful in disease control.

FRUIT ROT

Coconut fruit (a drupe) is also inflicted by fungi *in situ* and/or upon development. Syndromes such as fruit rot, nut fall, nut rot, nut spot etc. have been documented widely. The

fruit rot, also called as 'Mahali' (nut fall) was reported from Kerala by Sundararaman and Ramakrishnan (1924). It has since been reported from other countries as well. Decaying of immature nuts, especially during the monsoon rainy season, and their fall is the distinct feature of the disease. In the early stage water-soaked lesions appear at the stalk end of the nuts, the lesions turn brown and become sunken. The tissue decay extends into the husk and even up to the endosperm cavity if the shell is yet to be hardened. The fruit rot is caused by *Phytophthora palmivora* (the pathogen of bud rot). The pathogen develops lesion inversely proportional to the age of the nuts (Bennett *et al*, 1986). High humidity during the wet season might be an important predisposing factor. Integrated measures of phytosanitation, regular prophylactic pre-monsoon and post-monsoon spraying with 1 per cent Bordeaux mixture and agronomic practices help effective disease prevention. *Phytophthora katsucae* was associated in Hawaii (Uchida *et al*, 1992). The hybrid PB 121 had an excellent tolerance level to *Phytophthora katsucae* (Bordeaux *et al*, 1992).

Other species of fungi are also implicated in the fruit disease. *Colletotrichum* sp. causes nut splitting. *Colletotrichum gloeosporioides* caused fruit rot (Anthracnosis) in Brazil (Almeida Jr. and Aquino, 1978) wherein harvested fruits are mainly affected. Dark brown-blackish necrotic lesions with black, undulated margins appear. These lesions enlarge, coalesce and may cover a large part of the entire fruit. Later fruit desiccation, deformation and longitudinal fissures are observed. In India, *Botryosphaeria rhodina* was also reported to cause immature nut fall (Sulladmath and Shantappa, 1979). *Lasiodiplodia theobromae* was reported to cause nut rot in Indonesia, Vietnam, nut lesion in Samoa and ring disease in immature nuts in Papua New Guinea. Gunasekaran and Srinivasan (1998) observed the involvement of *Lasiodiplodia theobromae* also in fruit rot in Kerala State, India and the disease seemed to be widespread, and more during dry weather conditions; presence of *Colletotrichum gloeosporioides* also in the diseased nuts was recorded by these authors. In Brazil, *Ceratocystis paradoxa* developed fruit rot (Camargo and Gimenes-Fernandes, 1997).

PROBLEMS OF UNCERTAIN ETIOLOGY

Though fungal etiology of several diseases of coconut are satisfactorily established, certain problems for which the precise cause(s) is probably either still eluding or somewhat complicated.

Button Shedding and Immature Nut Fall

In nature, coconut palm is endowed with the mechanism of regulating its bearing capacity and the tree exhaustion is prevented by events, as, for example, non-fertilization of a substantial number of female flowers which ultimately drop. However, there are several other factors that seem to play a role in shedding of buttons and/or immature nuts affecting the average yield of palms. Numerous reports have appeared on the subject. This group of

maladies may be termed as abnormal nut fall (Child, 1974). Intense button shedding has been reported in dry (summer) period as well as during wet (monsoon) season. A number of factors are considered to be involved, singly and/or in combination, in diverse ecological conditions leading to button shedding/immature nut fall. Biotic agents (fungi, insects, nematodes, etc.), unfavourable weather, edaphic factors, nutritional imbalances, physiological stress/disturbances, consequences of other diseases are some of the probable causes of this kind of problem. *Phytophthora palmivora*, *Colletotrichum* sp. and *Lasiodiplodia theobromae* may be cited as some of the examples of fungal pathogens for nut fall. *Cochliobolus hawaiiensis* has been associated with button shedding/premature nut fall in India (Karthikeyan and Bhaskaran, 1996). A variety of insects such as coreid bug, scale insects, mealy bugs, etc. are commonly associated in coconut gardens and these insects may directly or indirectly (favouring pathogens) cause premature nut fall. Rodents also potentially damage nuts leading to their drop. Mineral deficiency (for example, boron), prolonged drought (abscission), water logging (anaerobic condition), incidence of various infectious diseases and other factors are found associated with button shedding/immature nut fall (Menon and Pandallai, 1958; Ohler, 1984; Nambiar, 1994). Hence, identification of the right factor(s) is of paramount importance in seeking remedial measures. The cause of immature nut fall at one locality might be quite different from others. Control measures should be therefore specific and logical, thereby reducing the nut fall or prevention it.

Pencil Point Disease

Reduced vigour and stunted growth are the principal symptoms of this malady. Leaves become chlorotic, in succession from lower fronds to upwards. Leaf size and number and spathes are reduced. Ultimately the affected palms fail to produce nuts. In fact, the girth of the stem is gradually reduced and the tip presenting itself as a 'pencil point' further growth is arrested. Although this problem is reported from different countries, no biotic agent could be so far associated with this malady. In Tamil Nadu, a higher incidence was reported (Jagannathan and Ramaswami, 1979); there it is called as tapering stem wilt (Bhaskaran *et al*, 1979). Poor drainage, waterlogging, hard subsoil pan, senility, nutrient deficiency, etc. were opined as the possible factors of the malady (Menon and Pandalai, 1958; Nambiar, 1994). Location specific understanding of the factor(s), better management practices, provision of adequate drainage, supplementation of micronutrients with usual fertilizers are some of the suggested measures for management of this malady.

POST-HARVEST PROBLEMS

The fruits of the coconut, after harvest, either for seed purpose or for human consumption also undergo biotic infestations. *Leiosphaerella longispora* has been found to infect seed coconuts and carried via imports (Raju and Reedy, 1984). Several other fungi (fruit rot pathogens) like *Lasiodiplodia theobromae* may also be carried through harvested

nuts and may pose problems in storage, nurseries, copra, etc. *Drechslera hawaiiensis* was reported to enter the fruit even while attached to the bunch itself; later the fungus attacks the copra (Sharma *et al*, 1985). Several fungi attack the copra leading to its spoilage or poor quality. The important fungi (Nambiar, 1994) usually attacking copra are *Aspergillus niger*, *Aspergillus glaucus*, *Aspergillus flavus*, *Aspergillus fumigatus*, *Aspergillus ochraceous*, *Aspergillus* sp., *Penicillium citrinum*, *Penicillium frequentans*, *Lasiodiplodia theobromae*, *Rhizopus oryzae*, *Ceratostomella adiposum*, *Drechslera hawaiiensis*, *Thielaviopsis paradoxa*, *Cephalosporium* sp., *Microsporium* sp, *Botrytis* sp. and *Fusarium* sp.

High moisture condition is a predisposing factor for fungal invasion of copra. *Aspergillus* sp. rapidly penetrates into the copra under such condition and develops discolouration of meat. Various species of fungi enhance the activity of cellulolytic enzyme in the copra. Contamination with aflatoxin B1 and ochratoxin A (Zohri and Saber, 1993) in fungal infected coconut and increased iodine, saponification and free fatty acids in oil due to contaminated copra (Sundaravadivelu and Palaniswami, 1993) were reported. In the monsoon period development of fungal and bacterial population in copra is most common (Susamma *et al*, 1980). Bacterial incidence (*Bacillus* sp.) has also been noticed. *Serratia rubidaea* was isolated from spoiled coconuts in Ivory Coast (Bollet *et al*, 1989). In view of the seriousness of post-harvest problems, infected seed-nuts should be culled out during harvest/storage; rapid drying of copra and proper storage are necessary to minimize losses. The copra (with 5 per cent moisture) kept in polythene sheet lined gunny bags was reportedly moderately free from infestation (Susamma and Menon, 1983).

PART-C: DISEASES CAUSED BY OTHER AGENTS

VIRUS DISEASE

The coconut foliar decay or Vanuatu wilt, a lethal disease in Vanuatu, is so far the only convincing example of a virus disease of coconut and it is caused by a single stranded DNA virus (CFDV-DNA) (Randles *et al*, 1986). Following the introduction of coconut germplasm into New Hebrides Islands in 1962, a disease with wilt symptoms developed on the Red Dwarf and other varieties. Calvez *et al* (1980) described the symptoms of the disease. Yellowing of the leaflets at the base of a middle leaf appear that spreads towards the tip and other leaves. The leaf turns brown from the tip downwards, the stalk breaks, all the middle leaves may hang down, the bottom leaves may be still green for sometime. Later the lower leaves turn yellow, then become brown and dry. Only the young leaves remain for long with slow growth that also finally turn yellow and dry. Small brown spots characteristically appear in the leaflets with early symptoms of yellowing. Inflorescence rot, failure of spathes to open, nut fall, poor development of nuts are visible. No apparent symptoms in the stem but root drying and rotting occur. The symptoms of the disease may slightly vary among the varieties (Ohler, 1984). The virus is a circular DNA and the virus particles are 20-nm

icosahedral (Randles and Hanold, 1989). CFDV-DNA has been found localized within the phloem, but its distribution in the phloem/infected palms is uneven (Randles *et al*, 1992)

The disease is transmitted by a species of insect, *Myndus taffani* (Julia, 1982; Randles *et al*, 1992). Only the "New Hybrides Tall" was tolerant or resistant to the disease. 'Malayan Red Dwarf', 'Rennell Tall" and their hybrids were susceptible. In 'Rennell Tall' variety occasional recovery from the disease was observed (Ohler, 1984).

VIROID DISEASE

A slow decline disease known as cadang-cadang (meaning to 'die' or 'dying') in the Philippines (Randles, 1975; Hanold and Randles, 1991a; Maramorosch, 1993; Velasco, 1997) and a similar disease known as Tinangaja in Guam (Keese *et al*, 1988) is the example of coconut disease due to viroids. The cadang-cadang, also called as yellow mottling disease, was first recorded in the Philippines in 1926. Since then it killed millions of palms in various islands of the country and believed to be a very dangerous disease. The early symptoms of the disease appear as small rough circular bright yellow spots (0.5-1.5 mm) in the lamina of the third or fourth leaf below the spindle and the size of the spots increases as the leaf matures. Consequently the spots become irregular, forming blotches/streaks and produce yellow mottle. With the fusion of spots, they turn orange-yellow. The leaflets are smaller and brittle and the leaves of the diseased palms appear bronze or orange-yellow coloured. The fronds are also smaller and more brittle and they gradually become erect in the crown. The leaves fall off, leaving only a few small, erect, yellowish-green leaves at the apex. Stem tapering may also occur. Flowers are dwarfed. Initially there are more buttons than usual, but they fail to develop. Nut production ceases in 1-4 years. Though no abnormalities are observed in stem, primary and secondary roots are reduced. (Ohler, 1984).

The progress of cadang-cadang is slow and the spread of the disease is also slow. Other palm species such as oil palm (Hanold and Randles, 1991b) are also susceptible to the disease. The disease can be mechanically transmitted and the minimum latent period was thought to be one-a-half years. The natural mode of disease spread is still uncertain, though the spread seems to be from tree to tree. However, Pecumbaba *et al* (1994) gave evidence of pollen and seed transmission of the viroid; the viroid was also detected in seedling embryos and plantlets grown *in vitro* originating from naturally infected palms. Control can be possible by resistance, which remain to be realized. Yield improvement may be possible to a certain extent by cultivation methods and improved agronomic practices. Replanting programmes are considered to be economically feasible.

The Tinangaja disease was also called as yellow mottle decline and was first reported in 1917. The co-identity of cadang-cadang with Tinangaja was established with the discovery of similar viroids in both the cases (Ohler, 1984). Hanold and Randles (1991, 1992) reported

the discovery of viroid-like nucleic acid sequences, in apparently healthy palms and other undergrowth vegetation.

PROTOZOAN DISEASE

Fatal wilt or Hart rot is prevalent in Cuba, Venezuela, Peru, Ecuador and Colombia and Cedros wilt in Trinidad. An unusual flagellate protozoan was detected in the phloem of Hart rot diseased coconut palms in Surinam (Parthasarathy *et al*, 1976) and later in Cedros wilt in Trinidad (Waters, 1978) and Colombia. This organism resembled a group of protozoa, *Phytomonas*, and related to Trypanosomes. This organism was shown to cause diseases in coconut and oil palm (fatal wilt) in several countries in Latin America and the Southern Caribbean, from Brazil to Costa Rica. The coconut protozoan pathogen is presently called as *Phytomonas stahelii* (McGhee and McGhee, 1979).

The early symptoms of the disease is yellowing of leaves, followed by browning from the oldest leaf upwards; necrotic patches appear at the tips of the unopened inflorescence; flowers and nuts become blackened; brown patches appear on the spear; oldest leaves hang along the trunk and all nuts fall. The symptoms progress rapidly and within 3-4 months the affected palms succumb. The *Phytomonas* are transmitted by bugs (Heteroptera) and possibly euphorbs (for example, milk weed, *Asclepsis* sp.) as alternate hosts. The pathogen has been grown in axenic culture and found in the sap of the husk, calyx and coconut water of nuts in the infected palms (Nanden-Amattaram and Prasadi-sewkaransing, 1989).

The flagellates are apparently confined to the phloem sieve-tubes; there they literally plug the food conduction to growing regions bringing about the death of the palm. Fatal wilt affects palms of all ages, spread from tree to tree, appear randomly or in small pockets, follow a jump spread or continuously over the plantation. Surinam Dwarf was the most susceptible variety to the disease and also Ceylonese Dwarf and Malayan Dwarf. Surinam Tall was found to be the resistant variety. Poisoning of diseased trees with systematic insecticides was considered for control of the disease spread. Perhaps integrated measures are necessary for effective disease management (Ohler, 1984; Renard, 1989).

BACTERIAL DISEASE

The incidence of a bacterial stripe disease was reported in the Philippines. The disease appears on nursery seedlings as well as in the field. Yellowish white to bronze coloured translucent stripes appear at the apex of the unfurling leaf and it usually is in the lamina between the veins. These stripes later merge, exhibit pale yellow-bronze colour, necrotic and the leaf blighted. The disease was reproduced even while the identity of the bacterium was undetermined (Ohler, 1984). A bacterial leaf blight was reported from New Britain (Dwyer, 1937). The disease symptoms appear at petiole base or along the length of the leaf. The lesions are with wavy margins, larger and distinct. The edges of lesions are

raised on the leaf undersurface, yellow coloured, become light green and transparent water-soaked areas appear. Whitish bacterial exudate can also be seen. The identity of the bacterium is not known (Ohler, 1984).

A lethal palm blight disease was reported from Florida, USA (Dunaway and Mead, 1979). *Pseudomonas aeruginosa* was isolated from infected tissues. Inoculation tests revealed no pathogenic relation of the bacterium even while. *Pseudomonas aeruginosa* could be retrieved from inoculated tissues. Therefore, the pathological relation of the bacterium with the disease is uncertain (Ohler, 1984).

NEMATODE DISEASE

Many nematode species parasitic to coconut have been reported. Among them the disease, Red ring (previously called as root disease in Trinidad), early described by Stockdale in 1907 and later named as Red ring by Nowell (1919) seems to be the most important. The disease is widespread in Central America, the Caribbean area and South America (Mexico to Brazil). The disease is not known to occur in South and South-East Asia. Yellowing of the leaves is considered as the first symptoms of the disease; however, slight variations are also reported. Generally abnormal browning of leaves from the tips inwards is visible. Commonly the lowest leaves are first affected and successively involve the young leaves. In a later stage, the palms are conspicuously coloured with brown, orange and yellow in all leaves except the central young leaves. The central leaves in due course become greyish and collapse. Premature nut fall may occur. Red and yellow discolouration is seen from the base of the petiole onwards, if cut open. The cross section of the stem shows a red ring (2-4-cm wide) and 3-5 cm from the periphery. The longitudinal section shows extension of the discoloured zone from the base upwards for several feet, up to below the bud. The disease is fatal (Ohler, 1984).

Red ring is caused by *Rhadinaphelenchus cocophilus* Cobb). The nematode is seen in the stem, leaves, roots, etc. of the infected palms. It is transmitted by the black palm weevil (*Rhynchophorus palmarum*) and can also be transmitted through the roots. Effective control of the disease lies in the burning of diseased palms and control of the weevil (Ohler, 1984)

In India, 44 genera of nematode species have been reported on coconut. Among them the burrowing nematode, *Radopholus similis* (cobb) Thorne was thought to be important (Koshy, 1994). It is generally a root parasite, small, elongated, orange lesions are produced on tender roots. These lesions enlarge causing root rooting; the number and mass of tertiary roots are reduced. As a result, the plant growth may be limited. The nematode is a common root parasite of several other crops also. The nematode is controlled in the nursery by application of phorate or Phenamiphos at the rate of 25 kg a.i./ha. during May, September and December. In plantations, integrated measures are suggested: Use of resistant variety.

use of nematode free planting material, application of cow dung. FYM, green manure, etc., application of recommended level of phorate, etc. (Koshy, 1994).

PART – D: NON-PATHOLOGICAL DISORDERS
NATURAL DISORDERS

DROUGHT AND FLOODING

Drought or flooding is a common and recurrent problem in crop productivity in monsoon dependent countries. Although coconut is a sturdy and tolerant crop to adverse conditions, prolonged drought in certain countries or excessive flooding affect the palm crop. In South India, drought conditions had been experienced in the past and coconut was affected.

The common events due to drought are yellowing, drying and drooping of leaves in the outer whorl of the crown. Depending upon the severity of drought, these symptoms are extended to the inner leaves also. Literally, the palm undergoes dehydration and loses capacity from normal female flower production.; as a result, less female flowers are produced; button shrinking, button and nut shedding is observed. Abortion of inflorescence and buckling of bunches also occur. If the drought is acute, wilting and death of palms is common especially in unirrigated plantations. If the planting is shallow, the drought effect is rather fast. The drought may also predispose severe incidence of insects (for example, scales, mealy bugs) and disease (for example, basal stem rot, gray leaf spot, leaf blight) aggravating the problem. Management of drought is important in view of the perennial nature of the crop. Measures of reducing the transpiration loss of water (by removal of 3-5 older leaves), moisture conservation in basins (husk burial), mulching, cover cropping, increasing water holding capacity of soil (organic manure), addition of common salt at the rate of 2kg\palm (hygroscopic effect) and control of insects and diseases are suggested to tide over the drought conditions (Menon and Pandalai, 1958; Rethinam, 1987; Nambiar, 1994).

Prolonged flooding of the young palms can render yellowing of leaves and make the plants look sickly. Excess flooding during monsoon followed by severe dry condition may also lead to a shock in young palms resulting in their death. Adequate drainage provision in coconut gardens is essential to manage the ill effect of flooding.

LIGHTNING EFFECT

Lightning strike of coconut is a common and regular occurrence (natural calamity) in tropical countries during monsoon (Nambiar, 1994). The disorder is also referred to as "False bud rot" of coconut. Less damage (to the crown) to severe effect (death of palm) may occur depending upon the lightning intensity. Splitting of trunk, decapitation, palm on fire or instant death may happen. Ramaiah (1990) described symptoms of lightning effect: deep fissure along the entire length of the trunk, loss of palm vigour, partial scorching of leaves

from outer to inner whorls are the chief events. Hanging of outer leave and petiole split are the other associated events. The spindle and young leaves may not show symptoms in the beginning but in due course they also dry-up giving a 'burnt-up' appearance. The lightning strike is usually observed in a circular fashion among palms, wherein the seriously affected palms die first and others manifest the symptoms slowly. Palms in the periphery of the circle show broken tips of fronds, which hang down. Breaking of fronds is observed in young palms also and rotting of spindle in young seedlings do occur. The lightning affected palms attract insects like rhinoceros beetle and red palm weevil. The partially affected palms show some salmon pink exudation from the trunk and certain insects like *Xyleborus* sp. can invade. Obviously, there is no strong measure to manage the lightning injury. However, sanitation of dead palms should be strictly followed by prompt removal of such palms to avoid pest infestation and fungal colonization (*Ganoderma* sp. colonize commonly).

NUTRITIONAL DISORDERS

Nutritional disorders, especially due to deficiency of certain nutrients, are known to be detrimental to crop plants. In coconut, deficiency due to Boron, Magnesium and Sulphur are fairly known.

Boron Deficiency (Crown Choke)

Boron deficiency was considered as a reason for the disorder called 'Crown Choke'. In India, the malady (previously called as Crown rot) was observed in 1964 in Assam (Chakarborthy *et al,* 1970) and subsequently in other States such as West Bengal, Kerala and Tamil Nadu. Though the disorder is generally observed in young palms, it can occur occasionally in adult palms also. A related disorder called 'Frond choke', 'Bristle top' was already known in other countries such as New Guinea, Indonesia, Ivory Coast, Sri Lanka, Philippines, etc. (Ohler, 1984; Nambiar, 1994).

Early symptom of the disorder is the appearance of crinkled, deformed leaflets in shortened leaves. Decreased number of leaflets with their tip necrosis is also observed. A choked appearance of the frond due to unfurling of leaflets is attained. The tips of the leaves may become hooked and the leaf-stalk emerges 'stick-like' which is necrotic and black with advancement of the disease. While the leaves in the lower whorl remain green and healthy, the inner leaves crowd and the flag leaf fails to unfurl. In the advanced stage of the malady, the meristem tissue is necrotized and the crown dies. But the palm dies after slowly losing its vigour and vitality in 3-4 years. In the stem, usually no symptom is noticeable. Premature nut fall, set back in inflorescence emergence, etc. are the other associated symptoms (Chakaraborthy *et al*, 1970; Rethinam *et al*, 1990; Baranwal *et al*, 1989; Nambiar, 1994).

Deficiency of Boron in soil/leaf has been related to disorder (Cecil and Pillai, 1978). In affected palms the calcium/boron ratio is higher than in healthy ones; the boron level was

low (5.4 ppm) in affected palm leaves as compared to healthy ones (7.4 ppm) (Baranwal *et al*, 1989). Several others also related Boron deficiency to 'Crown choke' like symptoms (Nambiar, 1994). Similar disorders due to genetic malformation, physiological derangements, other soil factors, etc. were also suggested and natural recovery of disorders like 'Frond choke' (Dwyer, 1937) was reported.

Application of borax was considered as a remedial measure for control of 'Crown choke' (Chakraborthy *et al*, 1970). Hence borax at the rate of 50 g/palm after monsoon is recommended and palms in the early stage of the disorder respond relatively better. Surgical correction of 'frond bending' (Chakraborthy *et al*, 1972) and cutting down of hardened leaves have also been reported that facilitates easy emergence of fresh spindles (Dwyer, 1937; Nambiar, 1994).

Magnesium Deficiency (Foliar Yellowing)

Magnsium is one of the major elements for plant nutrition—its presence in the chlorophyll molecule and functional role in numerous metabolisms result in making it a major contributor for yield. Coconut leaf analysis has shown critical Mg level (for a rank 14 leaf on an adult Tall palm) fixed at 0.24 per cent of dry matter.

Magnesium deficiency leads to a loss of chlorophyll in leaves. Hence the marked visual symptom is the yellow discolouration of the leaflets of the oldest frond and the yellowing spread from the leaflet tips towards the rachis of the leaf. The yellowing becomes intense on part of the palms exposed to the sun. The discolouration is less pronounced along the mid-vein of leaflets. With acute deficiency of the mineral, the leaflets tend to lose almost all its pigments. Necrosis is seen on the tip of leaflets which turn reddish brown and translucent patches appear on the yellow leaflets. Magnesium deficiency affects the number of nuts per tree.

Magnesium deficiency can occur in soils with poor exchangeable Mg. In coconut, it seems the deficiency can also occur due to high potassium (K) level. An antagonism between Mg and K exists and excess K can induce Mg deficiency. Such induced Mg deficiency may be more common in Dwarf and DXT hybrid coconuts than in Talls (Ollivier, 1993). With the correction of K level, there would be a correlation between Mg levels and the number of green leaves. Soil groups of Kerala are said to be deficient in Mg and the sandy loam soils of coastal alluvium were more deficient (Prema and Jose, 1994).

Magnesium fertilizer, usually magnesium sulphate at 27 per cent MgO, can effectively correct the deficiency disorder. The remission is reflected with foliage becoming green again and a significant growth of the palms. As K has a depressive effect on Mg contents, care should be taken in drawing up the fertilizer schedules to balance K and Mg and fertilizer application should be optimized.

Sulphur Deficiency (Rubbery Copra)

Rubbery copra disorder of coconut was reported to be due to acute deficiency of S. In Papua New Guinea and Madagascar extensive occurrence of S deficiency was reported (Cecil *et al,* 1991).

Orange yellow colour of young as well as old leaves, weakening of the rachis followed by leaflet necrosis are the major S deficiency symptoms (Ollagnier and Ochs, 1972). With the increase in the age of the leaves, chlorosis and necrosis increases and even the first leaf may be yellowed. The number of leaves and nuts are reduced. The nuts are small with apparently normal kernel which upon drying become soft, flexible, leathery and brown (referred to as rubbery copra) with poor quality. In rubbery copra, oil content is less with high nitrogen, ash and sugar contents, and the oil has high iodine value with low saponification value as compared to the oil from normal copra.

Sulphur deficiency can be corrected by the application of S or S-bearing fertilizers. Though S deficiency does not seem to be an immediate problem for coconuts in the West Coast of Kerala, India, continued application of S-free fertilizers without bulky organic manure could lead to S-deficiency disorder (Cecil and Pillai, 1976). Hence, inclusion of any one of the S-bearing fertilizers in the fertilizer schedule is necessary; as for example (Cecil and Amma, 1991), regular addition of magnesium sulphate (13 per cent S) or Ammophos (15 per cent S) could raise the foliar S to a desired level on a S-deficient sandy soil.

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