Chapter 10

Diseases

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1. Introduction

Among several species of palms, coconut is the most widely cultivated palm from ancient times for its versatile use to human beings. The palm has economic lifespan of more than 50-60 years and has a capacity to grow in highly variable soil conditions while being limited to moderate to high rainfall regions. The palm having a long life span like many other tree crops, needs to withstand the adverse climatic vagaries including the outbreak of biotic and abiotic stresses. The coconut is affected by number of diseases from its germination to harvest, some are lethal and some causes economic loss by reducing the quality/quantity of nut yield. The microbial pathogens namely fungi, bacteria, viruses, viroids and phytoplasma are known to cause diseases in coconut (Table 10.1). About 173 fungi, few species of bacteria, viruses, viroids and phytoplasma have been found to be associated with coconut, however only few diseases are economically important. The causal agents of various diseases formerly regarded as diseases of unknown etiology have been discovered. However, this is not always the case with the vectors that help the spread of the diseases. In general, curative treatments against diseases are unknown or if they are known they are very costly and uneconomic. Susceptibility to diseases may differ widely between different varieties of coconuts. In general, selection and breeding of tolerant species remains the best solution to counter the disease effects, but sometimes this may be a long and difficult path. Plantation sanitation, timely prophylactic control measures and integrated disease management can be important instruments in avoiding or reducing disease attack. The Phytoplasmal diseases for which are yet uncontrollable pose the greatest threat. Not only as a threat to

Table 10.1: Diseases Reported on Coconut

Sl.No.	Disease	Pathogen/Causal Organism	Distribution	References
	Fungal Diseases			
1.	Bud rot disease	*Phytophthora palmivora, P. heveae, P. katsurae, P. nicotianae, Fusarium moniliforme, F. solani, Graphium* sp.	India, Ivory coast, Indonesia, Jamaica, Puerto Rico, Africa, Peninsular Malaysia and the Philippines	E. J. Butler, 1906; Menon and Pandalai, 1958; Quillec *et al.,* 1984; Uchida *et al.,* 1992
2.	Nutfall and Mahali disease	*P. arecae, P. katsurae*	India, Sri Lanka and Ivory coast	Erwin and Ribiero, 1996; Quillec *et al.,* 1984
3.	Basal stem rot	*Ganoderma lucidum, G. applanatum, G. zonatum, G. boninense*	India, Florida, South America, Java, tropical Africa, Australia, Japan, Indonesia, Malaysia, Philippines, Samoa, Sri Lanka and Tasmania	Peries, 1974; Bhaskaran and Ramanathan, 1984; Satyanarayana *et al.,* 1985
4.	Stem bleeding of coconut	*Thielaviopsis paradoxa/ Chalara paradoxa*	Sri lanka, India, Indonesia, Malaysia, Philippines, Fiji, Ghana, Trinidad	Petch, 1906; Sundararaman, 1922; Briton Jones, 1940
5.	Leaf rot	*Exserohilum rostratum/Colletotrichum gloeosporioides/Fusarium solani* and *Fusarium moniliforme*	India	Varghese, 1934; Menon and Pandalai, 1958; Radha and Lal, 1968, Srinivasan and Gunasekaran, 1999
6.	Grey leaf blight	*Pestalotiopsis palmarum*	Guyana, India, Malaysia, New Hebrides, Sri Lanka, Trinidad, Nigeria	Copeland, 1931; Cook, 1971; Holliday, 1980
7.	Leaf blight	*Lasiodiplodia theobromae*	India	Johnson *et al.,* 2014
	Phytoplasmal diseases			
1.	Lethal yellowing	16Sr IV group Phytoplasma	Western Jamaica, Cuba, southern USA (Florida and Texas), southern Mexico	Nutman and Roberts, 1955; Ashburner *et al.,* 1996; Eden-Green, 1997; Harrison *et al.,* 1994, 2002; Myrie *et al.,* 2007
2.	Root wilt/Kerala wilt disease	16Sr XI group Phytoplasma	India	Solomon *et al.,* 1983; Manimekalai *et al.,* 2010
3.	Coconut lethal disease	16Sr IV group Phytoplasma	Tanzania, Kenya, Mozambique	Schuiling *et al.,* 1992; Mpunami *et al.,* 1999

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Sl.No.	Disease	Pathogen/Causal Organism	Distribution	References
4.	Awka disease/Bronze leaf wilt	16Sr XXII group Phytoplasma	Nigeria (West Africa)	Ekpo and Ojomo, 1990; Tymon *et al.*, 1998
5.	Cape St Paul wilt	16Sr IV group Phytoplasma	Ghana	Tymon *et al.*, 1998
6.	Kaïncopé disease	Phyoplasma	Togo (West Africa)	Steiner, 1976
7.	Kribi disease	Phyoplasma	Cameroon (West Africa)	Dollet *et al.*, 1977
8.	Kalimantan wilt, Natuna wilt	16Sr XI group Phyoplasma	Indonesia	Warokka *et al.*, 2006 Jones *et al.*, 1999
9.	Sulawesi yellows	Phyoplasma	Indonesia	Simatupang, 1999
10.	Tatipaka disease	Phyoplasma	India	Rethinam *et al.*, 1989
11.	Weiligama wilt	16Sr XI group Phyoplasma	Sri Lanka	Perera *et al.*, 2010
12.	Coconut yellow decline	16Sr XIV group Phyoplasma	Peninsular Malaysia	Nejat *et al.*, 2009
	Viral diseases			
1.	Coconut foliar decay or Vanuatu wilt	*Coconut Foliar decay virus* (CFDV)	Vanuatu	Calvez *et al.*, 1980; Randles *et al.*, 1986
	Viroid diseases			
1.	Coconut Cadang-cadang disease	*Coconut cadang-cadang viroid* (CCCVd)	Philippines	Randles, 1975
2.	Coconut Tinangaja disease	Coconut tinangaja Viroid (CtiVd)	Guam on Marianas Island	Boccardo, 1985
	Protozoan diseases			
1.	Fatal wilt or Heart Rot or Hartrot	*Phytomonas stahellii*	Central America (Costa Rica, Honduras and Nicaragua), SouthAmerica (Brazil, Colombia, Ecuador, Guyana, Peru, Surinam and Venezuela) and the WestIndies (Grenada, Trinidad and Tobago)	Waters, 1978; Dollet, 1984

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Sl.No.	*Disease*	*Pathogen/Causal Organism*	*Distribution*	*References*
	Other minor diseases			
1.	Lethal bole rot	*Marasmiellus cocophilus*	Kenya and Tanzania	Jackson and Firman, 1982; Jackson and McKenzie, 1988
2.	Anthracnose	*Colletotrichum gloeosporoides*	Brazil, India	Almeida J. J. L. De and Aquino, M. De L. N, 1978
3.	Leaf spots	*Bipolaris incurvata*	Hawaii, Florida, Jamaica, Asia, Australia, Oceania (French Polynesia, Fiji) Philippines, and the Seychelles	Uchida and Aragaki, 1991
4.	Algal leaf spot	*Cephaleuros virescens/ Cephaleuros parasiticus*	Hawaii	Ploetz *et al.,* 1999; Harrison, N., and P. Jones, 2003
5.	Thread Blight	*Pellicularia filamentosa Pellicularia koleroga Corticium penicillatum*	Sri Lanka, Fiji, Papua New Guinea, Samoa and Solomon Islands in Oceania	Kohler *et al.,* 1997

existing plantations but also as a reason for farmers to plant or replant coconuts. A brief review of the important diseases of coconut is summarized in this chapter.

2. Diseases Caused by Fungi or Oomycetes

There are many fungi or oomycetes affecting coconut but only a few are of great importance, although some of the minor diseases cause severe damage in some localities only the most important fungal diseases namely bud rot and nut fall, basal stem rot, stem bleeding, grey leaf blight, *Lasiodiplodia* leaf blight and fruit rot are discussed.

2.1. Bud Rot and Nut Fall

Bud rot is one of the most common diseases of coconut around the world, especially in the tropical humid regions. Usually, only sporadic cases of bud rot occur in plantations, but sometimes the disease may kill a few per cent of the palms each year. Even though it affects the palms of all ages, young palms in low lying and moist situations are more susceptible to the disease. Resurgence of bud rot disease has become common and sometimes even up to 50 per cent of the coconuts planted initially are killed by bud rot (Quillec *et al.*, 1984). In certain humid locations bud rot occurred regularly killing hundreds of trees. In India, though the total per cent of bud rot incidence is less than 1 per cent, but in certain pockets up to 20 per cent of the coconut palms succumb to bud rot depending upon the climatic conditions, soil and nutritional factors and varieties. Increase in incidence of bud rot in India is attributed to non-adoption of scientific cultivation practices by farmers, erratic rain fall, non-adoption of prophylactic measures, shortage of skilled labours coupled with high wages (Sharadraj and Chandramohanan, 2013).

2.1.1. Symptoms

The first visible symptom is withering of the spindle marked by pale colour. The spear leaf or spindle turns brown and bends down. The affected spindle can easily be pulled out as the basal portion of the spindle is completely rotten emitting a foul smell. Later the inner leaves also fall away one by one leaving only outer whorl of matured leaves in the crown (Figure 10.1). Ultimately the palm succumbs to the disease with the death of the growing bud (Menon and Pandalai, 1958; Radha and Joseph, 1974; Quillec and Renard, 1984).

Sometimes the same bud rot pathogen was found to infect coconut fruits and cause fruit rots and immature nut fall. Water-soaked lesions appear on the surface of the nuts especially near the perianth and the lesions turn brown and the nut detaches from the bunch. The fruit rot common during rainy season (Figure 10.2).

2.1.2. Etiology

The genus *Phytophthora* was erected by Anton de Bary in 1876. Butler (1906) described bud rot in palmyra (*Borassus flabellifer* L) and coconut palms and isolated a fungus which he named as *Pythium palmivora*. He placed it in the genus *Phytophthora* in 1919, and reproduced the symptoms by inoculation. In 1923, McRae published the results of successful inoculations of both palmyra and coconut palms. Studies have clearly shown the existence of two dominant *Phytophthora* spp. *viz.*, *P. palmivora*

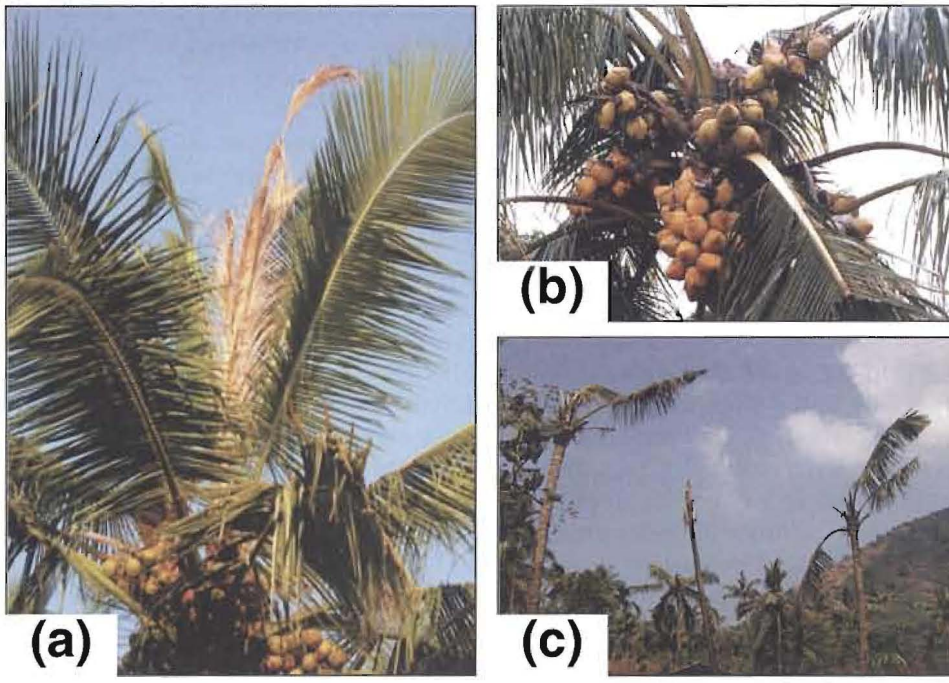

Figure 10.1: Bud Rot.
a) Whithering of spindle leaf, b) Death of growing bud,
c) Severe incidence of bud rot.

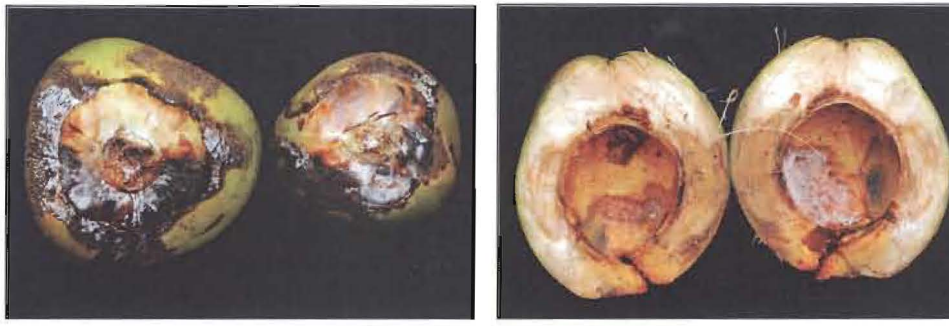

Figure 10.2: Symptoms of Coconut Fruit Rot Caused by *Phytophthora palmivora*.

and *P. katsurae* as incitants of bud rot of coconut (Thomas *et al.*, 1947; Uchida *et al.*, 1992; Chowdappa *et al.*, 2003). Sharadraj (2014) reported that out of 137 isolations made from bud rot affected samples collected from different locations of India, 130 were identified as *P. palmivora*, four isolates were identified as *P. nicotiane*, one each of *P. meadii and P. capsici*. The causal organism of bud rot and premature nut fall of coconuts in Cote d'Ivorie has been reported *as P. katsurae. P. katsurae* is also reported to cause fruit rot and heart rots of coconut in Jamaica (Steer and Coates-Beckford, 1990) and Hawaii (Ooka and Uchida, 1984; Uchida *et al.*, 1992). Veena *et*

al. (1997) reported *P. katsurae* causing bud rot of coconut in Kuttiadi area in Kerala. Joseph and Radha (1975) found that *P. palmivora* causes dry rot in coconut prior to wet rot in later stages which is due to the secondary invaders such as *Fusarium* spp. and bacteria. *P. palmivora* forms oogonia and oospores only when A1 and A2 mating strains are paired (Brasier and Griffin, 1979). Tucker (1931) reported that sporangia of coconut isolates were variable in size with an average length of about 26 to 88 µm and 18 to 41 µm in breadth with an L/B ratio of 1. 7:1. Variablity in sporangial morphology of *P. palmivora* isolates causing bud rot collected from different locations was reported (Figure 10.3). Sporangia of all the 34 isolates of *P. palmivora* were ovoid to ellipsoid in shape with round base and conspicuous papilla. Both single and double papillate sporangia were present in the isolates (Rasmi, 2003). Sharadraj and Chandramohanan (2016) also found that there was a significant inter and intra-specific variability among the *Phytophthora spp* infecting coconut in India. Chowdappa *et al.* (2003) reported identical ITS-RFLP and AFLP pattern among the *Phytophthora palmivora* isolates from cocoa and coconut and observed that AFLP fingerprints are useful for assessing the intra-specific population variation.

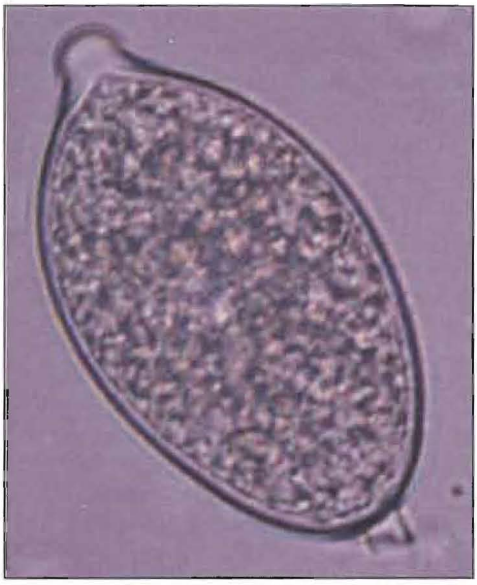

Figure 10.3: Sporangia of *P. palmivora.*

2.1.3. Epidemiology

The disease is generally noticed during both south west and north east monsoon periods when wet weather prevails and younger palms are more vulnerable (Menon and Pandalai, 1958). Though the rainfall in the southern districts of Kerala is well distributed it does not favor the buildup of constant high relative humidity in the plantations. Thus, rainfall aggravates *Phytophthora* infection in coconut and young palms in low lying and moist conditions are more susceptible (Thevenin *et al.*, 1992; Brahmana *et al.*,1992; Mangindaan *et al.*, 1992; Pohe, 1992). High rainfall intensity in certain areas causes low solar radiation and high air humidity. Bud rot disease is favoured by conditions of high humidity such as found in low lying badly drained lands, in plantations with a very dense stand and under conditions of continuous high rainfall (Menon and Pandalai, 1958; Darwis, 1992). Radha and Joseph (1976) observed that temperature range of 20 - 24°C and relative humidity of 98 - 100 per cent were optimum for the development of the bud rot disease. Contiguous occurrence of such "favourable days" determines the development of the disease. Such "favorable days" occurred more frequently in young palms (below 20 years) compared to that of older palms. It was also noticed in the survey that the disease incidence in the hilly areas continued even after the cessation of the North - East

monsoon up to January. However, in the plains the disease incidence was recorded only up to September. This may be due to the misty condition and low temperature that prevail in the hilly areas in these months even after the monsoon season (Rasmi, 2003). Rhinoceros beetle (*Oryctes rhinoceros* L.) infestation was very common in bud rot disease endemic areas. The wound caused by the beetle could be considered as one of the major pre-disposing factors for higher bud rot incidence (Sharadraj and Chandramohanan, 2016). Sharples (1925) reported definite associations of bud rot with injury caused by the attack of *O. rhinoceros* in Malaysia.

2.1.4. Disease Management

Effective management of bud rot can be achieved only if the integrated plant protection measures are adopted at the right time. Reducing the inoculum load in the garden by cut and removal of palms, which are in the advanced stage of bud rot or palms dead due to the disease is one of the very important steps in effective control of the disease. Regular cleaning of the crown and prophylactic spraying of Bordeaux mixture (1 per cent) to the crown just before the onset of monsoon and one more spray after 35-40 days help in reducing the bud rot incidence. Sharadraj and Chandramohanan (2012) advocated pouring 5g of mancozeb dissolved in 300 ml of water or 300 ml of potassium phosphonate (0.5 per cent) to innermost leaf axil and keeping two perforated mancozeb sachets as a prophylactic measure to prevent the bud rot disease based on the field trial conducted in the bud rot endemic area. The affected palm can be cured if the symptoms of bud rot are detected in the early stage. Remove the entire rotten portion of the spindle by cutting with a sharp knife and apply 10 per cent Bordeaux paste to the wound and cover with polythene sheet to prevent entry of rainwater. The protective covering has to be retained till normal shoot emerges. The entire rotten portion removed from the crown should be destroyed by burning (Menon and Pandalai, 1958; Nambiar, 1994).

2.2. Basal Stem Rot

Basal Stem Rot, Ganoderma Wilt, or Thanjavur Wilt, affects coconut palms as well as oil palm and arecanut palm. The disease occurs widespread over the world. It is the most destructive disease in Tamil Nadu, India, where it was first observed after a cyclone in 1952. Often, it occurs in badly managed groves. Losses of up to 31 per cent have been reported (Bhaskaran and Ramanathan, 1984), and complete plantations were destroyed within 7–8 years when control measures were not used (Bhaskaran, 2000). *Ganoderma* spp. has a wide host range attacking a variety of palms and several forest, avenue and fruit trees. According to Naidu *et al.* (1986), hosts belonging to 19 families, 36 genera and 48 species have been reported to be affected by *Ganoderma* spp. Coconut palms in the age group of 10–30 years are easily attacked by the pathogen. The fungus first infects the root system and during the very early stage of infection no external disease symptoms are clearly visible. The disease incidence is positively correlated with mean maximum soil temperatures, and it is not correlated with minimum temperatures, rainfall or relative humidity (Bhaskaran *et al.*, 1989). The disease progresses rapidly in dry areas and more slowly in wet areas. Soil water stress may predispose the palms to infection (Nambiar and Rawther, 1993). Infected palms may die within months in dry areas and survive

another five to six years in areas with higher rainfall (Peries *et al.*, 1975). Infection occurs primarily through root contact.

2.2.1 Symptoms

The characteristic symptoms are outer whorl of the leaves turn yellow initially, later they exhibit light to moderate browning followed by drooping. In the crown, the leaflets wilt. As the disease advances, the remaining leaves droop down in quick succession and the spindle alone remains in palm (Figure 10.4). Appearance of bleeding patches at the base of the stem near the ground level and extensive rotting and discoloration of root system. Normal development of flowers and bunches are arrested. Aggravation of the disease leads to button shedding and also in advanced infection there is a formation of sporocarp (fruiting body) at the base of palm (Vijayan and Natarajan, 1972; Rethinam, 1984; Bhaskaran *et al.*, 1989).

2.2.2. Etiology

Ganoderma applanatum (pers.) Pat., *G. lucidum* (leys) Karst., were isolated only from roots of infected palms and pathogenecity was established by artificial inoculations (Bhaskaran *et al.*, 1989).

2.2.2. Epidemiology

Generally the disease is prevalent in sandy or sandy loam soils in coastal areas where coconut is grown under rainfed conditions and also in neglected plantations. Coconut palms in the age group of 10–30 years are easily attacked by the pathogen (Kandan *et al.*, 2010). Lack of soil moisture during summer months, water logging in rainy seasons favour disease development (Anonymous, 1976; Ramasami *et al.*, 1977; Bhaskaran *et al.*, 1989). The disease incidence was more between March and August. It was positively correlated with the mean maximum temperature and number of bleeding patches and not correlated with minimum temperature, rainfall and relative hummidity (Ramasami *et al.*, 1977; Lewin *et al.*, 1983).

2.2.3. Disease Management

An integrated disease management involving the application Trichoderma enriched neem cake, root feeding and soil drenching with fungicides and intercropping with banana is recommended. Isolation of the affected palm from the healthy ones by digging a trench (2 ft deep and 1 ft wide) around the affected palm (4 ft away from the base of the trunk) help in preventing the spread of infection to nearby palms. Application of neem cake (5 kg) fortified with *Trichoderma harzianum* (CPTD 28) talc formulation (50 g) per palm per year at six monthly intervals helped in reduction in the incidence and recovery of the affected palm. Root feeding of hexaconazole @ 2 per cent (100 ml solution per palm) and soil drenching @ 0.2 per cent (40 L solution per palm) or with 40 L of 1 per cent Bordeaux mixture at quarterly intervals is recommended. In the earlier studies, Bhaskaran *et al.*(1978) reported that irrigation along with FYM application and burying coconut husks in circular trench around the palm and Bordeaux mixture drenching was most effective in reducing the intensity. Burying 500 coconut husks in circular trench around the diseased palms contained the disease (Vijayan and Natarajan, 1975). Benomyl, thiruam and

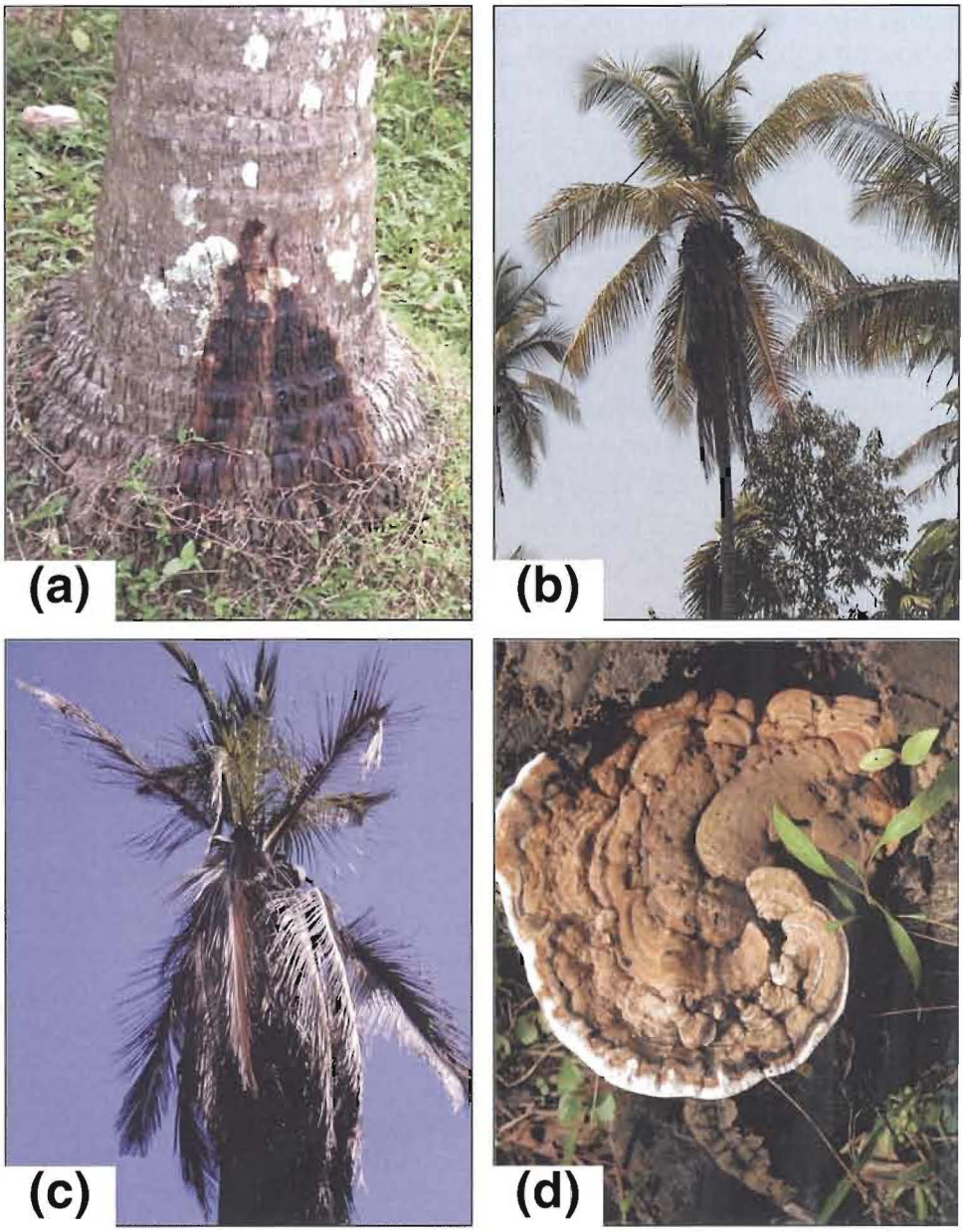

Figure 10.4: Basal Stem Rot
a) Exudation of reddish brown viscous fluid from the basal portions of the trunk wilting of leaflets, b) Yellowing of leaflets, c) Wilting, drying and drooping of leaves in the outer whorls of leaves, d) Fruiting bodies of *Ganoderma.*

captafol were most effective as soil drenches when the soil moisture was 100 per cent. (Koti Reddy and Saraswathi, 1976). Field trials indicated that drenching the

base of palm with captan or carbendazim at 0.3 per cent concentration was effective in preventing the spread of the disease to the neighbouring palms (Sampath Kumar and Nambiar, 1990). Field trial conducted at Palghat (Kerala) by CPCRI, Kasaragod showed that in tridemorph and aureofungin solution treated palms, the disease was less (Anonymous, 1988). Soil application of neem cake 5 kg/palm/year and root feeding of trideomorph (2 per cent) or kitazin (0.3 per cent) was effective in reducing basal stem rot at Aliyar Nagar (Anonymous, 2000). Application of neem cake fortified with *Trichoderma* to diseased palms encouraged the saprophytic soil microflora in coconut basins and was effective in control of Ganoderma wilt (Gunasekaran *et al.*, 1986; Bhaskaran, *et al.*,1988).

2.3. Stem Bleeding

It is a major disease of coconut occurring in almost all coconut growing countries. The disease was first reported from Sri Lanka (Petch, 1906) and later from India (Sundararaman, 1922), and Philippines (Lee, 1922). In recent years the occurrence has been reported from Brazil (Warwick and Passos, 2009) and Hainan, China (Yu *et al.*, 2012). The disease is usually non-lethal but in extreme cases, the palms become barren and die.

2.3.1. Symptoms

The typical symptoms are exudation of reddish brown fluid from growth cracks and the trunk,which becomes black on drying. In the initial stages of infection bleeding symptoms appears only one or two longitudinal cracks at the base and later spreads upward on the stem (Figure 10.5). The longitudinal patches may coalesce to form large patches (Menon and Pandalai, 1958; Radha, 1962; Nambiar and Rawther, 1993).

2.3.2. Etiology

The etiology of the disease was not established for a long time, Lily (1984 a,b) isolated *Phomopsis cocoina* and *Schizophyllum commune* from the stem bleeding affected palms but their pathogenicity was not established. Nambiar *et al.* (1986). established the etiological nature of *Theilaviopsis paradoxa* in the disease through artificial inoculation. Later perithecial stage of the causal agent *Ceratocystis paradoxa* has been isolated from infected trees. *Theilaviopsis paradoxa* produces pale brown to brown hyphae, conidiophores are slender arising laterally from the hyphae and produce cylindrical to oval endoconidia, when mature they are hyaline to pale brown and smooth walled (6-24x2-5.5 µm). Chlamydospores are obovate, thick walled brown coloured and are in chains.

2.3.3. Disease Management

Since wound and the trunks predispose the palms to infection care should be taken not to injure the palms while doing cultural operations. Application of neem cake (5 kg/palm) was found to increase soil microflora including *Trichoderma* population, which are found to inhibit the pathogen *in vitro* (Gowda, 1987) and on detached coconut leaf petiole (Usman, 1988). Indeed they identified *T. viride*, *T. harzianum* and *Aspergillus niger* as potential antagonist to the pathogen. Later,

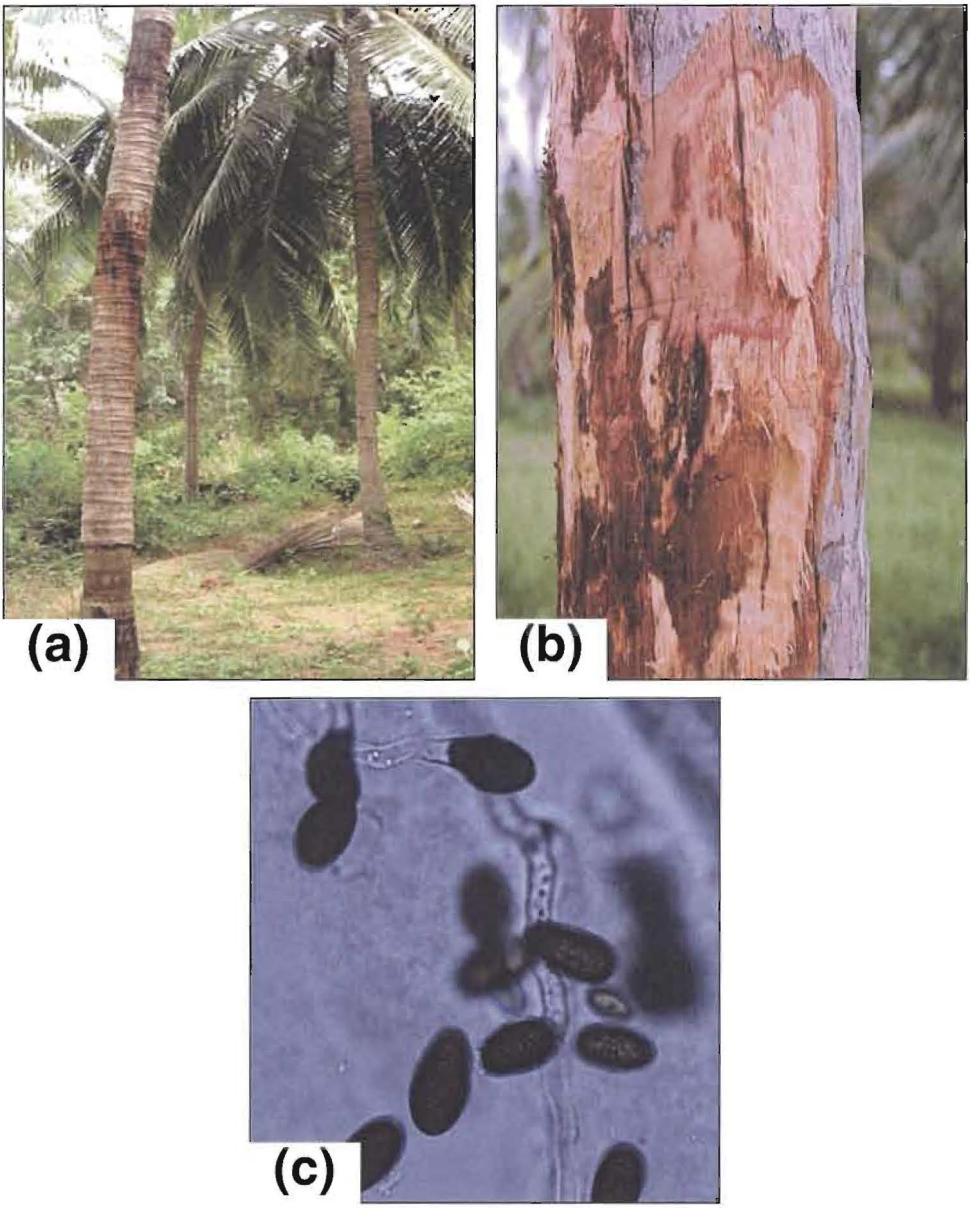

Figure 10.5: Stem Bleeding Disease
a) Exudation of reddish brown fluid from the cracks, b) Tissue decay, c) Conidia of *Thielaviopsis paradoxa.*

Ramanujam (1997) developed integrated management practices for effective management of stem bleeding of coconut involving root feeding (100 ml at quarterly intervals) and wound dressing (50-200 ml) of tridemorph (4 per cent) followed by coaltar sealing (100-300 g) and soil application of *Gliocladium virens* (1 kg), neem cake (5 kg), FYM (50 kg) and NPK fertilizer (500 : 320 : 1200 g/palm/year).

2.4. Leaf Rot

Radha (1961) first coined the name leaf rot for foliar necrosis of coconut found in the root (wilt) tract of Southern Kerala. Since beginning of the century, it is well established that the palms affected by root (wilt) are generally superimposed by leaf rot disease (Sundararamn, 1925; Varghese, 1934; Nagaraj and Menon, 1956; Srinivasan, 1991). The palms weakened by Phytoplasma might result in the breakdown of defense mechanism leading to susceptibility to leaf rot disease

2.4.1. Symptoms

Leaf rot starts as minute, water soaked lesions on the emerging spindle with different shades of color and shade. These lesions enlarge, coalesce freely leading to extensive rotting. The rotted portions dry up, turn black and fall off. Tips of leaflets and midribs often become blackish and shriveled. The inner whorls of leaves are vulnerable to the disease. Continues attack of newly emerging spindle leaves results in the gradual exhibition of similar symptoms in all the leaves in the crown (Srinivasan and Gunasekharan, 1992). Sometimes the decayed leaflets are glued together so that spindle does not open out (Figure 10.6). Though the disease does not kill the palm outright, its slow progress in the crown causes steady decline in the yield. Palms of all ages are susceptible to the infection (Radha and Lal, 1968; Srinivasan and Gunasekharan,1992).

2.4.2. Etiology

Radha and Lal (1968) showed the association of a number of fungi with leaf rot disease. They were identified as *Colletotrichum gloeosporioides* (Penzig) Penzig and sacc. *Exserohilum rostratum* (Drechler) Leonard and Suggs, *Gliocladium Vermoeseni* (Biourge) Thom, *Cylidrocladium scoparium, Fusarium solani, Theilaviopsis paradoxa* (Date) *Rhizoctonia solani, Curvularia* spp. etc. Of these, *Colletotrichum gloeosporioides* (Penzig) Penzig and Sacc, *Exserohilum rostratum* (Drechler) are considered as major pathogens of leaf rot disease based on their frequency of occurrence and pathogenicity (Srinivasan and Gunasekaran, 1998).

2.4.3. Epidemiology

The tender leaf was most susceptible (Lily, 1963). The susceptibility of the seedlings decreased with age. Seedling up to 19 months may get severe infection. The incidence of *C. gloesporoides* was higher in frequency and population during monsoon with a peak during June-July. Its incidence was positively correlated with rainfall and relative humidity and negative correlated with maximum temperature and sunshine hours. Thus *Colletotrichum gleosporoides* was implicated as the principal pathogen of leaf rot during monsoon. Incidence of *Exserohilum rostratum* was less frequent and not well correlated with weather.

2.4.4. Disease Management

A field trial conducted for three years on 20 year old palms revealed that pouring of trideomorph (1 per cent) into leaf axil and spraying of mancozeb 0.3 per cent along with phytosanitary practices reduced the disease intensity (Srinivasan and Gunasekaran, 1998). An integrated management system involving need

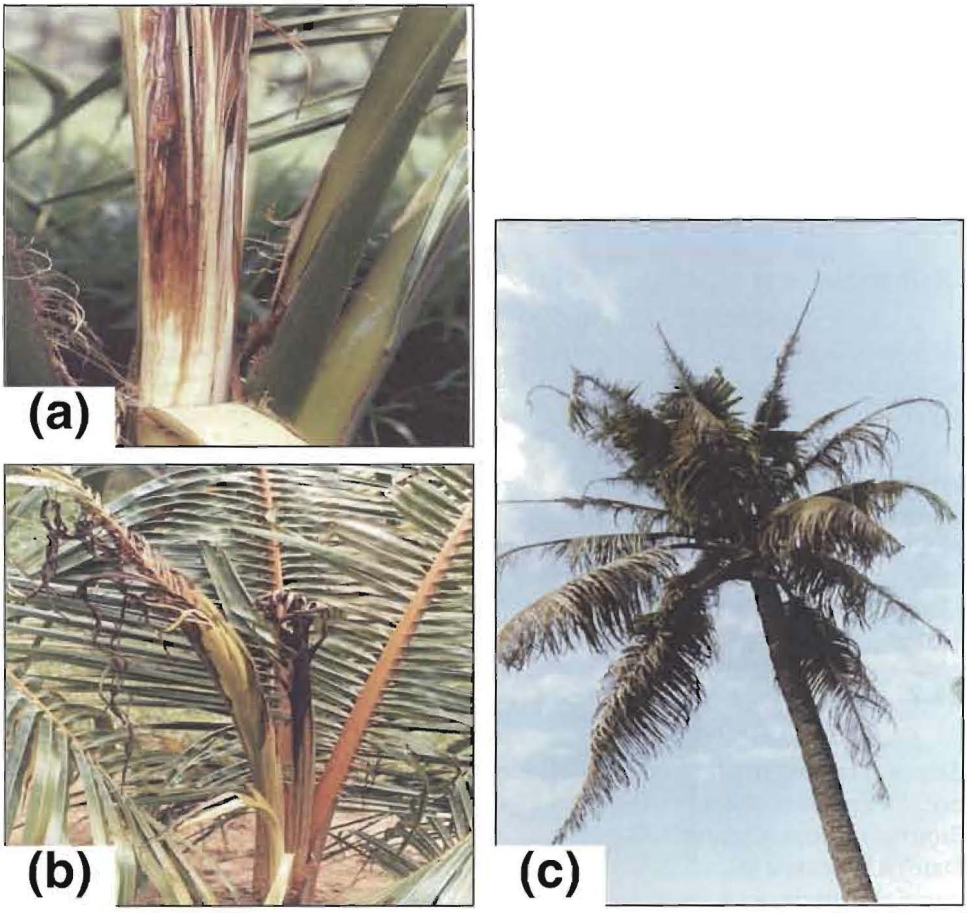

Figure 10.6: Leaf Rot
a) Reddish brown symptoms, b) Rotting of spindle leaves, c) Leaf rot on root (wilt) affected palm.

based pruning of infected spindle leaf and few leaves close to spindle and use of hexaconazole are the most important in controlling leaf rot (Srinivasan and Gunasekaran, 2000).

2.5. Grey Leaf Spot

This disease is widespread over all coconut producing countries, but is of little importance in well managed plantations. The disease causes serious damage in nursery plants as well as in adult palms. Grey leaf blight incidence reduces coconut yield to the extent of 10-24 per cent (Karthikeyan, 1996). Coconut palms severely affected with grey leaf blight disease have flowered relatively late than the less affected ones (Abad, 1975).

2.5.1. Symptoms

Initially, symptoms develop only on the outer whorl of leaves, especially on older leaves. Symptoms appear as minute, yellow spots, each surrounded by a grayish margin on the leaflets of older leaves. The centre of these spots later become greyish and spots may coalesce, giving the leaves a blighted appearance. Complete drying and shrivelling of the leaf blade occur, giving a blighted or burnt appearance. Some varieties are more susceptible than others (Figure 10.7).

Figure 10.7: Symptoms of Grey Leaf Spot.

2.5.2. Etiology

The fungus *Pestalotiopsis palmarum* (Cooke) Steyaert is the is causal agent of the disease. *Conidiomata* acervulus, globose or ellipsoidal, subepidermal in origin. *Conidiophores* indistinct. *Conidiogenous cells* discrete, simple, short, filiform. *Conidia* 17–25 × 4.5–7.5 µm, fusiform to ellipsoid, mainly straight, 4-septate; three median cells, concolorous, olivaceous, lower cell of 3 sometimes paler, together 11.5–16.5 µm long; apical and basal cells hyaline; with three appendages, 5–25 µm long, arising from the apex of the apical cell; filiform basal appendage, 2–6 µm long (McKenzie, 2013).Over the years there has been confusion with the names *Pestalotia* and *Pestalotiopsis*. Guba (1961) accepted over 200 names in *Pestalotia*, but Sutton reviewed the genera and placed those species with 5-celled conidia into *Pestalotiopsis*, while retaining *Pestalotia* for those species with 6-celled conidia. Thus, the older literature uses the name *Pestalotia* while the modern literature usually refers to *Pestalotiopsis* as the most common genus encountered on coconut. Maharachchikumbura *et al.* (2012, 2014) described several species based on molecular studies and epitypification of species. Brown (1973) found that this fungus was the cause of the most common leaf spot of coconuts in Solomon Islands and noted distinct differences between lesions associated with *P. palmarum* and with three other *Pestalotiopsis* species on coconuts.

2.5.3. Management

Cutting and removal of severely affected lower leaves and spraying of fungicides like carbendazim (0.1 per cent) or Bordeaux mixture (1 per cent) to affected palm immediately after the appearance of symptoms will reduce the incidence.

2.6. *Lasiodiplodia* Leaf Blight of Coconut

Reports of this disease have come from various parts of the world, such as Trinidad, Brazil, Malaysia, Sri Lanka and India (Ram, 1993; Bhaskaran *et al.*, 2007; Monteiro *et al.*, 2013). The fungus accelerates the death of palms having been weakened by other causes namely lack of drainage, moisture stress and malnutrition. Leaf blight is an emerging serious problem in Coimbatore, Erode, Dindigul, Tirunelveli and Kanyakumari districts of Tamil Nadu. Though leaf blight is present in coconut growing areas of other states of India, the disease is not a serious problem.

2.6.1. Symptoms

The pathogen causes damage in leaf and nuts. Affected leaflets start drying from the tip downwards and exhibit a charred or burnt appearance (Figure 10.8). The leaves in lower 3 to 4 whorls are affected. Leaf blight induces apical necrosis of lower leaves with an inverted "V" shape, and symptoms similar to those induced by drought (water deficit) and other stresses. The leaflets have extensive necrotic lesions with defined edges and without transition areas between the necrotic and healthy tissues. The pathogen can internally colonize the rachis, inducing internal necrosis that moves upward toward the stem (systemic invasion). The necrotic tissues develop exposed cracks that release gums under the leaf rachis and at petiole insertion (Souza-Filho *et al.*, 1979) on coconuts, small black sunken region appear near the perianth of immature nuts. The eryiophyid mite attacked nuts are infected by the pathogen and cause immature falling of nuts and rotting. When nearly mature/mature nuts were infected the infection spread internally into mesocarp without any external symptoms. The affected nuts are desiccated, shrunk, deformed and drop prematurely causing 10 to 25 per cent loss in nut yield (Venugopal and Chadramohanan, 2006).

3. Phytoplasma Diseases

Until 1967, numerous yellow-type diseases of plants were thought to be caused by viruses in view of their infective nature and transmission by insects. However, for the first time Doi *et al.* (1967) from Japan identified mycoplasma like organisms (MLOs) in the phloem elements of plants infected with aster yellows are as the causal agent of the disease. MLOs are very small bacteria that are enveloped only by a single membrane and do not possess a cell wall like typical bacteria. Later the plant pathogenic MLOs were termed as phytoplasmas and are grouped under the class *Mollicutes* (Firrao, 2004). Due to their inability to grow *in vitro*, they were poorly characterized until the advent of molecular biology. Similarly the phytoplasma infecting palms were poorly characterized until recently and the diseases caused by them were called by different names in different countries based on the symptoms. The detection and sequencing of 16S rDNA now showed association of phytoplasma belonging to different taxonomic groups with coconut (Table 10.2).

Figure 10.8: Symptoms of *Lasiodiplodia* Leaf Blight.

3.1. Lethal Yellowing

Lethal yellowing (LY) is the single most important disease threatening coconut production worldwide. LY of coconut was first recorded in Grand Cayman Island in 1834 and Jamaica in 1884. Currently the LY is destroying the palms in Southern United States, Central America and Caribbean as well as west and east Africa. The disease caused by LY group phytoplasma is being known by different names in west Africa *viz*. Cape St paul Wilt in Ghana, Kribi disease in Cameroon, Kaincope disease in Togo, Akwa disease in Nigeria. In east African countries like Tanzania, Kenya

Table 10.2: Phytoplasmal Diseases of Coconut and Taxonomic Group of Associated Phytoplasma

Disease	*Country*	*Vector*	*Taxonomic Group*	*References*
Lethal yellowing	Florida	*Myndus crudus*	16SrIV-A	Harrison *et al.*, 1994
Lethal yellowing	Jamaica	Not identified	16SrIV	Myrie *et al.*, 2007
Lethal disease	Tanzania	*Diastrobus mkurangai, Meenoplus* spp.	16SrIV-C	Mpunami *et al.*, 1999
Lethal decline	Mexico	Not identified	16SrIV-B	Harrison *et al.*, 2002
Awka disease	Nigeria	*Meenoplus proximus, Melenia cocos*	16SrXXII	Tymon *et al.*, 1998
Cape St.Paul wilt	Ghana	*Myndus adiopodoumeensis*	16SrIV	Tymon *et al.*, 1998
Coconut yellow decline or Malaysian wilt	Malaysia	Not identified	16SrXIV	Nejat *et al.*, 2009
Weligama wilt	Sri Lanka	Not identified	16SrXI	Perera *et al.*, 2002
Kalimantan wilt	Indonesia	Not identified	16SrXI	Warokka *et al.*, 2006
Root (wilt)	India	*Proutista moesta, Stephanitis typica*	16SrXI	Manimekalai *et al.*, 2010

and Mozambique, it is known as 'lethal decline' (Brown *et al.*, 2007). The disease has killed millions of coconut trees in Caribbean and This disease, has significantly reduced the number of tall-type *Cocos nucifera* (coconut) in Florida and the Caribbean Basin, and localized outbreaks continue to occur (Harrison *et al.*, 2014).

3.1.1. Symptoms

The symptoms of LY disease in coconut palms consist of essentially four stages. The first stage which is sometimes called shelling, involves premature nutfall with most of the nuts having a black or brown water-soaked area under the calyx. The second stage involves the necrosis of the inflorescence. The third stage involves the yellowing of the fronds of the coconut palm. This usually begins with the oldest fronds eventually advancing to the crown of the plant. Fronds that exhibit these symptoms will eventually die and can be easily removed. Death of the bud occurs about halfway through the yellowing sequence. The newly emerged spear leaf will collapse and hanging down within the crown. The fourth stage involves complete defoliation, the top of the tree falls away leaving what is known as the telephone pole effect (Brown *et al.*, 2009; Harrison *et al.*, 2014).

3.1.2. Etiology

When the LY disease was first named lethal yellowing, it was thought to be a virus (Nutman and Roberts, 1955). In 1972 transmission electron microscopy studies conducted on samples obtained from Jamaica, identified the pathogen to be a mycoplasma (now known as phytoplasma) (Beakbane *et al.*, 1972; Heinze *et al.*, 1972; Plavsic-Blanjac *et al.*, 1972). PCR-based assays developed in the late 1980's and early 1990's have served to further advance the diagnostics of phytoplasma diseases. These assays provide a much more sensitive means than serological tests or DNA-DNA hybridization for the detection of phytoplasmas. Over the last 15 years, phytoplasma universal as well as group specific primers have been developed based on the highly conserved 16S rRNA gene sequences, 16S-23S intergenic spacer region gene sequences, conserved ribosomal protein (rp) gene and elongation factor EF-Tu (tuf) gene sequences (Lim and Sears, 1991; Davis and Lee, 1993; Firrao *et al.*, 1993; Lee *et al.*,1993). The coconut lethal yellowing group of phytoplasmas have been classified as being members of group 16SrIV according to the classification of Lee *et al.* (1998) and has been divided into four subclades (16SrIV-A, 16SrIV-B, 16SrIV-C and 16SrIVD). Recently, phytoplasmas from other 16S ribosomal groups have been associated with diseases showing similar symptoms in palms in other parts of the world (Table 10.2). Scientists were attempting to culture the phytoplasma for many years and could not succeed. However, Contaldo *et al.* (2012) from Italy were able to obtain axenic culture of phytoplasma belonging to various groups. This breakthrough finding will help to understand these tiny pathogens further.

3.1.2. Epidemiology

The disease affects palms at all ages, including transplants as young as 18 months and affected palms die within 4-6 months of the onset of symptoms (McCoy *et al.*, 1983; Been 1995; Donselman 1999). Phytoplasmas are phloem limited, as a result only phloem feeding insects can potentially acquire and transmit the pathogen.

Phytoplasmas are transmitted in a persistent manner. The cixiid, *Haplaxius* (*Myndus*) *crudus* was shown in transmission trials to be a vector of LY in Florida (Howard *et al.*, 1983). In Jamaica, during a search for vectors of LY, leafhoppers were found to predominate in the undergrowth, while planthoppers (Fulgoridae) were the most prevalent group on palms (Dabek, 1981). Then the cixiid, *Haplaxius* (*Myndus*) *crudus*, was the most abundant plant hopper, and was the prime suspect of LY in Jamaica, although extensive transmission trails failed to confirm this possibility (Schuiling *et al.*, 1976; Eden-Green, 1978; Eden-Green and Schuiling, 1978; Dabek, 1981). In a study conducted by Brown *et al.* (2006), the LY group of phytoplasmas were detected in 30 per cent of the *Cedusa* species of Derbids analysed. It was also noted that variation in the phytoplasma could be seen in 6 of the insects that were tested positive for LY. With the exception of work done by Howard *et al.* (1983), no vector has yet been identified for LY outside of Florida. It is generally accepted that seed transmission of LY is unlikely because the phloem sieve elements in which phytoplasma usually reside lack any direct connection to seed. Cordova *et al.* (2003), conducted *in situ* PCR of 72 coconut embryos, 13 of which were found to have LY phytoplasma. Though detection of DNA in embryo is suggestive of possible seed transmission, seed transmission of LY could not be demonstrated as yet. Apart from coconut, at least 36 palm species have been documented as susceptible to lethal yellowing, but coconut palm (*Cocos nucifera*) is most vulnerable to the disease, followed by *Pritchardia* species, Christmas palm (*Adonidia merrillii*), and date palm (*Phoenix dactylifera*).(Harrison and Elliott, 2008).

3.1.3. Disease Management

In controlling phytoplasmal diseases, the primary concern is often prevention rather than treatment, which include control of insect vectors and weed plant hosts act as sources of inoculums, rogue out and destroy symptomatic plants and avoid planting susceptible crops next to crops harboring phytoplsma (Lee *et al.*, 2000). To reduce the rate of spread of the disease, eradication of diseased palms and those showing disease symptoms is fore most important. Development of tolerant or resistant varieties was demonstrated in Jamaica in 1974 when a cross between the Malayan Dwarf and the Panama Tall produced the MayPan hybrid that was shown to be resistant to LY for 15 years. This resistance has now been broken and no cultivar currently being cultivated in the north-east where the disease is active has shown any sign of possessing a high or any level of resistance (Wallace, 2002). Good farm practices such as the proper fertilizing and care of the coconuts will result in healthy plants that will be better able to withstand the disease than an unhealthy plant. Antibiotic application of oxy-tetracycline-HCL could be used to suppress LY symptoms (McCoy *et al.*, 1976).

3.2. Root (Wilt) Disease

Root (wilt) is disease of coconut is prevalent in India for the last 135 years and is one of the major causes of low productivity of coconut in disease endemic tracts. The disease was first noticed in erstwhile princely state of Travancore around 1874 and became very much evident after the great floods of 1882. The disease is prevalent in a contiguous manner in all the 8 southern districts of Kerala starting

from Trivandrum to Trichur and in isolated patches in the remaining 6 northern districts of the state. Apart from this the disease has also been noticed in isolated pockets in Tamil Nadu mainly in the districts adjoining to Kerala. The annual crop loss due to the disease is estimated to be about 968 million nuts. 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 coconut price index was of the order of about Rs. 3000 million. The details about the history and work done on root (wilt) disease of coconut is reviewed by many scientists who worked on the disease (Koshy, 1999; Solomon and Geetha, 2003; Ramjegathesh *et al.*, 2012)

3.2.1. Symptoms

The most diagnostic symptom of the disease is abnormal inward bending or ribbing of the leaflets in mid whorl termed as flaccidity (Radha and Lal, 1972). The other associated symptoms are foliar yellowing and marginal necrosis (Figure 10.9). An index method has been developed on the basis of the quantitative evaluation of the foliar symptoms of 7000 palms of varying age (George and Radha, 1973). Rotting of root system, drying of spathe and necrosis of spikelets are observed in certain cases (Menon and Pandalai, 1958). The nuts from diseased palms have thinner husk and fibers are definitely weaker and less firm. The kernel is thinner and never dries up into hard brittle copra but remains soft and flexible.

3.2.2. Etiology

Initially, a number of biotic agents and abiotic factors were implicated as cause of root (wilt) disease of coconut in India. Systematic study carried out over the year's upto 1980s has ruled out the association of fungi, bacteria, viruses or viroids or nutrient deficiency as causal agent of the disease. Concentrated and intensive research carried out at Central Plantation Crops Research Institute, Kasaragod, India has resulted in the identification of a phloem bound mollicute – phytoplasma as the cause of the disease. Solomon *et al.* (1983) reported the association of *Phytoplasma* in the sieve tubes of juvenile tissues such as sub-meristem, petiole of developing leaves, rachilla of unopened inflorescence and root apices of diseased palms through electron microscopy study. Phytoplasma are coccoid forms and size range of 200-450 nm, bound by a triple layered unit membrane and have well defined internal structures such as ribososmes and DNA strands. Generally they occupy parietal position close to the sieve area. Later etiology of root (wilt) disease of coconut was established as phytoplasma by electron microscopy, transmission studies with vectors, transmission through dodder and light microscopic staining techniques, symptom remission through application of oxytetracycline etc. (Solomon, 1991; Pillai *et al.*, 1991; Mathen *et al.*, 1987; Rajan *et.al.*, 2000). Experimental transmission of phytoplasma from coconut to periwinkle (*Catharanthus roseus*) using *Cassytha filiformis* and the remission of symptoms in oxytetracycline treated palms reinforced the pathogenic role of phytoplasma in coconut root (wilt) disease. Further the association of phytoplasma with root (wilt) disease of coconut was confirmed by nested PCR amplification and sequencing of of 16S rDNA. *In silico* restriction digestion study of phytoplasmal 16S rRNA gene region between primers R16F2n/R16R2 produced restriction profile identical to 16SrXI-B sub group. This formed

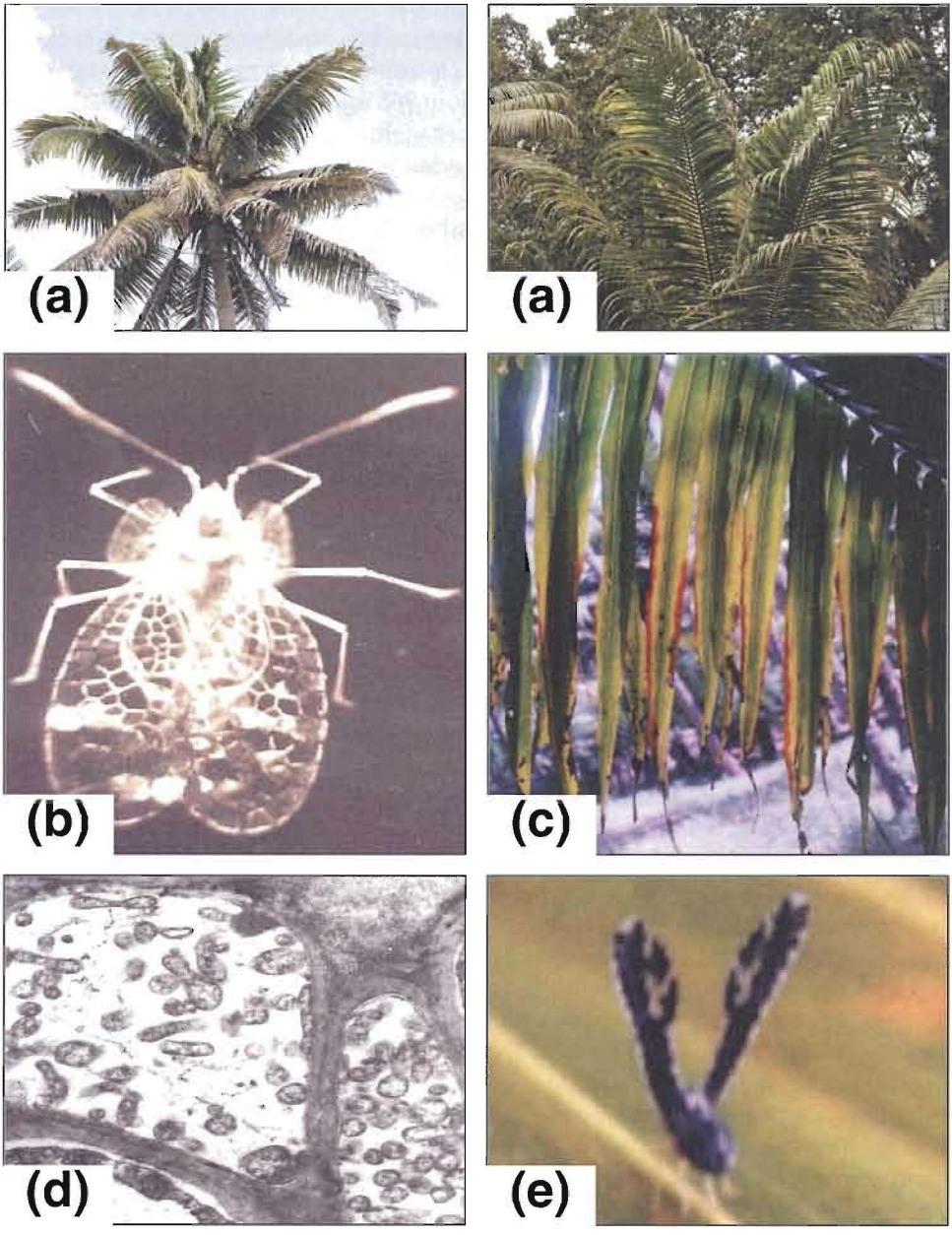

Figure 10.9: Root (Wilt) Disease of Coconut.

a) RWD affected coconut palm showing yellowing and flaccidity; b) ***Stephanitis typica***; c) Yellowing and Marginal necrosis of the leaf; d) Electron micrograph showing of phytoplasma in the sieve tubes of affected coconut; e) ***Proutista moesta.***

the first report of the association of 16SrXI-B phytoplasma with coconut in the world (Manimekalai *et al.*, 2010). In the phylogenetic analysis based on 16S rRNA gene (F2nR2 region), the RWD phytoplasma clustered with the rice yellow dwarf (RYD) and bermuda grass white leaf (BGWL) group phytoplasmas. However, in the subcluster, the root (wilt) disease phytoplasma grouped with the sugarcane white leaf (SCWL), arecanut yellow leaf disease (YLD) and sugarcane grassy shoot (SCGS) phytoplasmas, all belonging to the RYD group. For finer differentiation, the *sec A* gene based phylogeny was also considered. The RWD phytoplasma clearly clustered with the YLD, NGS, and SCGS phytoplasma, all belonging to the RYD group. Hence, the coconut RWD phytoplasma belongs to the RYD group identified as '*Ca.* Phytoplasma *oryzae*'-related strain (Manimekalai *et al.*, 2014).

3.2.3. Transmission

Root (wilt) disease is transmitted in nature by insect vectors *viz.* lace bug *Stephanitis typica* and plant hopper *Proutista moesta* (Figure 10.9) as evidenced by experimental transmission studies (Mathen *et al.*, 1990; Rajan *et al.*, 2002). The lace bug inoculated seedlings showed the presence of phytoplasma between 9 and 27 months after first inoculation and by 17th month after inoculation, 50 per cent of the inoculated seedlings showed flaccidity, the diagnostic symptom of the disease. In the case of plant hopper, 6/8 inoculated seedlings showed the presence of phytoplasma in 5-24 months after first inoculation and five of the seedlings exhibited flaccidity symptom confirming the transmission of the disease. A systematic inventory of all insect visitors to coconut garden made using various traps and confirmation of their occurrence in coconut foliage by direct over a period of two years led to the identification of a leaf hopper *Sophonia greenii* Distant, a plant hopper, Proutista moesta West wood and lace bug, *Stephanitis typica* (Rajan and Mathen, 1984, 1985). The potential of theses insects to acquire the organism was verified using electron microscopy and phytoplasma was observed in brain and salivary glands of lace bug *Stephanitis typica* and plant hopper *Proutista moesta* given an acquisition plus incubation period ranging from 18-23 days (Mathen *et al.*, 1987; Rajan *et.al.*, 2002).

3.2.4. Epidemiology

Epidemiological investigations on root (wilt) disease revealed that the spread was erratic and irregular irrespective of soil conditions and occurred in jumps/ leaps. The rate of spread was 1-4 km from the nearest source of infection. The pattern of spatial distribution or galaxial outbreak of the disease was suggestive of the involvement of aerial vector(s) in the spread of the disease. Experimental transmission studies conducted in the past proved the transmission of the disease through the lace bug, *Stephanitis typical* (Distant) [Tingidae : Heteroptera] in the field and in the insect proof house. A systematic inventory of insects in root (wilt) prevalent gardens was made using various traps *viz.*, rotary trap, suction trap, light trap and sticky traps and confirmation of their occurrence in coconut foliage by direct examination of 500 young coconut seedlings over a period of two years. In this investigation, besides lace bug, a leaf hopper, *Sophonia greeni* (Distant) and a plant hopper, *Proutista moesta* (Westwood) were recorded (Rajan *et al.*, 2000).

3.2.5. Diagnosis

Initial investigations on the development of diagnostic techniques were based on the biochemical tests of altered host metabolisms detectable in the form of either accumulation or depletion of substances consequent to differential enzymatic activity in diseased palms. But these changes can also be induced by other biotic and abiotic stresses. With the establishment of the phytoplasmal etiology more thrust was given on the development of a rapid and reliable diagnostic technique. Physiological changes like stomatal resistance and transpiration rate in healthy and diseased palms were also used for diagnosis of RWD. High stomatal resistance with a correspondingly low transpiration rate was recorded in healthy palms in contrast to low stomatal resistance and high transpiration rate in diseased palms (Rajagopal *et al.*, 1986). Intensive research in purification of phytoplasma led to the development of standardized protocols for the purification of RWD phytoplasma by percoll double density gradient centrifugation, production of polyclonal antisera specific to coconut RWD phytoplasma and Direct Antigen Coated-Enzyme Linked Immunosorbent Assay (DAC-ELISA) for the detection of RWD phytoplasma even 24 months before symptom manifestation. For the screening of large number of samples the indirect DAC-ELISA has been refined in to a simple and rapid detection technique in which the results could be obtained within 7 h. (Sasikala *et al.*, 1998; Sasikala *et al.*, 2001).

Till 1990s, EM and serology-based techniques were used for the detection of phytoplasma in coconut and vectors. With the advent of molecular biology, efforts were made by scientist to develop polymerase chain reaction (PCR) protocol for rapid and reliable detection of RWD phytoplasma. Preliminary attempts made to detect the coconut RWD phytoplasma using 16S rRNA sequence based universal primers failed to give consistent results in direct and nested PCRs.

Molecular detection of phytoplasma associated with RWD was achieved by modification of phytoplasma enrichment technique for DNA extraction by addition of 5 per cent polyvinyl pyrrolidone, designing six highly sensitive primers and semi-nested PCR technique. Immature spindle leaves and midribs have been found to be ideal for DNA extraction. PCR conditions for the custom- designed primers sets were also standardized. The semi-nested primer pair 3Fwd/3Rev–3Fwd/5Rev produced an amplicon of 1.3 kb size. The primer pair 1F7/7R3 semi-nested with 1F7/7R2 amplifies a 493 bp fragment of 16S rRNA region of coconut RWD phytoplasma (Manimekalai *et al.*, 2010, 2014). Later a real -time PCR protocol was also developed for detection of RWD phytoplasma (Manimekalai *et al.*, 2011). However it was observed that there was no consistency in detection of phytoplasma by nested PCR, real-time PCR and loop-mediated isothermal amplification (LAMP) when large number of root (wilt) affected coconut samples were tested and further refinement of these techniques are necessary for reliable and rapid detection of RWD in the early stage of infection (Hegde *et al.*, 2016).

3.2.6. Disease Management

Root wilt is not a lethal disease but a debilitating malady and responds to good management. Yield of the palms can be sustained or even improved through

the adoption of integrated management practices. Several experiments were done earlier to manage the diseased garden to improve the yield levels and income of the farmers by recommending balanced fertilizer application, recycling of organic wastes,irrigation of palms during summer months etc. (Bavappa *et al.*,1982; Sahasranaman *et al.*,1983; Amma *et al.*,1983; Thomas *et al.*,1993; Rajagopal *et al.*, 1987). Identification of coconut varieties/hybrids resistant/tolerant to root (wilt) disease was initiated as early as 1953. Intensive large scale screening of coconut gerplasam and varieties for resistance to root (wilt) was also carried out and no palms resistant to root (wilt) was observed. Further systematic evaluation trials at ICAR-CPCRI Regional Station, Kayamkulam for developing varieties with resistance/tolerance to root (wilt) disease has led to the release of three coconut varieties for the root (wilt) disease prevalent tract namely, Kalparaksha (selection of Malayan Green Dwarf), Kalpsree (selection from Chowghat Green Dwarf) and a hybrid Kalpa Sankara (Chowghat Green Dwarf X West Coast Tall). Based on the experimental results on managing the root (wilt) disease for many years, management practices advocated for RWD has also been refined from time to time. The following integrated disease management practices are recommended at present.

i) In Mildly Affected Areas

- ☆ Eradication of the disease by removal of affected palms
- ☆ Replanting with resistant/tolerant varieties *viz.*, Kalparaksha, Kalpasree and the hybrid Kalpa Sankara.

ii) In Heavily Disease Affected Tracts

- ☆ Remove severely affected and uneconomic adults palms (yield < 10 nuts/palm/year) and all diseased palms in the prebearing age.
- ☆ Apply the recommended dose of N (560 g), P (320 g) and K (1200 g), 3 kg magnesium sulphate and 50 kg organic manure per palm. Apply the fertilizers in two splits, one third during April-May and two-third during September–October under rainfed condition and in four splits during January, April, July and October under irrigated conditions.
- ☆ Adopt mixed farming by growing fodder crops (guinea grass or hybrid napier) as mixed crops in coconut garden coupled with dairy enterprise.
- ☆ Grow suitable intercrops like banana, nutmeg, pineapple, pepper and tuber crops like elephant foot yam etc.
- ☆ Basin management with green manure/cover crops such as cowpea, sunhemp calopogonium etc.
- ☆ Irrigation during summer months with 250 to 300 litre of water once in 4 days for adult palms; 75-80 litres once in 4 days for 3 to 4 year old seedlings; 25 to 30 litres once in 2 days for 1 to 2 years old seedlings.
- ☆ Management of leaf rot as mentioned in the previous section 2.4.4 in this chapter.

3.3. Tatipaka Disease

The disease is named after the village 'Tatipaka' in Andhra Pradesh, India where the disease was first noticed following a cyclone in 1949. The disease is endemic in east and west Godavari, Srikakulam, Nellore, Krishna and Guntur districts (Subbaih and Rao, 1963; Rethinam *et.al.*, 1989). Survey carried out during 1990 identified about 8179 coconut palms affected by Tatipaka disease in these districts (Rajamannar *et al.*, 1993). The disease is non lethal but of debilitating nature, generally affecting coconut palms in the age group of 20-60 years. Palms below 20 years of age are very rarely affected. Spread of the disease is not contiguous but sporadic at slow pace of 3.5 per cent over a period of five years (Solomon and Geetha, 2003).

3.3.1. Symptoms

The disease affected coconut palms generally bear profusely for 2 to 3 years before the expression of foliar symptoms. With the onset of disease, there is a reduction both in number and size of leaves. The leaves exhibit characteristic chlorotic water soaked spots and the fronds bend abnormally sometimes twisting in loops. In the advanced stage there is a severe reduction in size of the crown. The leaves give a fascinated appearance due to improper unfolding of leaflets. The spathes produced are very small with very few rachilla. The bunches contain a mixture of normal and atrophied nuts. 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 oozing of gummy exudates. In the advanced stages of the disease, the stem tapers, produces smaller spathes and inflorescence which ultimately do not bear any fruit (Ramapandu and Rajamannar, 1981).

3.3.2. Etiology

Like root (wilt), initially the disease was considered as that of an uncertain etiology. The possible involvement of fungi, bacteria was ruled out since these microbes could not be isolated consistently form diseased palms. Sap transmission studies and electrophoresis of isolated DNA from the diseased palms ruled out the virus or viroid as the causal agent (Randles and Hatta, 1980; Rajamannar *et al.*, 1984). Electron microscopic examination of tender roots, meristem, petioles of developing leaves and rachilla of tender inflorescence of diseased palms revealed the presence of phytoplasma in the sieve tubes. Light microscopy with Dienes stain and fluorescence microscopy with aniline blue as fluorochrome also revealed the presence of phytoplasma in the diseased coconut palms (Rajamannar *et al.*, 1993).

3.3.3. Disease Management

Since there are no prophylactic or curative measures for phytoplasma diseases, regular surveillance and removal of the diseased palms is recommended to arrest the further spread of the disease. Since the spread of the disease is very slow, removal of the affected palms has helped in eradication of the disease.

3.4. Weligama Wilt Disease

During late 2006, an unusual yellowing and flaccidity of leaflets on coconut palms were observed in the Weligama area in the Matara district in the southern part of Sri Lanka (Wijesekara *et al.*, 2008; Perera *et al.*, 2010) and the syndrome was named as "Weligama coconut leaf wilt disease"(WCLWD). At present it is prevalent in the divisional secretariat regions Akuressa, Athuraliya, Devinuwara, Dickwella, Hakmana, Kamburupitiya, Kirinda-Puhulwella, Malimboda, Matara, Pitabeddera, Thihagoda, Weligama and Welipitiya of Matara district, Galle and Habaraduwa of Galle district and Beliatta, Ookewela, Tangalle and Walasmulla of Hambantota districts in southern Sri Lanka (Perera *et al.*, 2012).

3.4.1. Symptoms

The earliest symptom of the disease is the flattening and downward bending of leaflets giving to the frond a ribbed or flaccid appearance. Crowns of such palms appear dark green in color. This symptom is first seen in the younger leaves and becomes more prominent when the fronds are fully opened. The intense yellowing of lower whorls of fronds, which is more prominent just after the rainy season up to about the 12th frond, is also a specific symptom of the disease. Occasionally, yellowing of mid whorls of fronds is seen in some palms. In such cases, yellowing is restricted to about 6 to 8 fronds in the middle whorl. Subsequently, drying up of the leaflets starts from the margins of the affected fronds and dried fronds hang in the crown for some time before falling from some severely affected palms, the fronds also curl down-ward giving a ragged appearance to the crown. The tips of fronds become twisted or break and hang down in some palms. Unopened bud leaves lose their rigidity and bend downwards in severely affected palms. With the reduction in the number of fronds, the crown becomes smaller and the trunk begins to taper. As the disease progresses, female flower production declines and the palm becomes unproductive (Wijesekara *et al.*, 2008; Perera *et al.*, 2010, 2012).

3.4.2. Etiology

Phytoplasma belonging to 16SrXI *Candidatus* Phytoplasma *oryzae* group has been reported as causal agent of the weligama wilt in Sri Lanka. The phytoplasma was found to be highly similar but not identical to Sugarcane white leaf phytoplasma (99 per cent), Sugarcane grassy shoot phytoplasma (99 per cent) and coconut root (wilt) phytoplasma (99 per cent) based on the phylogenetic analysis of the 16S rDNA sequences amplified through PCR (Perera *et al.*, 2012).

3.4.3. Epidemiology

The syndrome is prevalent in all type of soils from flat coastal areas to undulating lands inland, and the affected palms are distributed in pockets, sometimes several kilometers away from each other.

3.4.4. Disease Management

The strategies adopted for disease management in Sri Lanka are; establishment of 88 km long and 3 km wide barrier around boundaries of the three districts to prevent the spread of the disease to the other major coconut growing areas of the

country. This barrier is maintained by the Coconut Research Institute, Sri Lanka. The diseased palms were detected using molecular diagnostic techniques which have been perfected by the Coconut Research Institute. Removal of severely diseased palms in order to reduce the pathogen density and this in return will reduce the rate of spread (Source: www.news.lk).

4. Virus Diseases

4.1. Coconut Foliar Decay or Vanuatu Wilt

The coconut foliar decay is virus disease of introduced coconut palms in Vanuatu. It is also known as foliar decay *Mindus taffini* or New Herbides coconut disease. The name *"Mindus taffini"* belongs to the plant hopper insect at the transmits the disease. The disease is economically important because of its influence on regional coconut industry and internationally as a quarantine pathogen.

4.1.1. Symptoms

The first symptom on palms in the field is yellowing of a few leaflets on any of the fronds between position seven and 11 from the spear leaf. The yellowing spreads along the fronds, and the fronds break near the base so that they hang down through the still green lower leaves. As the younger leaves age, reaching positions seven to 11, they, too, turn yellow, break and hang down. In time diseased palms have a few green fronds at the top, and broken mid-section fronds hanging through the lower, still green fronds below. As the disease progresses, the trunks narrow towards the top, and the palms die after 1-2 years, except for those that are tolerant to the disease and show remission of symptoms. Foliar decay is a more serious on "Malayan Red Dwarf" coconut introduced to Vanuatu. Coconut cultivars Vanuatu tall and Vanuatu Dwarf are usually not affected by the disease, though they are the host for the foliar decay virus i.e they act as symptomless carrier. (Randles *et al.*, 1992; Hanold *et al.*, 2003).

4.1.2. Etiology

The disease is caused by a very small circular single stranded DNA virus and which is named as *Coconut foliar decay virus* (CFDV, an unassigned species under family *Nanoviridae*) (Randles *et al.*, 1986). The virus is found at very low concentrations in coconut palms. It is difficult to see the virus particle in the sap viewed by electron microscopy. The virus occurs in the phloem of the coconut. *Cocus nucifera* is the only known host of the virus. Virus occurs in leaves, roots, embryo, trunks and even on the husk of the nut. Seed transmission is not yet established.

4.1.3. Transmission

The disease is transmitted by *Myndus taffini* Bonfils (Cixiidae) a plant hopper which breeds on the roots of *Hibiscus tiliaceous*, a tree commonly found in Pacific seashores. The adults of this insect feed on coconut leaves. Wefels *et al.* (2015) reported the molecular evidence for a persistent circulative association between CFDV and its vector. Both vector and virus are apparently limited in distribution in Vanuatu archipelago.

4.1.4. Disease Management

The disease is best controlled by either planting selected Vanuatu tall or the hybrid, Vanuatu Tall x Vanuatu Red Dwarf which are tolerant to the disease. The removal of the host tree of the insect that spreads the virus is likely to be beneficial but it is not possible. The *FAO/IBPGR Technical Guidelines for the Safe Movement of Coconut Germplasm* recommend that coconuts should be moved as embryos growing in a sterile tissue culture medium. As a special note, the Guidelines recommend that embryos, seedlings and palms from which pollen is collected should be tested for viroids and *Coconut foliar decay virus*. If that is not possible, seednuts may be transferred if they are germinated in intermediate (third country) quarantine, and indexed for viroids and *Coconut foliar decay virus*.

5. Viroid Diseases

Two viroid diseases namely Coconut cadang-cadang and Tinangaja are recorded on coconut and their distribution is limited to Philippines and Guam respectively.

5.1. Cadang-cadang Disease

In the early 1930s a devastating epidemic of a lethal disease of coconut palm named as cadang cadang was reported from southern Luzon in the Philippines (Randles, 1987). This disease caused tremendous economic losses in coconut plantations in the Philippines. Cadang-cadang disease is widely distributed on the Bicol peninsula, Masbate, Catanduanes, Northern Samar and other smaller islands in Philippines. Outbreaks have been found in and around Infanta, Quezon, in Eastern and Western Samar and Maripipi. Small isolated groups of infected palms have been found northeast of the main boundary at Atimonan. At present, the northernmost boundary of disease occurrence is at General Nakar, Quezon and the southernmost at Calicon, Guiuan, Eastern Samar.

5.1.1. Symptoms

In the early stage, newly developing nuts become more rounded and have equatorial scarifications. Chlorotic leaf spots begin to appear and inflorescences become stunted. In the medium stage, spathe, inflorescence and nut production decline and then cease. Leaf spots become more numerous. By the late stage, the fronds decline in size and number and the leaflets become brittle. Leaf spots coalesce, giving a general chlorosis. The crown size is reduced and later the palm dies. This progression of symptoms is remarkably constant with some variation in intensity. The Early stage lasts an average of about 2-4 years, the Medium stage about 2 years and the late stage about 5 years. Usually, palms become naturally infected only after they have reached the age of flowering. In the rare cases where younger palms become infected, they are stunted and fail to produce inflorescences, although they survive well past the age of first flowering.

5.1.2. Etiology

The detection of two small disease-associated RNAs in 1975 provided the initial clue to the etiology of cadang-cadang. Electron microscopy, nucleotide sequencing

and transmission experiments that demonstrated the infectivity of these RNAs finally proved that cadang-cadang is caused by a viroid. It is now referred to as the coconut cadang-cadang viroid or CCCVd. Viroids are the smallest known pathogens and have been found only in plants. Unlike viruses, they do not have a protein coat, and consist solely of a small circular, single-stranded infectious RNA molecule that can replicate in the host cell and be transmitted independently of any other microorganism (Hanold and Randles,1991).

5.1.3. Epidemiology

The mode of natural inoculation in the field is not known. No insect vector has been found. Positive transmission was obtained through assisted pollination of mother palms with pollen from diseased palms. A small percentage of the progenies produced as well as seed nuts collected from cadang-cadang infected palms were positive for CCCVd. CCCVd was also successfully transmitted to palms thru contaminated harvesting scythes.

5.1.4. Disease Management

At present, there is no direct control measure that can be recommended to control cadang-cadang but several possible strategies can be considered. Strict enforcement of quarantine regulations by concerned government agencies on the safe movement of coconut germplasm from infected areas will prevent further spread of cadang-cadang into disease-free areas. Continued research on cadang-cadang runs parallel to the coconut improvement program in the Philippines. To minimize the risk of an epidemic occurring in new plantings, attempts have been made to find individuals or populations that are resistant or tolerant to cadang-cadang.

6. Conclusion

Coconut being a perennial crop standing for about 60-80 years is vulnerable for various vagaries of the nature at its various growth stages. Though several pathogens infecting coconut are known, the three major pathogens threatening coconut production word over is phytoplasma, *Phytophthora* and *Ganoderma*. The lethal yellowing of coconut and other lethal yellow type diseases caused by phytoplasma are major threat to coconut, since there are no effective control measures for these diseases. Managing bud rot and nut fall caused by *Phytophthora palmivora* and *P. katsurae* is a major challenge owing to the difficulty in climbing the tall trees and taking up prophylactic measures. Other diseases are important locally and a constant regular monitoring and surveillance is essential to check the re-emergence of minor diseases and emergence of new diseases in the era of climate change. Strict domestic and international quarantines are essential for preventing the spread of the disease to healthy area. Though PCR based techniques for diagnosis of phytoplasma, viruses and viroids are available, development of field level reliable quick diagnostic kit is essential.

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