

Diseases and Disorders of Coconut

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1. INTRODUCTION

Coconut palm, in spite of its hardy nature is affected by many diseases and disorders. Coconut diseases may be classified as those affecting the bud, the leaf, the stem and the root. Some diseases like fungal diseases are of known etiology and some others are of uncertain etiology. The major diseases and disorders are described in this chapter.

2. BUD ROT

Bud rot occurs commonly in almost all coconut growing countries. The disease was first noticed in Grand Cayman before the turn of this century (Tucker, 1926). The disease is now known to occur in Cuba, India, the Philippines, Jamaica, East Africa, Puerto Rico, Dominican Republic and Santo Domingo, Fiji, Papua New Guinea, Colombia, Sri Lanka, French Polynesia and Vanuatu (Butler, 1906; Menon and Pandalai, 1958; Child, 1974). The disease is sporadic in nature (Radha and Joseph, 1974), however, outbreaks of epidemics are also common. The disease occurs commonly in West and East Coasts of India (Menon and Pandalai, 1958).

2.1 Symptoms

Briton-Jones (1940) described the symptoms of the disease first. The first visible symptom is the withering of the spindle marked by pale colour. The spear leaf or spindle turns brown and bends over. Such symptom is later observed in younger leaves next to the spindle. On dissecting such affected trees, rotting of internal tissues could be observed. These tissues assume pale pink colour with a brown border. The affected spindle can easily be pulled out at this stage. The spindle droops down among the neighbouring leaves. One by one, the inner leaves also fall away, leaving only fully matured leaves in the crown (Fig. 1). A foul smell is emitted by the rotting tissue. The palm ultimately succumbs to the disease with the death of the spindle (Briton-Jones, 1940; Menon and Pandalai, 1958; Lingaraj, 1972). It took a few weeks for the decay to involve the entire bud before destroying the meristem. The tender leaves and leaf sheaths sometimes show water soaked sunken lesions at their base in the early stages.



Fig. 1 : Bud rot of coconut.

These lesions turn brownish later (Radha and Joseph, 1974). The leaflets also show lesions which in severe cases result in leaf bitten disease (Menon and Pandalai, 1958). The older nuts persist on the crown for some time, while the younger ones may fall off.

2.2 Losses

Radha and Joseph (1974) reported an incidence of 0.1 to 6.5 per cent in Kerala (west coast of India) and 0.4 to 6.7 per cent in Tamil Nadu (east coast of India) (Table 1). Occasional heavy incidence (35-40%) of the disease has been observed by them in some plantations in Kerala and Karnataka. Sporadic occurrence of the disease (between 0.9 and 10%) was observed in some gardens in Andhra Pradesh.

Table 1 : Bud rot disease incidence in Kerala and Tamil Nadu (Radha and Joseph, 1974)

State	District	% disease incidence in palms		
		Below 10 years	10-30 years	Above 30 years
Kerala	Cannanore	0.77	1.94	0.36
	Kozhikode	1.66	1.32	0.26
	Malapuram	1.10	0.10	—
	Palghat	2.10	0.10	—
	Trichur	2.10	0.20	—
	Emakulam	2.00	0.10	—
	Kottayam	0.53	1.71	—
	Alleppey	6.50	4.40	—
	Quilon	5.02	5.66	—
	Trivandrum	4.50	3.80	—
Tamil Nadu	Kanyakumari	1.60	2.90	—
	Thirunelveli	6.70	4.20	—
	Trichinopoly	5.60	0.40	—
	Ramanathapuram	1.80	1.10	—
	Thanjavur	5.90	1.50	—
	South Arcot	4.50	2.10	—

2.3 Causal Agent

Earlier workers ascribed *Pestalotia palmarum* (Busck, 1902) to this disease. However, Butler (1906), and Shaw and Sundararaman (1914) reported the causal organism as *Phytophthora palmivora* Butl. In the Philippines, *P. faberi* Maubl. has been reported as the causal agent of bud rot, while *P. heveae* in the Ivory Coast. These pathogens also cause lesions on nuts, quite independent of bud rot, and cause nut fall.

Joseph and Radha (1975) observed bud rot symptoms in 2-5 year-old seedlings of coconut inoculated with sporangial suspension of *P. palmivora* both in the laboratory and in the field. The fungus could be isolated from bud tissues affected by dry rot which occurred prior to wet rot caused by secondary saprophytic colonisers like *Fusarium*, *Xanthomonas*, *Pseudomonas* and *Erwinia*.

The hypha of *P. palmivora* is of fairly uniform diameter (3-8 μm); smooth without hyphal swellings. The much branched hyphae run more or less straight. The hyphal contents are first clear, then granular. The hyphae are usually intercellular, but occasionally intracellular.

Sporangia develop abundantly, especially in light than in darkness. Sporangia are ellipsoid or ovoid with the widest part near the base; papillate and with a short pedicel. The sporangia measures 35-60 \times 20-70 μm . The base of the sporangium is usually rounded and attaches with the sporangiophore almost at right angle. Sporangiohores

are up to 6 μm ; shows sympodial growth with a sporangium at the tip and a branch from immediately below. Zoospores are released from the sporangia; at 22-24°C. The sporangia germinate within 3 h releasing zoospores. The zoospores are biflagellate and motile in nature. The zoospores germinate and infect the tissues.

Chlamydospores (30-40 μm) are found in abundance. Sex organs (both antheridia and oogonia) have been reported. Oospores are spherical (16-30 μm) and appeared within 4 weeks in inoculated coconut petioles (Radha and Joseph, 1974).

The pathogen was reported to exist in a number of morphological and pathological forms (Briton Jones, 1940). Thomas *et al.* (1947) observed presence of 'plus' and 'minus' strains which, on pairing, produced oospores. Kumar and Hegde (1987) reported that coconut isolates belonged to A2 mating type.

2.4 Epidemiology

The fungal infection and disease development are more related to relative humidity (Menon and Pandalai, 1958). They also noticed that rainfall aggravated infection and that young palms in low and moist situations were more susceptible. Most of our knowledge on epidemiology of this disease comes from detailed studies made by Radha and Joseph (1974). Radha and Joseph (1974) studying the influence of microclimatic parameters on bud rot incidence found that minimum temperature of 21-24°C and high relative humidity of 97-100 per cent were optimum for the incidence of disease. Disease development was found to be determined by the duration of such 'favourable days' (Table 2). It was also noticed that in young palms between 5 and 10 years, the occurrence of 'favourable days' was more frequent and hence there was more disease incidence in such young palms (Radha and Joseph, 1974). Inoculation of young seedlings (one to two years old) grown in pots and provided with conditions like temperature of 24°C and RH of 98-100 per cent resulted in infection and death of spindle. It took one week for the fungus to complete its life cycle from sporangia and a minimum of 5 weeks for manifestation of symptoms (Radha and Joseph, 1974). Like the earlier workers, Radha and Joseph (1974) found that the disease was mainly confined to young palms below 25 years. While the disease incidence was between 0.5 and 6.5 per cent in palms below 10 years in Kerala, it was between 1.6 and 6.7 in Tamil Nadu. The percentage disease

Table 2 : Weather factors versus disease incidence in Kerala during 1970-71 (Radha and Joseph, 1974)

Factors	Kasaragod	Kayangulam
Rainfall (mm)	3568	1379
No. of rainy days	89	77
Min. temp. (°C)	21.1 to 21.6	22.5 to 24.8
Max. temp. (°C)	29.6 to 33.6	31.3 to 33.3
Favourable days*	15	4
No. of diseased palms	24	1

*Days of high humidity (95-98%) and low temperature (23.0 to 26.0°C) in microclimate at 9.30 h.

incidence in palms between 10 and 30 years ranged from 0.1 to 5.7 per cent in Kerala and 0.4 to 4.2 per cent in Tamil Nadu.

Menon and Pandalai (1958) reported that the fungus might remain dormant in the leaf bases during dry months. Radha and Joseph (1974) could isolate the pathogen from the basal part of the crown of naturally infected palm 5 months after the disease was noticed in the field. Oospores were also observed in the tissues of infected crown and also in culture medium when the culture was contaminated. The observation on the presence of *P. palmivora* in the roots of even healthy coconut palms by Harris *et al.* (1984) prompted them to caution that a significant infestation of coconut soil might pose a threat in the form of severe outbreaks of bud rot at a later period.

2.5 Host Range

In addition to coconut, the fungus also infects palmyrah (*Borassus flabellifer*), arecanut (*Areca catechu* L.) (Menon and Pandalai, 1958) and oilpalm (*Elaeis guineensis* Jacq.) (Joseph and Radha, 1975). Radha and Joseph (1974) found that the fungus also infected rubber (*Hevea brasiliensis*), *Bougainvillea* sp., *Hibiscus rosa-sinensis*, *Artocarpus incisa* and *A. integrifolia*. Das and Cheeran (1986) studying *Phytophthora* isolates from coconut, arecanut, black pepper, rubber, cacao and cardamom found that cross inoculations were successful.

2.6 Control

The methods of control have been described by Menon and Pandalai (1958), Peiris (1962), Radha and Joseph (1974) and Celino (1970). When the disease is diagnosed in the early stages, curative measures can save the palm. The appropriate period would be the stage when the spindle has just started showing symptoms of withering. The infected tissues are removed, and the wound is applied with 10 per cent Bordeaux paste. A few healthy leaves around the spindle may have to be removed if necessary to help thorough cleaning and removal of dead tissues from the base of the spindle. The treated bud is covered (Menon and Pandalai, 1958) with polythene sheet to prevent entry of rain water. The protective covering is retained till normal shoot emerges. In cases of advanced stages of infection, where such treatment is not of any avail in saving the palm, it is advisable to cut and remove the palms. The diseased tissues should be burnt after their removal. As a prophylactic measure, all the healthy palms in the surroundings should be sprayed with 1 per cent Bordeaux mixture. Since it is known that young palms up to 25 years are more susceptible to the disease, prophylactic spray with 1 per cent Bordeaux mixture should be given. The spraying should be mainly directed to the spindle and to the base of 2-3 inner most leaves. In certain dwarf palms, phytotoxic symptoms like brown sunken spots followed by nut fall have been observed (Nambiar, unpublished) when Bordeaux mixture is sprayed. Such phytotoxic symptoms have been reported by other workers too (Schut, 1975) when 'Kocide' was used against bud rot in dwarf coconuts. Hence, spraying copper fungicides to dwarf palms should be done with care.

Radha and Joseph (1974) found that demosan (1200 ppm) effectively checked infection in laboratory tests. Renard and Quillec (1984) reported that two rounds of stem injection with systemic fungicides like Aliette (Fosetyl-Al) and Ridomil (metalaxyl) 3 g a.i./injection per palm were effective in protecting the palms from the bud rot of coconut caused by *P. heveae*. So far, no cultivar has been found to be resistant to this disease under field conditions. Quillec *et al.* (1984) observed that hybrids were less sensitive to bud rot than West African Tall. Brahmana and Aziz Kelana (1988) reported that dwarf palms are more sensitive and tall varieties or tall x tall hybrids more resistant. Franqueville *et al.* (1989) found that among the progenies of Malayan Yellow Dwarf x West African Tall hybrid (PB 12), some were sensitive while others were highly tolerant.

Ohler (1984) suggested that measures like improved drainage, wide spacing, weed control, etc., would help in reducing the relative humidity resulting in an indirect prevention of disease incidence.

3. STEM BLEEDING

Stem bleeding disease is known to occur in all coconut growing regions in the tropics. Petch (1906) stated that this disease was first reported from Sri Lanka. Later, the disease was reported in India (Sundararaman, 1922). This was followed by reports of the disease occurrence in other countries like the Philippines, Malaysia, Trinidad, Papua New Guinea, Fiji, Ghana and Indonesia (Briton-Jones, 1940; Chona and Adansi, 1970; Menon and Pandalai, 1958; Renard *et al.*, 1984). In the early stages, of the disease, there is not much yield loss. However, in late stages, there is a steady yield decline causing considerable loss and in advanced stages even death of affected palms occurs (Nambiar and Sastry, 1988). The disease has been found to occur in all soil types, but more in laterite soils and sandy soils on the sea shore or back water areas.

3.1 Symptoms

The disease is characterised by development of dark brown patches appearing at the basal portion of the trunk. In course of time, this progresses upwards. A dark reddish brown liquid exudes from the longitudinal growth cracks present on the stem bark and form irregular streaks of exudation. These streaks may coalesce and form larger lesions (Fig. 2). No oozing is seen from old lesions. The exudates eventually dry up to form black encrustations with brownish orange margin. The tissues beneath the discoloured patch show decay. The internal decay can be observed even in areas beyond the margins of external lesions indicating that the internal decay is not confined to the area of external symptoms. As the decay progresses, the tissues become black and fibrous. This process is fast in young palms. As a result of this, cavities are formed from which liquid comes out, when the bark is pressed or punctured. Serious infection was found to lead to reduced yield and death of young palms (Ohler, 1984; Nambiar and Sastry, 1988). Radhakrishnan (1987) observed maximum symptoms of bleeding in July under red lateritic loam conditions at Pilicode (Kerala) and the least in summer months in irrigated trees growing on sandy loam soils at Nileswar (Kerala).

In the crown also, symptoms appear. The outer whorl of leaves become yellow rather prematurely, droop and finally dry up. Though nut fall is noticed in later stages of disease, it is more in palms exposed to drought conditions. The trunk gradually tapers towards the apex and the crown size is reduced. Crown symptoms are more pronounced during the summer season. During the rainy season, as well as in well managed and irrigated gardens, the crown symptoms are not conspicuous till very late. Based on the aerial symptoms, Mathew *et al.* (1989) worked out a method for indexing the disease severity.

3.2 Causal Agent

Thielaviopsis paradoxa (de Seynes) von Hohnel has been isolated from affected stem tissues. The perithecial stage (*Ceratocystis paradoxa* (Dade) Moreau) has also been recorded from the affected palms. The fungus remained a suspected pathogen till recently (Menon and Pandalai, 1958; Ohler, 1984). Nambiar *et al.* (1986) reproduced the symptoms by artificially inoculating healthy trees with the fungus thus establishing its role as a pathogen. Splitting of husk and gummy exudation on nuts of all stages have been observed in one or two gardens. *T. paradoxa* was isolated from such affected nuts (Nambiar, unpublished). Though *Phomopsis cocoina* Cke. (Punith) (Lily, 1984a) and *Schizophyllum commune* Fr. (Lily, 1984b) were recorded from stem bleeding affected coconut, their pathogenicity could not be established.

T. 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, smooth walled (6-24 × 2-5.5 µm). Chlamydospores terminal in chains, obovate to oval, thickwalled, brown, 10-25 × 7.5-20 µm. The perithecial stage is *Ceratostomella* (= *Ceratocystis*) *paradoxa* (Dade) Moreau; Perithecia partly immersed, light brown, 190-350 µm dia with numerous appendages; long and black neck, tapering up to 1400 µm, osteolar, hyaline; ascospores ellipsoid, often with unequally curved sides, hyaline, non-septate, smooth, 7-10 × 2.5-4 µm. The optimum temperature for mycelial growth of *T. paradoxa* was found to be 30°C (Nishitha Naik, 1990).

3.3 Epidemiology

The fungus is a weak pathogen and enters the trunk through wounds/growth cracks. Growth cracks may develop after sudden heavy rains following prolonged dry period (Ohler, 1984) or after heavy manuring (Briton-Jones, 1940). Trash burning has also been found to cause damage to the stem (Nambiar and Sastry, 1988). Poor drainage, formation of hard laterite pan leading to crippled root growth, imbalanced mineral nutrition or other physiological disorders are other predisposing factors (Sulladmath *et al.*, 1980). Climatic factors also could play a role in symptom expression. From inoculation studies, it has been found that the progress of the disease was faster during months (July-November) of high humidity and moderate temperature (Nambiar *et al.*, 1989).

Potty and Radhakrishnan (1978) reported that increased levels of P caused increased disease incidence and that application of N at the rate of 0.35 kg/palm reduced

the disease, though a further rise did not reduce the incidence. Mathew and Ramanandan (1980), on the contrary, could not observe any significant difference in major nutrient content between healthy and diseased palms, nor was disease incidence related to soil pH and electrical conductivity. In Indonesia, chlorine deficiency is reported to be associated with the disease and correction of chlorine deficiency is reported to prevent the disease (Renard *et al.*, 1984; Van Uexkull, 1985). Nagarajan (1985) reported that excessive salinity with high sodium during summer is associated with stem bleeding. The very fact that the disease is often noticed on the banks of back water areas or on sea shores is testimony against the association of chlorine or sodium deficiency with the disease.

3.4 Control

The control measures adopted till recently mainly consisted of phytosanitation measures involving removal of affected bark tissues with a chisel and application of hot coal tar or Bordeaux paste to protect the wound. The chiselled tissues are destroyed by burning. Reddy *et al.* (1985) found that *C. paradoxa* developed tolerance to copper sulphate in *in vitro* tests. Nambiar and Sastry (1988), and Anon., (1990) obtained encouraging results in control when Bavistin or Calixin were applied through root feeding. Radhakrishnan (1990) reported that drenching Calixin (0.1%) at 25 l per tree once in every two months arrested the spread of the disease. Since wounds on the trunks predispose the palms to infection, any type of such wounding of palms is to be avoided. Care should be taken not to injure the stem base while ploughing the garden with tractor.

Provision of summer irrigation to avoid extreme variation in soil moisture in the garden or adopting practices to conserve soil moisture are beneficial in reducing growth cracks. With a view to increasing the vigour of the palm, the recommended dose of organics and fertilizers is to be applied. Application of neem cake at 5 kg per palm was found to increase the soil microflora including *Trichoderma* spp. which were found to inhibit the pathogen *in vitro* (Gowda and Nambiar, 1987) and in tissues under lab conditions (Usman, 1988). Radhakrishnan and Potti (1980) reported in their preliminary studies that among the indigenous coconut cultivars tested, West Coast Tall was the most resistant.

4. LEAF ROT

Leaf rot disease was in existence in the erstwhile Travancore-Cochin state in the last century itself (Menon and Nair, 1948). The name leaf rot was suggested by Radha and Lal (1968) for the foliar necrosis found in the root (wilt) tract of southern Kerala. Palms affected by root (wilt) disease are generally superimposed by leaf rot disease (Sundararaman, 1925; Varghese, 1934). Nagaraj and Menon (1956), and Radha and Lal (1968) observed that palms having latent infection of root (wilt) symptoms are easily infected by leaf rot pathogen. Occasional occurrence of the disease in nursery seedlings and young palms has also been noticed. Palms affected by leaf rot yielded an average of 70 per cent less than healthy palms (Radha *et al.*, 1962).

4.1 Symptoms

The earliest visible symptoms are the blackening and shrivelling of the leaflet tips of younger leaves. These affected tissues break off when dried and are blown off by wind leaving only the midrib giving a fan-like appearance to the affected leaflet. The central spindle also shows signs of infection. Reddish brown sunken spots appear on the spindle and tender leaves. These lesions enlarge in size and soft rot develops gradually. During dry weather, the decayed portions get dried, turn black and are blown off. Continuous attack of newly emerging spindle results in the gradual exhibition of similar symptoms in all leaves on the crown. Sometimes, the decayed leaflets are glued together so that the spindle does not open out (Fig. 3). Though the disease does not kill the palm outright, its slow progress in the crown causes steady decline in yield. The disease is seen in palms of all ages specially on palms below 25 years. Intensity of

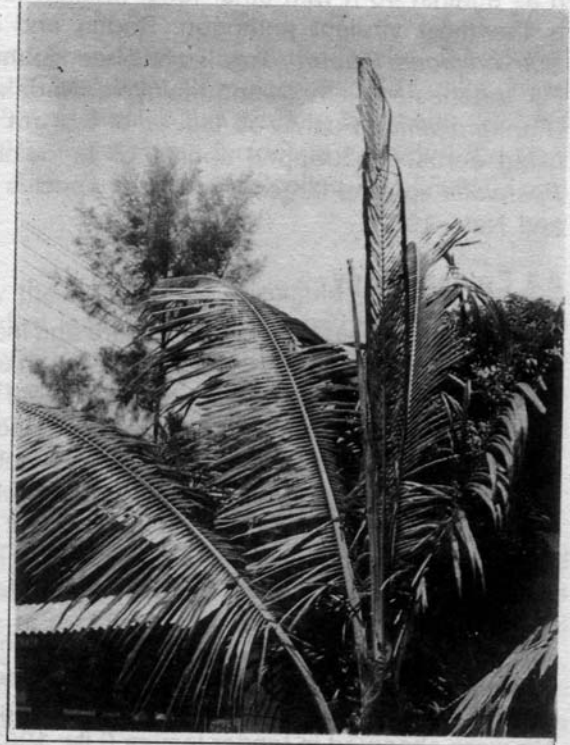


Fig. 3 : Leaf rot of coconut—decayed leaf glued together in the spindle.

← Fig. 2 : Stem bleeding disease of coconut—bleeding patches on the stem.

the disease increased with age of the palms and the advancement of root (wilt) disease (Radha and Lal, 1968).

4.2 Causal Agent

The disease is caused by *Bipolaris halodes* (*Helminthosporium halodes* or *Drechslera halodes*). In Papua New Guinea, *B. incurvata* causes the disease. *B. incurvata* has been recorded recently in Andaman Nicobar Islands and was found to affect both seedlings and adult palms (Rao, 1988). In the case of *B. incurvata*, the disease starts as a small yellow spot which enlarges in course of time. Slowly, the tissues in the centre of the lesion die and the lesion dries out. It results in severe retardation of growth and substantial loss of seedlings. Along with *B. halodes*, fungi like *Gloeosporium* sp., *Gliocladium roseum*, *Pestalotiopsis palmarum* are also often found associated (Radha and Lal, 1968). Pathogenicity trials showed that *B. halodes* induced infection within 12 h as against 48 h with other fungi (Menon and Nair, 1951) indicating that *B. halodes* is the most virulent pathogen. Radha and Lal (1968) also confirmed this finding. *Botryodiplodia theobromae* also has been found to cause leaf rot in countries like Brunei, Fiji, Indonesia, Vietnam and Malaysia. Nadakal (1968) reported a genus of nematode (*Panagrolaimus* Fuchs) on leaves of leaf rot affected palms. Menon and Nair (1948) using aeroscope trapped spores of *B. halodes*, *Gloeosporium* sp., *G. roseum* and *Pestalotia* sp. The largest number of species was present during May-August (Menon and Nair, 1952).

4.3 Epidemiology

High humidity, low temperature and increased dose of nitrogenous fertilizers are favourable for the disease incidence (Menon and Nair, 1951; Radha *et al.*, 1961; Radha and Lal, 1968). Low night temperature and leaf wetness either due to dew or rain favoured fungal infection. Anon. (1978) failed to demonstrate the toxic effect of culture filtrate on tender leaves. Potassic fertilizers were found to increase the resistance to this disease. George and Samraj (1966) found that leaf rot affected palms responded favourably to boric acid application suggesting involvement of boron deficiency. Tendermost leaf was found to be highly susceptible (Lily, 1963). The susceptibility of seedlings decreased with age. Seedlings up to 19 months old may get severe infection.

There was no significant difference in the amino nitrogen levels, ascorbic acid, total phenols or sugars (Anon., 1976) between leaves of healthy and leaf rot affected palms. However, higher moisture levels, total and non-protein nitrogen, P, K, Ca and Mg were observed in tender leaves (Lily, 1963). Lily and Ramadasan (1979) found that as a result of infection the total phenols increased in the leaves.

4.4 Control

Prasannakumari *et al.* (1960) reported that Bordeaux mixture was the most effective in controlling the disease. Nair and Radha (1959) and Radha (1961) found that regular manuring and spraying with copper fungicides checked the disease. By

regular spraying intensity of leaf rot could be brought down from 40 to 7.8% during 1976 to 1981 (Anon. 1984a). Aerial spraying of oil based copper fungicides was reported to be useful in controlling the disease (Gregory, 1960). However, Samraj *et al.* (1966) reported that aerial application of oil based copper fungicide did not control leaf rot as it did not adequately protect the spindle leaves. Spraying the leaves sequentially with Bordeaux mixture (1%), Dithane M-45 (0.3%) and Fytolan (0.5%) at quarterly intervals after removing all severely affected leaves, was found to reduce further incidence of the disease in Kerala, India (Anon., 1984a, 1985). Varieties like Karkar, Tahiti Island, Rangiroa, etc., are more susceptible (Subak, 1975; Quillec and Renard, 1975; Triharso, 1979). Radha (1961) reported that Andaman Ordinary and New Guinea were more tolerant to the disease. Mathai *et al.* (1985) found that among the 8 coconut varieties studied, leaf rot disease incidence was the least in Cochin China with Java and West Coast Tall being highly susceptible.

5. GREY LEAF SPOT

It is widespread in all the coconut growing regions in the tropics. It was first reported from British Guyana and later from Malaysia, New Hebrides, Sri Lanka, India, Trinidad, etc. (Menon and Pandalai, 1958). Recently, the disease has been reported from Nigeria. It is generally presumed that the disease incidence indicated the poor nutritional status of the affected palms. The disease is also called leaf blight.

5.1 Symptoms

The disease is characterised by the appearance of minute yellow spots with a grey brown margin on the outer whorl of leaves. The spots may be oval in shape measuring up to 5 cm length (Menon and Pandalai, 1958). The centre of the spots becomes greyish white while the brown colour of the margin deepens. Many spots coalesce to form large irregular necrotic patches. The leaves in advanced stage of infection present a blighted appearance and hence the name leaf blight.

5.2 Causal Agent

The causal fungus is *Pestalotiopsis (Pestalotia) palmarum*. The fungus is considered as a weak pathogen. On the upper surface of the leaf, the black pycnidia of the fungus appear as black minute specks. The rate of conidial germination were higher (93%) on the upper surface as compared to 80 per cent on the lower surface (Lingaraju *et al.*, 1987). The fungus needs light for sporulation. However, it sporulated in dark on a medium containing sodium chloride (Mani and Swami, 1983). The disease causes serious damage in nursery plants, but in adult palms the infection does not cause much damage. Rao *et al.* (1975) found that the infection was high during August-November period. Many species like *P. brevisata*, *P. gibberosa* and *P. stellata* associated with the leaf spot have been reported (Ohler, 1984). Fernando and Mahindapala (1977) could not establish the pathogenicity of *P. palmarum* in inoculation experiments. Brown (1975) found that the fungus could not infect uninjured leaves and colonised spots caused by *D. incurvata*. Ramanujam (1983) found that the pathogen produced a phytotoxin of low molecular weight and phenolics in nature.

5.3 Control

Since potassium deficiency increased the blight (Alonzo and Palomar, 1980), regular application of potassium chloride reduced the incidence (Abad *et al.*, 1978). Britton-Jones (1940) reported the disease could be kept under check by improving the growing conditions of the diseased palms by balanced fertilizer application, spraying Bordeaux mixture, providing adequate shade in the nurseries, etc. Das and Mahanta (1985) reported that carbendazim, thiabendazole and thiram completely inhibited grey blight pathogen. Bhaskaran and Ramanathan (1983) found that though none of the cultivars or hybrids tested was immune to the pathogen, however, some variation in susceptibility could be discernible. Abad *et al.* (1978) reported that 'Catigar' variety showed consistent tolerance to the disease.

6. MINOR FUNGAL DISEASES

6.1 Alternaria Leaf Spot

This disease was reported for the first time in Andhra Pradesh in India (Rao and Subramanyam, 1975). The disease is seen on 1-3 year-old coconut seedlings as black, round or oval spots. During summer, adjacent spots used to coalesce to form large patches. The causal agent is *Alternaria alternata* (Fr.) Keissler. The fungus infects *Capsicum annum* also. Recently, this disease has been observed in some coconut gardens in Northern Kerala (Nambiar, unpublished). Mishra (1987) recorded *A. tenuissima* from affected leaves of coconut. Prasad *et al.* (1985) found that amylic activity was noticeably higher in diseased coconut leaves than healthy.

6.2 Anthracnose

The disease is also called fruit rot. It is caused by *Colletotrichum gloeosporioides* Penz. (Almeida Jr. and Aquino, 1978). The disease is prevalent in Brazil affecting 1.5 to 41 per cent of the harvested fruits in some gardens. In India, *C. cingulata* and *C. paucisetum* are known to cause leaf spot (Mitra, 1929; Johnston, 1965). The symptoms on fruits include development of dark grey to brownish black lesions with an irregular margin. The lesions coalesce and cover large parts of the fruits. The affected fruits are desiccated and deformed, showing longitudinal ruptures causing rotting of internal parts. In Kenya, nut splitting due to *Colletotrichum* infection has been reported (cited by Menon and Pandalai, 1958).

6.3 Curvularia Leaf Spot

Radha and Menon (1954) reported *Curvularia* sp. from leaves of root (wilt) affected palms, while Subramanian (1953) recorded *C. inequalis* and *C. palmarum* from dead leaves of coconut. However, a leaf spot disease caused by *C. maculans* was noticed in Malaysia (Chan, 1974). The disease is characterised by appearance of small, circular yellow spots on leaves in the nursery seedlings. The spots gradually turned brown, with the centre of the spot drying up having a sunken impression. The disease is serious

in seedlings grown under shade and spreads rapidly under conditions of high humidity (Tey and Chan, 1980). Other predisposing factors are lack of adequate nutrition and moisture stress. Chan (1974) found that spraying the affected seedlings with captan or difolatan controls the disease. Malayan Yellow Dwarf is highly susceptible while Malayan Tall is resistant.

6.4 Exosporium Leaf Spot

This disease was first reported in the Philippines (Reinking, 1919) as caused by *Exosporium durum* (Johnston, 1965). In India, Mitra (1929) reported a leaf spot caused by *E. palmivorum*. The disease is characterised by presence of jet black wart-like fructifications on the leaf surface. The disease occurs only in very neglected gardens and seldom causes damage.

6.5 Fruit Rot

The disease is also called nut fall or 'Mahali' in Kerala, India (Sundararaman and Ramakrishnan, 1924). The casual organism is *Phytophthora palmivora*. The disease is characterised by decaying of immature nuts and their fall during the rainy season. Water-soaked greyish green area develops at the stalk end of the nuts against dark green healthy area around. The lesion development was more fast at the stalk end of nuts. The lesions later turn brown and become sunken due to decay of underlying tissues. The rot extends into the husk and sometimes deep into the endosperm cavity if the shell has not hardened.

The fungus *P. palmivora* was found to be MF₁, mating type A1 in Indonesia (Bennett *et al.*, 1986). Rate of lesion growth was inversely proportional to age, being 61.8 per cent and 39.3 per cent in 4 and 10 month-old nuts. Wounding was essential for successful inoculation. The fungal pathogen also causes bud rot.

The disease can be effectively prevented by giving a prophylactic spray with 1 per cent Bordeaux mixture during pre-monsoon period. Sometimes, when rainfall is heavy and continuous, a post-monsoon spray will be required. Adoption of phytosanitation measures and regular plant protection, cultural and fertilizer application practices help prevent the disease incidence.

In Karnataka, immature nut fall was also found to be caused by another fungus *Botryosphaeria rhodina* (Berk, & Curt) V.Ar. (Sulladmath and Shantappa, 1979).

6.6 Petiole Disease

The disease was first reported by Stockdale (1907) from Sri Lanka and in 1917 from Malaysia. The disease was later recorded from Papua New Guinea and New Caledonia (Dwyer, 1937; Shaw and Booth, 1967). Recently, Sulladmath and Ponnappa (1978) recorded the disease from Karnataka state in India.

The disease makes its appearance as dark, chocolate brown, irregularly circular or elongated lesions on the adaxial surface of the petiole. The lesions merge to form larger ones and spread over the entire petiole. Isolated raised blisters or irregularly raised patches appear. The tissues of the infected petiole rot. Such affected petioles break at the distal end of the affected patch and the broken dry fronds hang down. The disease does not cause serious damage.

The disease is caused by *Anthostomella cylindrospora* Booth and Shaw, and *A. fusispora* Booth and Shaw. A jet black stroma is produced below the surface of infected petiole. The asci are hyaline, stalked and elliptical, $100 \times 21 \mu\text{m}$ and contain eight elliptical ascospores of $31 \times 8.5 \mu\text{m}$ size. The ascospores are discharged when RH is 96-100 per cent at 24-28°C by breaking of ascus wall in the locules and extruded through the pores (Sulladmath and Ponnappa, 1978).

6.7 Spear Rot

The disease, also called shoot rot, is mainly seen in the nursery seedlings in India (Sundararaman, 1925). It has also been reported from Malaysia (Tey and Chan, 1980). Dark brown stripes are noticed on the spear or the next leaf. The lesions traverse rapidly downwards. The base of the spear decays and the spear snaps at this point on a slight pull. The bud remains intact. The causal agent is *Fusarium* sp. In Indonesia, *Diplodia* sp. has been reported to cause rotting of seedlings (Leefmans, 1934). Management practices like phytosanitation, avoiding overcrowding in the nursery and application of copper fungicides or carbendazim were found to be effective against the disease.

6.8 Other Minor Diseases

Brown (1973) and Anon. (1979) have given a list of fungi recorded from coconut. Anon. (1979) listed 173 fungi on coconut along with country of occurrence. Brown (1973) recording 35 fungi from coconut leaves, mentioned that leaf spots were of minor importance and that the fungi were present only on senescing leaves. Occasionally, they become serious. Sulladmath and Ponnappa (1979) reported three new Deuteromycetes viz., *Cladosporium cladosporioides*, *Botryosphaeria rhodina* and *Simatosporium falcatum* occurring on blighted leaves of drought-weakened palms. Weak parasites like *Phomopsis cocos*, *Asochyta cocoina*, etc., have been reported on coconut palm (Ponnappa, 1969). A new leaf spot disease caused by *Periconia saraswatipurensis* was described by Gupta and Asha (1971). The affected leaves characterised by presence of purplish brown circular to elongate spots, 2-6 mm diameter with fructifications below the epidermis. *Leiosphaerella longispora* isolated from collapsed fronds from Papua New Guinea (Sivanesan *et al.*, 1976) has been found to infect seed coconuts imported into India from French Polynesia (Raju and Reddy, 1984).

6.9 Post-Harvest Diseases

Many fungi have been found to attack the copra and spoil the quality. Some important fungi attacking copra are *Aspergillus niger*, *A. glaucus*, *A. flavus*, *A. fumigatus*

and *A. ochraceous* (Cooke, 1932; Ward, 1937), *Ceratostomella adiposum* (Thompson, 1933), *Botryodiplodia theobromae*, *Aspergillus* spp. and *Rhizopus oryzae* (Rao *et al.*, 1971) and *Penicillium frequentans* (Nair and Nathan, 1971). *Endosporostilbe* sp. (Patil and Kelkar, 1975) has been observed recently (Nambiar, unpublished) on the copra and shell of coconut. *Penicillium citrinum* (Susamma and Menon, 1983) and *Drechslera hawaiiensis* (Sharma *et al.*, 1985) are also known to attack copra. *D. hawaiiensis* enters the fruit while attached to the bunch itself. Under high moisture conditions, *Aspergillus* spp. rapidly penetrate into the copra meat causing its discolouration resulting in an increase in free fatty acid levels. Susamma and Menon (1983) reported that copra having 5 per cent moisture and kept in gunny bags lined with polythene sheet remained moderately free from infection. Susamma (1980) reported that *B. theobromae*, *Aspergillus* spp. and *R. oryzae* enhanced the cellulolytic enzyme activity in the affected copra. Susamma *et al.* (1980) found that monsoon periods favoured the development of both bacterial and fungal populations. Some workers observed the frequent presence of *Bacillus* spp. (Subramanyam, 1965; Paul *et al.*, 1980) while others noted that bacterial population was replaced by fungi when the temperature rose and moisture content of copra fell (Paul *et al.*, 1980).

7. DEFICIENCY DISORDERS

7.1 Crown Choke

This disease was first noticed in Assam in India during 1964 (Chakraborty *et al.*, 1970). It was then called 'crown rot'. The disease was later reported from West Bengal. In parts of Kerala and Tamil Nadu also, palms having similar symptoms have been noticed. A recent survey in Assam shows that about 10 per cent of the palms are affected in that state (Anon., 1989; George *et al.*, 1990). Palms in the age group of 3-6 years are generally affected. However, the disease has been found to occur in the adult palms also. Dwyer (1937) reported a disease called 'Fronde choke' with similar symptoms from New Guinea. Disease with similar symptoms has been reported from Indonesia, Ivory Coast, Sri Lanka and the Philippines (Jayasekhara and Longanathan, 1988). Reinking (1961) and Ohler (1984) stated that 'bristle top' disease has similar symptoms.

The symptoms of the disease have been described by Chakraborty *et al.* (1970). Recently, Rethinam *et al.* (1990) gave a detailed description of the disease. The first symptom is the emergence of shorter leaves with deformed and crinkled leaflets. These leaflets are associated with severe tip necrosis and their number decreased progressively when the attack is acute. The affected leaflets do not unfurl and in many cases give a choked appearance to the frond. The tip of the leaves become hooked; the hooks are mostly seen in the terminal pinna, although they may occur in any position on the frond (Baranwal *et al.*, 1989). As the disease progresses, a severely necrotic black, stick-like leafstalk devoid of any leaflet emerges. The outer whorl of leaves looks healthy and remain green. In due course, the inner leaves crowd around the bud, and normal unfurling of the flag leaf is prevented as a result. In advanced stage, necrosis of the primordial tissues takes place and the crown dies. The stem does not show any tapering.

The affected palms do not die suddenly, but it slowly loses vitality and finally succumbs within 3-4 years. Premature nut fall has been also noticed. The inflorescence emergence is hindered resulting in a gradual decline in yield. Finally, the affected palm does not produce any nuts.

No pathogenic fungi or bacteria could be isolated so far from affected palms. Analysis of soil and leaf samples show that the palms are deficient in boron (Anon., 1988; Baranwal, 1989). Plants grow normally only when a certain balance exists in the intake of Ca and B. The Ca/B ratio is significantly low for healthy palms (95) as compared to diseased palms (145) (Baranwal *et al.*, 1989). The leaves from diseased palms had 5.4 ppm B as against 7.4 ppm in healthy (Baranwal *et al.*, 1989). Cecil and Pillai (1978), and Anon. (1988) had also made similar observations of lower boron content in diseased leaves as compared to healthy palms. Manicot *et al.* (1980) and Wahid (1984) described symptoms of boron deficiency in coconut and these symptoms are similar to those of crown choke affected palms. Fremond (1965) reported that a form of bud rot prevalent in the New Hebrides might be due to boron deficiency. Pillai *et al.* (1975) reported that 12 per cent of coconut soils in Kerala are deficient in boron. Brunin and Coomans (1973) found presence of fused terminal leaflets and bayonet shaped leaflets in 18-20 month-old coconut seedlings having boron deficiency. In extreme cases, leaf stumping was followed by a stop in leaf production. Application of 15 g boracine (46% B₂O₃) per tree, 2-3 weeks after planting followed by 15 g 6 months later, controlled the malady (Brunin and Coomans, 1973). In the case of 'Frond choke', Dwyer (1937) thought that the disease may be due to physiological derangement, soil factors or even genetic factors.

Dwyer (1937) noticed natural recovery at times in the case of frond choke disease. Application of borax was found to improve the foliar conditions of affected palms (Chakraborty *et al.*, 1970; Cecil and Pillai, 1978). Application of borax at 50 g per palm during post-monsoon period is beneficial in improving the condition of the palm. Palms in the early stage of disease respond better (Anon., 1989). Chakraborty *et al.* (1972) described a surgical method of correcting 'frond bending' due to crown choke disease. Dwyer (1937) recommended a method of surgery to the hardened fronds to enable free emergence of shoots.

8. NATURAL DISORDERS

8.1 Lightning Injury

Death of coconut palms due to lightning strike during monsoon season in tropical countries is a common experience. Such natural calamities in coconut garden was first recorded in Sri Lanka (Petch, 1915) followed by Malaysia (Sharples, 1933), Papua New Guinea (Dwyer, 1937) and India (Menon and Pandalai, 1958). The disease is also called 'false bud rot'. Petch (1915) could observe that as many as 500 palms were struck by lightning in a year in Sri Lanka. The damage due to lightning strike might vary depending upon the intensity of lightning. It might set the palm on fire, or cause instant death. Splitting of the trunk or decapitation are also observed, though rarely. Less serious damage is

caused to the crown in milder cases (Ramaiah, 1990). Ramaiah (1990) observed a deep fissure of 1-2 cm depth running along the length of the affected trunk. The affected palms lose their vigour. The leaves of the affected palms are partially scorched; the outer leaves hang down. The petioles may show splitting. Die-back starts in older leaves first and gradually passes on to the inner whorl. The spindle and surrounding younger leaves are attacked last. Sometimes, the central spear might collapse after a flash showing no signs of charring. Within a few days, the leaves in all the whorls start drying up and the rachis turn brown giving a burnt-up appearance. The internal tissues of the affected bole show discolouration. The affected palms are present usually in a circular area around a focus. Seriously affected palms die first. The adjacent palms, which may have been less seriously affected, might take some more time before they succumb to the malady. These palms will show many fronds broken at their tips and hanging down (Shaw, 1968; Ramaiah, 1990). Such debilitated or dead palms are easily attacked by red palm weevil and rhinoceros beetle. Hence, dead palms are to be removed without delay to prevent infestation by the dreaded pests or colonisation by fungi like *Ganoderma* spp. Young palms (3-5 years old) or newly transplanted seedlings when struck by lightning may show breaking of the frond in the middle (Ramaiah, 1990). In young seedlings, the rotting of spindle was mainly due to attack by secondary invaders. The roots die off without showing any particular symptom. The palms which are not killed outright, show some salmon pink exudation from the stem in a few days. The internal tissues are rapidly fermented and this results in invasion by insects like *Xyleborus* sp., *Diocalandra* sp., etc.

8.2 Drought Effect

Coconut palm is affected sometimes by prolonged drought in certain parts of the world. Menon and Pandalai (1958) reported that drought affected coconut palms in Sri Lanka and India. In South India, prolonged drought conditions were observed recently in Karnataka, Kerala and Tamil Nadu (Rethinam, 1987).

External symptoms in adult palms affected by drought are characterised by yellowing, drying and drooping of outerwhorl of leaves. Gradually, the inner whorl of leaves also show yellowing and drying depending on the severity of drought. The leaflets tend to fold slightly. Reduced capacity of female flower production, shrinkage in the buttons formed and their shedding alongwith nuts of various stages of maturity are observed, resulting in reduced nut production. The unopened inflorescences are sometimes aborted. Since the older leaves droop down, the bunches subtended by these leaves lose the support, resulting in buckling of bunches and drooping of nuts later. In unirrigated gardens, wilting and death of young and adult palms are commonly seen in severe drought years. Where surface planting system has been adopted in coconut, the effect due to drought is much more severe than that in gardens where coconut seedlings are planted in pits of 1 m³. In drought-affected gardens, the attack by insects like scales and mealy bugs and diseases like *Ganoderma* wilt, grey leafspot, etc., is likely to be high.

Anon. (1984) and Rethinam (1987) described the measures to be adopted in managing drought affected gardens. These measures consisted of removal of 3-5 leaves

of the lower whorls to reduce transpiration, provision of drip irrigation, husk burial in the basins, cover cropping and mulching with dry leaves in the basins to reduce moisture loss from the soil, addition of organic matter to improve the water holding capacity of soil, addition of common salt at 2 kg per palm to keep the soil moist and controlling pests and diseases.

9. DISEASES OF UNCERTAIN ETIOLOGY

9.1 Button Shedding and Immature Nut Fall

In coconut, which is cross fertilized tree, a large number of female flowers generally do not get fertilized and these fall off as a natural phenomenon. In heavily bearing palms, nature itself provides some safety mechanism by which exhaustion of the tree is prevented which might cause shedding of buttons. Apart from this, many other factors play a role in shedding of buttons and immature nuts (Petch, 1913; Sampson, 1923; Gangolly *et al.*, 1959; Abeyawadana and Mathes, 1971). Child (1974) used the term abnormal nut fall for this malady. Prasad Rao (1988) observed maximum button shedding during North-East monsoon period in Kerala, India, followed by summer period.

Many fungi, insects, nematodes, nutritional deficiencies, physiological disturbances, unfavourable conditions, indirect effect of other diseases, etc., have been reported as possible causes of the malady. *Phytophthora palmivora* has been observed to infect nuts of all ages resulting in their fall during the South-West monsoon season in India. Other fungi reported as causal organisms are *Colletotrichum* sp. and *Botryodiplodia* sp. (Menon and Pandalai, 1958; Bennett *et al.*, 1986). Insect pests which are generally associated with nut fall are a moth (*Acritocera negligens*), a beetle (*Diocalandra taitensis*) and a coreid bug (*Amblypelta cocophaga*), etc. (Menon and Pandalai, 1958). Scales and mealy bugs have been noticed in increasing proportion in many coconut gardens in Tamil Nadu, causing premature fall of nuts. Rodents are also responsible for damaging nuts leading to their fall (Advani, 1984). Mineral deficiency is another factor. In palms affected by crown choke disease, premature nut fall has been noticed. This disease is caused by boron deficiency (Rethinam *et al.*, 1990). Application of borax has helped in reducing or controlling nut fall in these cases. Incidence of various diseases like lethal yellowing, root (wilt), basal stem rot, etc., results in abnormal nut fall. Koshy (1986) reported that severe infection by burrowing nematode (*Radopholus similis*) can cause nut fall. Soil moisture stress during drought period resulting in increase in abscission is another cause. Excessive moisture or water logging conditions in the coconut garden is also found to be associated with nut fall (Ohler, 1984). Thus, a number of factors may be involved either single or in combination in button shedding and immature nut fall. Hence, control measures also should be directed specifically to correct or avoid the primary causative factor.

9.2 Pencil Point Disease

The disease was first reported from Burma (Furtado, 1923). Later, the disease was observed in New Guinea, Sri Lanka and India (Menon and Pandalai, 1958). The

disease is also called tapering stem wilt (Jagannathan and Ramaswami, 1977; Bhaskaran *et al.*, 1979). Jagannathan and Ramaswami (1977) had recorded up to 40 per cent incidence in some gardens in Tamil Nadu, India.

The important symptoms include reduced vigour, stunted growth and lanky appearance. The older leaves become chlorotic first to be followed by other leaves in succession. The leaves of affected palms are reduced in size. The number of fronds and spathes will be reduced. The trees in the advanced stage of disease cease to produce any nuts. There is a gradual reduction in the girth of the stem towards the tip presenting the appearance of a 'pencil point'. In course of time, the growth ceases and the palm succumbs to the malady.

Jagannathan and Ramaswami (1977) could not find any pathogenic organism or insect to be associated with disease. Water logging and improper drainage were attributed to this disease (Menon and Pandalai, 1958) while Park and Fernando (1941) opined that the disease was due to senility. Nutrient deficiency was also suspected (Jagannathan and Ramaswami, 1977). Menon and Pandalai (1958) suggested provision of good drainage conditions in the garden and adoption of better management practices to control the malady.

Lewin *et al.* (1983) reported that application of micronutrients like boron, manganese sulphate and zinc sulphate along with NPK fertilizers helped in alleviating the symptoms.

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