Chapter 10
Coconut: Maladies and Remedies

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Abstract Coconut, a perennial palm with a life span of many decades, suffers from a number of biotic and abiotic stresses during various stages of its growth. Microbial pathogens including fungi, bacteria, viruses, viroids and phytoplasmas are known to be the causative organisms of these diseases. Among the 173 fungal species reported on coconut, only a few are lethal, while others cause economic losses of varying degrees. The causal agents of various diseases, formerly regarded as *diseases of unknown etiology*, have now been determined. While curative measures are available for some maladies like stem bleeding, prophylaxis is the best option for some of the lethal diseases like bud rot. Some of the debilitating non-lethal diseases have been found to respond favourably to nutritional management measures. Hence, adopting an integrated approach involving tolerant varieties, cultural, nutritional, prophylactic and phytosanitary measures is the best option for managing the diseases of coconut at present. In view of difficulties due to the tall stature of the palm, modern technologies like computer-aided, remote-controlled drones should be developed for plant protection operations. Remote-sensing methods using advanced technologies can help in regular surveillance for disease incidence, intensity and spread. Molecular techniques can also help in early diagnosis and in resistance breeding. Region-specific integrated disease management strategies for specific maladies have to be developed for attaining maximum efficacy in disease control. The distribution, symptoms, etiology, epidemiology and possible control measures of fungal, bacterial, virus, viroid and protozoan diseases affecting coconut are detailed in this chapter.

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10.1 Introduction

Various fungal, virus, viroid and protozoan diseases reported so far are listed in Table 10.1. Please refer to Chap. 11 for 'Phytoplasmal Diseases'.

10.2 Fungal Diseases

10.2.1 Bud Rot

10.2.1.1 Geographic Distribution

The first report of bud-rot incidence was from Grand Cayman, an Island in the West Indies, in 1834 (Tucker 1926). Since then, Quillec et al. (1984) reported the incidence from Sri Lanka, Indonesia, the Philippines, Colombia, Papua New Guinea, Vanuatu, Fiji, French Polynesia, the Dominican Republic and Côte d'Ivoire. Butler (1906) had first reported this disease from India.

10.2.1.2 Symptoms

Unopened tender leaf or spindle is affected leading to rotting of the terminal bud and death of palms. The first visible symptom is withering of the spindle marked by its pale colour. The spear leaf or spindle turns brown and bends down. Light brown specks are present on the petiole bases of the youngest leaf, and on those of the older leaves, large yellowish to brownish necrotic areas may be observed. The affected spindle can easily be pulled out as the basal portion of the spindle including the terminal bud is completely rotten, emitting a foul smell (Fig. 10.1).

10.2.1.3 Aetiology

Though primary infection is a dry rot caused by *Phytophthora palmivora*, Butl., subsequent colonisation by secondary invaders such as *Fusarium* sp. and bacteria like *Xanthomonas, Pseudomonas* and *Erwinia* results in wet rotting (Radha and Joseph 1974), with the affected tissues emitting a foul smell. The older leaves remain green and retain their position for several months, which is very characteristic of this disease (Ohler 1999). Even though the palm may not die immediately, it succumbs finally due to loss of the single apical bud. The older nuts persist on the crown for some time, while the younger ones may fall off (Radha and Joseph 1974). Later, the inner leaves also fall off one by one leaving only the outer whorl of mature leaves on the crown.

Table 10.1 List of diseases reported on coconut

Sl. no.	Disease	Pathogen/causal agent	Distribution	References
I. *Major fungal diseases*				
1.	Bud rot	*Phytophthora palmivora, P. heveae, P. katsurae, P. nicotianae, Fusarium moniliforme*, *F.solani*, *Graphium* sp.	West Indies, India, Côte d'Ivoire, Indonesia, Jamaica, Puerto Rico, Africa, Peninsular Malaysia and Philippines	Butler (1906), Tucker (1926), Menon and Pandalai (1960), Quillec et al. (1984), and Uchida et al.(1992)
2.	Fruit rot or mahali	*P. arecae, P. katsurae*	India, Sri Lanka and Côte d'Ivoire	Erwin and Ribiero (1996) and Quillec et al. (1984)
3.	Basal stem rot	*Ganoderma lucidum, G. applanatum, G. zonatum, G. boninense*	India, Florida, USA, South America, Java, Tropical Africa, Australia, Japan, Indonesia, Malaysia, Philippines, Samoa, Sri Lanka and Tasmania	Pieries (1974), Bhaskaran and Ramanathan (1984), and Satyanarayana et al. (1985)
4.	Stem bleeding	*Thielaviopsis paradoxa/ Chalara paradoxa*	Sri Lanka, India, Indonesia, Malaysia, Philippines, Fiji, Ghana, Trinidad	Petch (1906), Sundararaman (1922), and Britton-Jones (1940)
5.	Leaf rot	*Exserohilum rostratum/Colletotrichum gloeosporioides/Fusarium solani F. moniliforme*	India	Varghese (1934), Menon and Pandalai (1960), Radha and Lal (1968), and Srinivasan and Gunasekaran (1999)
6.	Grey leaf blight	*Pestalotiopsis palmarum*	Guyana, India, Malaysia, New Hebrides, Sri Lanka, Trinidad, Nigeria	Copeland (1931), Cook (1971), and Holliday (1980)
7.	Leaf blight	*Lasiodiplodia theobromae*	India	Johnson et al. (2014)
II. *Virus disease*				
8.	Coconut foliar decay or Vanuatu wilt	*Coconut foliar decay virus (CFDV)*	Vanuatu	Calvez et al. (1980) and Randles et al. (1986)
III. *Viroid diseases*				
9.	Coconut cadang-cadang disease	*Coconut cadang-cadang viroid (CCCVd)*	Philippines	Randles (1975)
10.	Coconut tinangaja	*Coconut tinangaja viroid (CtiVd)*	Guam on Mariana Islands	Boccardo (1985)

(continued)

Table 10.1 (continued)

Sl. no.	Disease	Pathogen/causal agent	Distribution	References
IV. *Protozoan disease*				
11.	Fatal wilt or heart rot	*Phytomonas staheli*	Central America (Costa Rica, Honduras and Nicaragua), South America (Brazil, Colombia, Ecuador, Guyana, Peru, Surinam and Venezuela) and the West Indies (Grenada, Trinidad and Tobago)	Waters (1978)
V. *Other minor diseases*				
12.	Lethal bole rot	*Marasmiellus cocophilus*	Kenya and Tanzania	Jackson and Firman (1982) and Jackson and McKenzie (1988)
13.	Anthracnose	*Colletotrichum gloeosporioides*	Brazil, India	Almeida et al. (1978)
14.	Leaf spots	*Bipolaris incurvata*	Hawaii, Florida, Jamaica, Asia, Australia, Oceania (French Polynesia, Fiji), Philippines, Seychelles	Uchida and Aragaki (1991)
15.	Algal leaf spot	*Cephaleuros virescens, Cephaleuros parasiticus*	Hawaii	Ploetz et al. (1999) and Harrison and Jones (2003)
16.	Thread blight	*Pellicularia filamentosa, Corticium penicillatum*	Sri Lanka, Fiji, Papua New Guinea, Samoa and Solomon Islands in Oceania	Kohler et al. (1997)

Sometimes the same bud-rot pathogen was found to infect coconut fruits and cause fruit rot 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 gets detached from the bunch. Nut drop and rotting are commonly seen during rainy season.

10.2.1.4 Epidemiology

Bud-rot disease is favoured by conditions of high humidity such as those found in low-lying badly drained lands, in plantations with a very dense stand and under extensive rainfall. Disease development is related to relative humidity (RH) (Menon

Fig. 10.1 Bud-rot disease. (Photo: Jacob John)

and Pandalai 1960; Darwis 1992). Rainfall aggravates the infection and young palms in the low-lying and moist conditions are more susceptible (Thevenin et al. 1992; Brahmana et al. 1992; Pohe 1992; Steer 1992). In the Philippines, Rillo and Paloma (1989) found higher incidence of *Phytophthora,* which was always preceded by high rainfall during previous months. Even though palms of all ages are susceptible to the disease, it was more rampant in palms which are below 20 years. The period elapsing between the first infection and withering of heart-leaf depends on a number of factors, the more important of which are RH and the point of infection. This period might vary from 3 to 9 months or even longer. With the onset of dry weather, the infection becomes less severe and pathogen remains dormant. The fungus was found to survive in the frond base or basal part of the crown (Menon and Pandalai 1960; Radha and Joseph 1974) or in roots (Harris et al. 1984). Seasonal factors are found to be associated with the incidence and spread of the disease. Disease incidence is found to be severe during monsoon when the RH is high. Studies conducted at ICAR-CPCRI, Kayamkulam, India (Radha and Joseph 1976), revealed that the favourable period of infection is when the RH is above 94% and temperature is below 24 °C. The rate of disease development was determined by the number of preceding favourable days. In young palms, between the ages of 5 and 10 years, the occurrence of favourable days is more frequent, and hence, more disease incidence was noticed in such young palms (Radha and Joseph 1974). An infection cycle of *P. palmivora* in coconut is completed within 6 days under favourable conditions of humidity (98–100%) and temperature (22–24 °C). Inoculation of young seedlings (1–2 years old) grown in pots and provided with these conditions resulted in infection and death of seedlings. It took 1 week for the fungus to complete its life cycle from sporangia to manifestation of the disease symptoms (Radha and Joseph 1974). Coconut is a sun-loving crop, requiring high solar radiation and

high humidity. However, high rainfall intensity in certain areas causes low solar radiation and high air humidity.

Bud-rot disease incidence was high in the hilly tracts where cooler temperatures coupled with very high RH prevailed for extended periods in the coconut crown because of the altitude of the location. The cooler post-monsoon weather also favours the formation of dew for extended periods, and this factor is conducive to the development of fresh disease incidence in the hilly tracts compared to the plains. In the plains, disease incidence was recorded only up to September. Initial incidence of bud-rot disease is always dependent upon the monsoon showers. However, the occurrence of bud rot in subsequent months, i.e. from October to January, could be attributed to the favourable microclimatic conditions inside the crown with consistently high humidity, low night temperatures and the presence of dew-water droplets (Rasmi et al. 2004).

10.2.1.5 Dissemination of the Pathogen

Epidemiological models applied to plots affected by *Phytophthora* in Côte d'Ivoire suggested the existence of two propagation phases, an aggressive phase during which transmission occurred from palm to palm and a regular phase during which new cases appeared some distance away from initial foci (Renard and Darwis 1993). The disease is primarily disseminated by wind and windblown raindrops and to a lesser extent by insects, birds and climbers. The disease spreads over large areas due to the influence of environmental condition in the plots. The wind pattern and direction are required to correctly interpret the disease spread. Dauzat and Lecoustre (1992) reported that there are two phases for bud-rot disease propagation. One is the 'cluster' phase during which contamination would be seen primarily to spread around contamination foci, and another is a regular phase during which new contamination occurs from foci. Rainwater acts as a carrier for the infectious propagules and plays an important role in the spread of the disease (Brahmana et al. 1992; Thevenin et al. 1992). Insects also spread the pathogen over large areas by moving from one place to another; their legs and mandibles seem to be helpful in transferring the propagules (Pohe 1992). Radioactive tracer studies have shown that certain flying insects, beetles, caterpillars and even snail species are involved in the spread of the pathogen. Evans (1973) reported two species of *Sogatella* as vectors of dry bud rot of young coconuts in Côte d'Ivoire.

10.2.1.6 Disease Management

Control measures are effective only when they are adopted in the initial stages of the disease. If the disease is detected when the central shoot just withers, chemical control by the application of 10% Bordeaux paste on the affected portion can check the disease. Bordeaux paste has to be applied after thorough cleaning and removal of infected material. The treated portion should be given a protective covering of plastic sheet to prevent washing off of the paste by rain. As a prophylactic measure,

adjacent healthy palms should also be sprayed with 1% Bordeaux mixture. Other fungicides advocated earlier against bud-rot disease include phenylmercuric urea applied in the form of powder or pellets in the leaf axils (Pieris 1962) or stem injection or root infusion of Aliette or Ridomil or Akomin (phosphonic acid). According to Schutt (1975) and Nambiar and Rawther (1993), regular spraying with copper fungicides at 40-day intervals, especially before and after monsoon, was found to be an effective preventive measure. In copper-sensitive palms, keeping perforated sachets containing Dithane M-45 in the leaf axils during rainy season is useful. Radha and Joseph (1974) found that Demosan (1200 ppm) effectively checked infection in laboratory tests. Renard and Quillec (1984) and Brahmana et al. (1992) reported that injection of the coconut trunk with systemic fungicides like Aliette (fosetyl-Al) and Ridomil (metalaxyl) at 3 g a.i. palm^{-1} was effective in protecting the palms from bud rot caused by *P. heveae*. Rohini Iyer (1997) found that stem injection/root feeding with systemic fungicides like aureofungin-sol (46.5%) 36.4 g palm^{-1}, Calixin (tridemorph 80%) 21 ml palm^{-1}, Aliette (fosetyl-Al 80% wp) 21 g palm^{-1} and Akomin (phosphonic acid) 16.8 ml palm^{-1} can protect the crown from *Phytophthora* attack for a period of 8 weeks.

Another long-term method of control is through the identification of resistant genotypes and breeding for resistance/tolerance. Characterisation of resistant palms can be made by assessing the field performance of planted varieties. Quillec et al. (1984) observed that MAWA hybrids were less sensitive than West African Tall (WAT). Brahmana and Kelana (1988) found that in Indonesia, dwarf palms are more sensitive while others are more tolerant. Among dwarf varieties, Nias Yellow Dwarf (NYD) seems to be the most susceptible one (Bennett et al. 1986). Bud rot is also observed predominantly in areas planted with PB-121 (MYD × WAT) coconut hybrids. Exotic talls such as Rennel Tall and local Indonesian talls are more tolerant (Renard and Darwis 1993). Malayan Yellow Dwarf × West African Tall hybrids are highly susceptible to bud rot in Indonesia (Mangindaan et al. 1992). Hybrids of Malaysian Yellow Dwarf × Rennel Tall were found to be less affected than PB-121. The NIWA hybrids obtained by crossing NYD × WAT are susceptible to bud rot. Rillo and Paloma (1989) found that *Phytophthora* incidence was higher in Cameroon Red Dwarf and MYD compared to Catigan. Coffey (1990) reported that in Indonesia, dwarf selections such as *Jombang* and *Raji* appeared to be more resistant to nut fall in inoculation trials.

In Asia, local ecotypes are generally more tolerant of *P. palmivora* than introduced ones, although the Polynesian Tall and Rennel Tall are less severely affected than the Bali Tall in North Sumatra. Malayan Red Dwarf (MRD) is susceptible to bud rot caused by *P. katsurae* in Côte d'Ivoire and is also susceptible to the same damage in Jamaica, whereas Red Dwarf × West African Tall coconut hybrids are more tolerant to bud rot than the Red Dwarf parent. Both Malayan Red Dwarf and West African Tall were susceptible to bud rot, whereas the Malayan Yellow Dwarf (MYD) and the Polynesian, Rennel and Malaysian Talls are highly tolerant in Jamaica. In the Philippines, MAWA hybrid (Malayan Yellow Dwarf × West African Tall was found to be susceptible to bud-rot infection. For reducing economic loss, it is recommended that the choice of planting material should be done by taking into account the environmental conditions also (Mangindaan et al. 1992).

Proper spacing among the palms is important for the management of the disease. Too close planting encourages rapid spread of the disease. A good spacing between palms favours air movement and dissipation of the excess humidity that can build up in the crowded gardens (Ohler 1984). Lowlands with generally high humidity are very favourable for the development of the disease, especially when the drainage is poor.

Organic matter application favours the growth of a variety of microbes including antagonists such as *Trichoderma* and *Gliocladium* spp. which multiply on them and help in reducing the population of soilborne pathogens like *Phytophthora.* Biological control is geared towards identifying microorganisms effective against *Phytophthora.* Regular use of site-specific fungicide is not recommended because fungicide-resistant isolates or strains of *Phytophthora* spp. may develop due to their continued use (Cohen and Coffey 1986). Copper injury, especially to certain dwarf varieties (Schutt 1975), high cost of fungicides and lack of trained labourers for spraying are constraints in advocating fungicidal control for bud rot. Though many bacteria, actinomycetes and fungi are antagonistic to *Phytophthora*, their activity in the field is limited. The use of microbial antagonism against *Phytophthora* is an important component of disease control (Malayczuk 1983; Shea and Broadbent 1983). However, the prospect of using biological antagonism is still uncertain, especially on a practical short-term basis (Shea and Broadbent 1983). The development of effective biological control of *Phytophthora* species has been fraught with difficulties because of their ability to produce several forms of inoculum (zoospores, sporangia, chlamydospores and mycelium) rapidly and repeatedly, wide host range, ability to penetrate and infect a host plant within a few hours and to exist in soil at depths of even 1 metre allowing them to escape most of the antagonists. Several genera of bacteria, actinomycetes and fungi have been shown to parasitise and lyse *Phytophthora* propagules in soil (Sutherland et al. 1984). These antagonists exert a lytic effect on mycelium, chlamydospores and oospores. Among the different fungi isolated from endemic plots, *Trichoderma harzianum* and *Trichoderma viride* were identified as the most effective antagonistic fungi of the bud-rot pathogen, *P. palmivorain vitro*. However, the result of pot experiments revealed that *T. harzianum* has a higher competitive saprophytic ability in soil compared to that of *T. viride.* Moosa et al. (1998) reported the occurrence of endophytic *Bacillus amyloliquefaciens* antagonistic to *Phytophthora palmivora* in coconut seedlings. The major problem for the use of fungicides is that crops like black pepper and cardamom are grown as intercrops in coconut gardens, whose export is highly sensitive to pesticide residues. Chemical control, though effective, is undesirable as it pollutes the environment, and the residues left in the products are hazardous to human health. To minimise the use of pesticides, biological control becomes imperative in integrated management of the disease. At present, there is a need for an effective, broad-based integrated control of *Phtyophthora* disease, involving the use of resistant varieties, improved cultural practices, new translocation fungicides and effective biological control methods (Tuset et al. 1984, 1992).

10.2.2 Basal Stem Rot

Basal stem rot is one of the most destructive diseases of coconut occurring in various coconut-growing regions of the world. The disease is known in India by various names such as '*Ganoderma* wilt' in Andhra Pradesh, 'Anabe' in Karnataka (Venkatarayan 1936; Rao et al. 1966) and as 'Thanjavur wilt' in Tamil Nadu (Vijayan and Natarajan 1972).

The disease has been reported from various places all over the tropical world, viz. India, Sri Lanka, West Indies, Seychelles and Guam. The disease was first reported by Coleman in 1911 on areca nut palms in Mysore, India. Pieries (1974) reported the disease as 'basal stem rot' from Sri Lanka. A severe outbreak occurred in 1952 in Thanjavur district of Tamil Nadu, India, and hence, the name 'Thanjavur wilt', although the disease is noticed in all districts of Tamil Nadu. Bhaskaran and Ramanathan (1984) found that the disease incidence ranged from 0.6% to 4.9% in Tamil Nadu. In the severely affected gardens, the disease incidence was as high as 31.4% (Bhaskaran et al. 1984). Apart from Tamil Nadu, the disease is also reported from Andhra Pradesh, Karnataka and Kerala in India. A disease with similar symptoms has been noticed in some parts in the Indian states of Maharashtra and Gujarat also.

10.2.2.1 Symptoms

Though the root system is affected, visible symptoms are seen in the crown as wilting of leaflets, similar to those of severe drought. The outer whorl of leaves turns yellowish, then gradually becomes brown and droops down from their point of attachment and hangs vertically downwards to form a skirt around the trunk apex. The apex of the trunk shows tapering with the advancement of the disease, and bleeding symptoms may appear on the bole region. The drooping leaves fall off one by one leaving only a few leaves at the apex. The crown is easily blown off by wind (Bhaskaran et al. 1984), leaving only the decapitated trunk. Male flowers become necrotic starting from the tip and spreading to the base of the spikelets. The few female flowers are also poorly developed. In the early stages, there is no button shedding seen. As the disease progresses, normal development of flowers and bunches is fully arrested. Most of the palms bear profusely just at the time of initiation of visible symptoms. As the leaves droop down, the subtended bunches also hang down. The nuts become barren and gradually the production stops. The roots are first affected and destroyed. The cortical region of the affected roots turns brown first, then the stele and the roots become friable and disintegrate. As the roots in contact with the soil die back, the palm puts forth new roots from higher up the trunk and sometimes new roots may be seen coming from healthy tissue piercing through the affected tissue. At the base of the stem, a characteristic reddish brown discolouration develops, accompanied by the exudation of a brown viscous gummy

substance. Initially these bleeding patches appear on several places as parallel vertical streaks. They soon coalesce, forming a discoloured band around the trunk. These brownish patches may extend up to 1 m from ground level. Occasionally, infected palms do not show any bleeding patch.

Symptoms of dry rot of internal tissue at the base of the stem are characteristic. Transverse and longitudinal sections in these areas show a light brownish rotting tissue marked by darker bands, often with an irregular outline. The edge of the lesion is marked with a distinct yellow margin 0.5–1.0 cm wide. The bole decays rapidly resulting in the formation of large cavities. In some palms, the bark from the base of the palm peels off. The sporophores (fruiting bodies) of *Ganoderma* appear as 'brackets' at the base of the trunk, generally after the death of the palm (Fig. 10.2) (Bhaskaran et al. 1989), and in some palms, just above the soil level. Usually it takes from 6 to 24 months for the affected palms to die.

10.2.2.2 Aetiology

In India, *Ganoderma lucidum* (Leyss.) Karst. was first reported to be the causal agent of the disease (Rao et al. 1966). However, later reports (Bhaskaran et al. 1989) showed that *G. applanatum* (Pers.) Pat. is also involved. Pieries et al. (1975) reported *G.boninense* Pat. as the inciter of basal stem rot in Sri Lanka.

The fungus is a soil dweller inhabiting dead as well as living plant material in the soil. It is a root parasite entering the host through wounds. The spread of the disease takes place mainly through soil and through root to root contact.

The fruit body (basidium) is bracket-shaped, perennial, stipitate, lateral and sometimes sessile. Its size varies from less than 2 to more than 50 cm in diameter.

Fig. 10.2 Basal stem rot disease. Note the fruiting bodies. (Photo: Jacob John)

The thickness of the fruiting body likewise varies up to 13 cm. The upper surface of the bracket is shiny, oxblood in colour, with solid thick-walled covering and concentrically furrowed. The shiny surface of the cap and stalk is most characteristic. When examined closely with naked eye or hand lens, numerous minute holes or pits will be seen all over the undersurface. It is in these tiny pores that the fungus produces its 'basidiospores'. The fungus is heterothallic and tetrapolar. The hyphae are hyaline, 1–2 μ in diameter covered with a deposit of calcium oxalate crystals. Clamp connections occur profusely in older hyphae. The fungus has a wide host range and attacks a variety of palms and several forest, avenue and fruit trees as well. According to Naidu et al. (1966), hosts belonging to 19 families, 36 genera and 48 species have been recorded. Some of these are *Areca catechu*, *Cocos nucifera*, *Cassia siamea, C. javanica, Pongamia glabra*, *Eucalyptus* spp., *Azadirachta indica*, *Morus alba, Artocarpus fraxinifolius*, *Acacia arabica*, *Casuarina equisetifolia* and *Dalbergia sissoo*. Papa Rao and Rao (1966) reported low frequency of incidence of the disease in heavy soils of Andhra Pradesh, probably due to their high moisture retention capacity. Pieries et al. (1975) also found that the disease progressed rapidly in dry areas, the destruction of the palms being faster (6–30 months) in areas receiving less rainfall (up to 1000 mm year^{-1}), as compared to areas receiving 2000 mm rainfall year^{-1}. Lewin et al. (1983) reported that soil moisture stress during the summer predisposed the palms to infection. A positive correlation between mean, maximum temperature and the number of bleeding patches has been observed by Jagannathan and Ramaswamy (1975).

Though the disease is prevalent in palms of all ages, Vijayan and Natarajan (1972) reported that palms in the age group of 10–30 years were more susceptible. Linear spread was found to be influenced by low rainfall and RH (Ramapandu et al. 1981). Since diagnosis of the disease at a very early stage is essential for taking up effective control measures, work has been done using colorimetric methods (Natarajan et al. 1986) or by methods employing physiological parameters like transpiration rate and stomatal resistance (Vijayaraghavan et al. 1987), which have given encouraging results. A diagnostic method using fluorescent antibody technique developed against *Ganoderma* disease in areca nut by Koti Reddy et al. (1984) is being employed in coconut also (Sampath Kumar and Nambiar 1993).

10.2.2.3 Disease Management.

On-farm trials conducted in Tamil Nadu and Andhra Pradesh states of India showed that the following integrated approach was very effective for containing the disease (Bhaskaran et al. 1989):

1. Removal of dead palms and palms in advanced stages of the disease and destruction of the boles and root bits of the diseased palms.
2. Isolation of neighbouring healthy palms by digging isolation trenches of 50 cm × 50 cm around the diseased palm.
3. Soil drenching with 40 l of 1% Bordeaux mixture thrice a year for 1 year.

Table 10.2 Performance of coconut cultivars and hybrids in *Ganoderma*-sick soils of Tamil Nadu, India

Sl. no.	Cultivars	No. of palms field planted	Percent survival	Mean nut yield (palm year^{-1})
1.	San Ramon	15	33.3	36
2.	Laccadive Ordinary	15	6.7	36
3.	British Solomon Islands	12	8.3	98
4.	Java Giant	15	26.7	80
5.	Straits Settlement Green	15	46.7	49
6.	WCT × COD	15	40.0	86
7.	COD × WCT	15	26.7	121
8.	VHC-1	10	10.0	66
9.	East Coast Tall (ECT)	10	40.0	86
10.	ECT × wilt-tolerant ECT	18	66.7	122

4. Addition of 50 kg farm yard manure or green leaf manure or 200 kg tank silt palm^{-1} year^{-1}.
5. Chiselling off the affected tissues and applying aureofungin-sol or Calixin (tridemorph) 5%.
6. Raising *Ganoderma*-resistant crops like banana as intercrop wherever irrigation is possible.
7. Root feeding of 2 g of aureofungin-sol and 1 g of copper sulphate in 100 ml of water thrice a year at quarterly intervals. Alternatively, use tridemorph (Calixin 5 ml in 100 ml of water). Fungicide treatments will be effective only for palms in early stages of the disease.
8. Ploughing and flood irrigation are to be avoided to prevent the spread of infective propagules. Irrigation through drip or channel is recommended.
9. Application of neem cake at 5 kg palm^{-1} year^{-1}.
10. Application of 500 g of *Trichoderma harzianum* multiplied in 50 kg farm yard manure as biocontrol.

Screening of 6 cultivars and 4 hybrids for their reaction to basal stem rot revealed that the hybrid East Coast Tall × wilt-tolerant ECT is more tolerant to basal stem rot, with 66.7% survival, compared to other cultivars (Bhaskaran et al. 1984). The details are given in Table 10.2.

10.2.3 Stem Bleeding

It is a debilitating disease prevalent in India, the Philippines, Malaysia, Trinidad, Fiji, Ghana, Papua New Guinea, Indonesia, Bangladesh, Sri Lanka, Mexico and many other coconut-growing countries. The disease was first reported from Sri

Lanka (Petch 1906) and later from India (Sundararaman 1922). Subsequently, its occurrence has been reported from Brazil (Warwick et al. 2009) and Hainan, China (Yu et al. 2012), although there is no report on the exact extent of loss due to this disease. The typical symptom is the oozing out of reddish brown liquid through the growth cracks on the trunk which later turns black on drying forming encrustation with brownish orange margin (Fig. 10.3). Bleeding patches progress both upwards and downwards and cover major portion/part of the trunk.

10.2.3.1 Aetiology

The aetiology of the disease was not established for a long time. Lilly (1984) isolated *Phomopsis coconina* Cke. (Punith) and *Schizophyllum commune* Fr. from the stem-bleeding-affected palms. However, their pathogenicity could not be proved in spite of repeated attempts. Although Menon and Pandalai (1960) suspected *Thielaviopsis (Ceratocystis) paradoxa* (de Synes) as the incitant of stem bleeding, it was Nambiar et al. (1986) who established *T. paradoxa* as the aetiological agent of the disease, through artificial inoculations. Later, the perithecial stage, *Ceratocystis paradoxa* (Dade) Moreau, of the causal agent was isolated from infected palms (Ramanujam et al. 1993). *T. paradoxa* produces pale brown to brown hyphae. Conidiophores are slender, arising laterally from the hyphae and producing cylindrical to oval endoconidia. When mature, they are hyaline to pale brown and smooth-walled (6–24 × 2–5.5 μ). Chlamydospores are also formed terminally in chains and are obovate, thick-walled, brown and 10–25 × 7.5–20 μ in size. The perithecial stage is *Ceratostomella* (*Ceratocystis*). Perithecia are partly immersed,

Fig. 10.3 Stem-bleeding disease of coconut. Note the oozing out of reddish brown liquid in the trunk. (Photo: Jacob John)

light brown and 190–350 μ in diameter with numerous appendages; long and black necked, tapering up to 1400 μ, osteolar and hyaline; and ascospores ellipsoid, often with unequally curved sides, hyaline, nonseptate, smooth and 7–10 × 2.5–4 μ. The optimum temperature for mycelial growth was reported to be 30 °C (Nishita Naik 1990). As many workers failed to isolate the fungus from infected trees, Anil Kumar and Nambiar (1991) developed a simple and highly reproducible technique for isolating *T. paradoxa* from the diseased tissues. A baiting technique was also developed for the isolation of *T. paradoxa* from the infected soils using sterile frond bits as baits. Gowda (1987) recorded variability among the 5 isolates derived from 5 localities in Karnataka and Kerala, India, with regard to colour, nature of colony, growth rate and conidial and chlamydospore production on various media. Nisitha Naik (1990) has demonstrated the variability among 7 isolates based on their optimum temperature, pH, carbon, nitrogen and vitamin requirements. Some of the isolates exhibited a characteristic fruity (pineapple) smell. Ramanujam et al. (1996) distinguished 2 sub-groups among the 12 isolates of *T. paradoxa* based on their pathogenic reaction to detached coconut petioles and also corresponding to their electrophoretic reaction.

All the 26 coconut cultivars (16 Talls, 6 Dwarfs and 4 hybrids), which were tested for susceptibility to *T. paradoxa* using the detached petiole technique, were found to be susceptible with varying degrees of disease intensities (Ramanujam et al. 1996). Banawali Green Round, Banawali Brown Round and Malayan Orange Dwarf were less susceptible, while Malayan Green Dwarf, Chowghat Orange Dwarf and Philippines Ordinary cultivars were more susceptible.

10.2.3.2 Epidemiology

Nambiar and Sastry (1988) reported that development of growth cracks, poor drainage, soil moisture stress, hard pan formation in soil, imbalanced nutrition, excessive soil salinity, stem injury, lightning and insect attack by *Diocalandra* and *Xyleborus* are the predisposing factors responsible for disease development as this fungus is a weak soilborne pathogen. It enters the coconut stem tissue through growth cracks and wounds and multiplies in the host tissue, producing endoconidia and chlamydospores, which survive in the soil during unfavourable weather conditions. When the conditions are favourable, the chlamydospores in the soil germinate and become capable of infecting coconut. Radha (1962) showed that fluctuations in soil moisture or ill-drained soil conditions could cause severe stem-bleeding disease. Mathew and Ramanandan (1980) could not find any significant differences in major nutrient contents between healthy and diseased palms. They also did not find any relation between disease incidence and soil pH and electrical conductivity. Nambiar and Ramanujam (1993) reported that chlorine deficiency does not seem to be a contributing factor for stem-bleeding disease in India, especially in Kerala, India, where on the banks of backwaters, this disease is noticed. Nambiar et al. (1989) studied the

conditions required for infection through artificial inoculation and found that the disease development was faster during July–November when high humidity and optimum temperature prevailed. Usman (1988) reported that maximum survival of *T. paradoxa* propagules was noticed in red loam soil followed by laterite soil and the least was noticed in sandy soil. The fungus attacks a wide variety of hosts like areca nut, palmyrah palm, date palm, banana, pineapple, sugarcane and papaya.

10.2.3.3 Disease Management

Prior to confirmation of the aetiological agents of the disease, control measures recommended mainly consisted of phytosanitary practices involving removal of affected bark tissues with a chisel and application of hot coal tar or Bordeaux mixture to protect the wound. Nambiar and Sastry (1988) reported improvement of palm conditions when carbendazim or tridemorph was root-fed. Further studies also indicated effectiveness of these chemicals in reducing the disease intensity and increasing the yields (Anil Kumar et al. 1992; Ramanujam et al. 1993). Their results also helped in the detection of residues of carbendazim in the stem along the feeding site only, while tridemorph was detected on the feeding side as well as on the opposite side of coconut trunk. No residues of carbendazim and tridemorph were detected in the nut water, in the palms, which received 5 g and 8 ml of chemicals, respectively (Ramanujam et al. 1993).

Since wounds on the trunks predispose the palms to infection, care should be taken not to injure the palms while doing cultural operations in the coconut garden. Providing summer irrigation and conserving the soil moisture by adopting suitable conservation practices are beneficial in reducing growth cracks. Application of neem cake (5 kg palm^{-1} year^{-1}) was found to increase soil microflora including *Trichoderma* population, which was found inhibitory to the pathogen in vitro (Gowda 1987) and on detached coconut leaf petiole (Usman 1988). They identified *Trichoderma viride*, *T. harzianum* and *Aspergillus niger* as potential antagonists to the pathogen. Later, Ramanujam (1997) identified *Gliocladium virens* as the most effective antagonist against *T. paradoxa* and also recorded rice bran neem cake (1:1) as the best substrate for mass production of this biological control agent.

Ramanujam (1997) also developed integrated management practices for effective management of stem bleeding of coconut involving root feeding of 5% tridemorph (100 ml at quarterly intervals) and wound dressing (50–200 ml) of tridemorph (4%) followed by coal tar sealing (100–300 g) and soil application of *Gliocladium virens* (1 kg), neem cake (5 kg), FYM (50 kg) and NPK fertiliser (500:320:1200 g palm^{-1} year^{-1}). Root feeding with hexaconazole (2 ml in 100 ml water at quarterly intervals) and wound dressing with the same fungicidal solution have been advocated. Application of paste of *Trichoderma harzianum* talc formulation on the bleeding patches on the trunk was also effective in preventing the spread of stem bleeding.

10.2.4 Leaf Rot

Radha (1961) first coined the name 'leaf rot' for foliar necrosis of coconut frond in the root (wilt)-affected tract of southern Kerala, India. Since the beginning of the last century, it was well established that the palms affected by root (wilt) disease are generally superimposed by leaf rot disease (Sundararaman 1925; Varghese 1934; Nagaraj and Menon 1956; Srinivasan 1991). In the palms weakened by the root (wilt), *Phytoplasma* might result in the breakdown of their defence mechanism leading to susceptibility to leaf rot disease. Crop loss due to leaf rot alone was not available earlier, as it is superimposed on root (wilt). Menon and Nair (1948) estimated the loss due to leaf rot as 5.6 million nuts annually. This is besides the loss in quality of the leaves rendering them unfit for thatching and other purposes. The loss due to leaf rot has been computed at 461 million nuts in Kerala, India, as it is prevalent in 0.41 million ha (Srinivasan and Gunasekaran 1999).

10.2.4.1 Symptoms

Leaf rot starts as minute, water-soaked lesions on the emerging spindle with different shades of colour. 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 black and shrivelled. The inner whorls of leaves are more vulnerable to the disease (Fig. 10.4). Continuous attack of newly emerging spindle leaves results in the gradual exhibition of similar symptoms in all the leaves in the crown (Srinivasan and Gunasekaran 1992). Sometimes the decayed leaflets are glued together so that spindle does not open out. Though the disease does not kill the palm outright, its slow progress in the crown causes steady decline in nut yield. Palms of all ages are susceptible to the infection (Radha and Lal 1968; Srinivasan and Gunasekaran 1992).

Fig. 10.4 Leaf rot disease of coconut. (Photo: ER Asokan)

10.2.4.2 Aetiology

Radha and Lal (1968) confirmed 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., *Cylindrocladium scoparium* Morgan, *Fusarium solani*. (Mart.) Sacc., *Thielaviopsis paradoxa* (Date), *Rhizoctonia solani* J.G. Kühn and *Curvularia* spp. Boedijn. Of these, *C. gloeosporioides* (Penzig) Penzig and Sacc. and *E. rostratum* (Drechler) are considered as major pathogens of leaf rot disease based on their frequency of occurrence and pathogenicity (Srinivasan and Gunasekaran 1999).

10.2.4.3 Epidemiology

The tender leaf is the most susceptible to the disease (Lilly 1963). The susceptibility of the seedlings decreases with age. Seedlings up to 19 months may get severe infection. Leaf rot infection is more severe during the monsoon season when the conditions of high humidity are prevalent (Menon and Nair 1951). Severity of leaf infection by *Helminthosporium halodes* was found to be correlated with high temperature and low humidity present during monsoon period (Radha et al. 1961; Radha and Lal 1968). The population dynamics of leaf rot disease-causing pathogens in relation to environmental variables was studied by monthly isolations from the spindle leaves of diseased palms. The incidence of *C. gloeosporioides* was higher in frequency, and its population is high during monsoon with a peak in June–July. Its incidence was positively correlated with rainfall and RH and negatively correlated with maximum temperature and sunshine hours. Thus, *C. gloeosporioides* was implicated as the principal pathogen of leaf rot during monsoon. Incidence of *E. rostratum* was less frequent and not well-correlated with weather. *Fusarium* spp. and *R. solani* were isolated most commonly during the dry season of January–May (Srinivasan and Gunasekaran 1996). There was no significant difference in the amino nitrogen levels, ascorbic acid, total phenols or sugars 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 (Lilly 1963). Lilly and Ramadasan (1979) found that as a result of infection, the total phenols increased in the leaves.

10.2.4.4 Disease Management

Nair and Radha (1959) and Radha (1961) reported that regular manuring and spraying with copper fungicides checked the disease. By regular spraying, the intensity of leaf rot could be brought down from 40% to 7.8%. Spraying the leaves sequentially with Bordeaux mixture (1%), mancozeb (0.3%) and copper oxychloride (0.5%) at quarterly intervals after removing severely affected leaves was found to

reduce further incidence of the disease. Subsequently, in vitro assay of contact fungicides [Mancozeb (Indofil M-45), copper oxychloride (Fytolan), Captaf (Captan) and Thiram] and systemic fungicides [hexaconazole (Contaf), tridemorph (Calixin) and aureofungin-sol)] against *C. gloeosporioides* and *E. rostratum* indicated that hexaconazole (Contaf) exhibited a broad-spectrum activity inhibiting all the pathogens of leaf rot disease (Srinivasan and Gunasekaran 1999). A field trial conducted for 3 years on 20-year-old palms revealed that pouring of tridemorph (Calixin) (1%) into leaf axils and spraying of Mancozeb (Indofil) and Dithane M-45 (0.3%) along with phytosanitary practices reduced the disease intensity (Srinivasan and Gunasekaran 1999). The bacterial antagonist *Pseudomonas fluorescens* inhibited the growth of *C. gloeosporioides* and *E. rostratum* under in vivo conditions and reduced the leaf rot onset. Of the 96 phylloplane and 21 rhizosphere isolates from coconut, 2 isolates each from phylloplane and rhizosphere have been identified as effective native antagonists against pathogens, *C. gloeosporioides* and *E. rostratum* (Srinivasan and Gunasekaran 1999; Gunasekaran et al. 2001).

Radha (1961) had observed that Andaman Ordinary and Papua New Guinea cultivars were more resistant to leaf rot. The control of leaf rot gained significance because of vulnerability of root (wilt)-affected palms to leaf rot. An integrated management system involving need-based pruning of infected spindle leaf and a few leaves close to spindle and the use of hexaconazole (Contaf) 2 ml in 300 ml of water poured onto the spindle are the most important measures for controlling leaf rot (Srinivasan and Gunasekaran 2000).

10.2.5 Leaf Blight

This disease is widespread over all coconut-producing countries, but is of little importance in well-managed plantations. The disease causes serious damage in seedlings as well as in adult palms. Grey leaf blight incidence reduces coconut yield to the extent of 10–24% (Karthikeyan et al. 1997). Coconut palms severely affected with grey leaf blight flowered relatively later than the less affected ones (Abad and Blancaver 1975).

10.2.5.1 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 greyish margin on the leaflets of older leaves. They are oval in shape and are about 1–5 cm long. The centre of these spots later becomes greyish, and spots may coalesce, giving the leaves a burnt appearance. Complete drying and shrivelling of the leaf blades occur in the later stages. Such a condition is referred to as ‘blight’. On the upper leaf surface, globose, spherical, rectangular or ovoid black pycnidia of the fungus are formed. Some varieties are more susceptible than others.

10.2.5.2 Aetiology

The fungus *Pestalotiopsis palmarum* (Cooke) Steyaert is the causal agent of the disease. *Conidiomata acervulii* are globose or ellipsoidal, subepidermal in origin. Conidiophores are indistinct. Conidiogenous cells are discrete, simple, short and filiform. Conidia measure 17–25 × 4.5–7.5 μm; are fusiform to ellipsoid, mainly straight and four-septate; have 3 median cells; and are concolorous and olivaceous, with lower cell sometimes paler, together measuring 11.5–16.5 μm long; with hyaline apical and basal cells; with 3 appendages, 5–25 μm long, arising from the apex of the apical cell; and with filiform basal appendage, 2–6 μm long. Over the years, there has been some 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 five-celled conidia into *Pestalotiopsis*, while retaining *Pestalotia* for those species with six-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) 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 3 other *Pestalotiopsis* species on coconut.

10.2.5.3 Disease Management

The disease incidence indicates the poor nutritional status of the affected palms. Imbalanced nutrition such as potash deficiency or too much inorganic nitrogen causes the seedling to be more susceptible (Karthikeyan et al. 1997). Leaf damage by insects may also provide an entry point to the fungus. The best way to avoid the disease is by improving the growing condition of the affected palms. Diseased palms may be treated with fortnightly sprayings of Bordeaux mixture, copper fungicides or carbamates containing zinc or manganese. Regular application of potassium chloride was reported to reduce the disease incidence. Cutting and removal of severely affected lower leaves and spraying of fungicides like carbendazim (0.1%) or Bordeaux mixture (1%) to affected palms immediately after the appearance of symptoms are advocated.

10.2.6 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 already been weakened by other causes such as lack of drainage, moisture stress and malnutrition.

Leaf blight is an emerging serious problem in certain districts of Tamil Nadu, India (Johnson et al. 2014). The same fungus also infects seed nuts (Raju 1984). Though leaf blight is present in coconut-growing areas of other states of India, the disease is not a serious problem.

10.2.6.1 Symptoms

The pathogen causes damage to both leaf and nuts. Affected leaflets start drying from the tip downwards and exhibit a charred or burnt appearance (Fig. 10.5). 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 are 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 colonise the rachis, inducing internal necrosis that moves upward towards 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 appears near the perianth of immature nuts. The eriophyid mite-attacked nuts are infected by the pathogen causing immature nut fall and rotting. When nearly mature or fully mature nuts were infected, the infection spreads internally into mesocarp without any external symptoms. The affected nuts are desiccated, shrunk, deformed and drop prematurely causing 10–25% loss in nut yield (Venugopal and Chandramohanan 2006).

Fig. 10.5 ***Lasiodiplodia*** leaf blight disease of coconut. (Photo: V Hegde)

10.2.6.2 Aetiology

The disease is caused by the fungus *Lasiodiplodia theobromae* (Pat.) The fungus is geographically widespread but is most common in the tropics and sub-tropics. It is plurivorous and has been associated with approximately 500 hosts. The pathogen has been reported to cause numerous diseases, including dieback, root rot, fruit rots, leaf spot and witches' broom, among many others (Punithalingam 1980). The main feature of the fungus is the presence of pycnidial paraphyses and longitudinal striations on mature conidia. So far, 20 species have been described and are differentiated on the basis of conidial and paraphyses morphology. Further reports indicate that *L. theobromae* is a complex of different cryptic species (Alves et al. 2008). Large number of isolates from coconut leaf blight needs to be collected and characterised to determine the exact species status.

10.3 Virus Disease

10.3.1 Coconut Foliar Decay or Vanuatu Wilt

The coconut foliar decay is a virus disease of introduced coconut palms in Vanuatu. It is also known as foliar decay *Myndus taffini* or New Hebrides coconut disease. The name '*Myndus taffini*' comes from the plant hopper insect that transmits the disease. The disease is economically important because of its influence on regional coconut industry and internationally on quarantine considerations.

10.3.1.1 Symptoms

The first symptom on palms in the field is yellowing of a few leaflets on any of the fronds between positions 7 and 11 from the spear leaf. The yellowing spreads along the fronds which break near the base so that they hang down through the still green lower leaves. As the younger leaves age, reaching positions 7–11, they, too, turn yellow, break and hang down. In due course of time, diseased palms will have the top and midsection fronds broken and hanging through the still green fronds below. As the disease progresses, the trunk narrows towards the top, and the palm dies after 1–2 years, except for those that are tolerant to the disease and show remission of symptoms. Foliar decay is more serious on Malayan Red Dwarf coconut introduced to Vanuatu. Coconut cultivars, viz. Vanuatu Tall and Vanuatu Dwarf, are usually not affected by the disease, which, though are hosts for the foliar decay virus, act as symptomless carriers (Randles et al. 1992; Hanold et al. 2003).

10.3.1.2 Aetiology

The disease is caused by a very small circular single-stranded DNA virus, 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 palm. Coconut is the only known host for the virus, which occurs in leaves, roots, embryo, trunks and even on the husk of the nut. Seed transmission is not yet established.

10.3.1.3 Transmission

The disease is transmitted by *Myndus taffini* Bonfils (*Cixiidae*), a plant hopper which breeds on the roots of *Hibiscus tiliaceus,* 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 the vector and virus are apparently limited in distribution in Vanuatu archipelago.

10.3.1.4 Disease Management

The disease is best controlled by either planting selected Vanuatu Tall or the hybrid, Vanuatu Tall × 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 though not practically possible. The '*FAO/IBPGR Technical Guidelines for the Safe Movement of Coconut Germplasm'* have recommended that coconuts should be moved as embryos growing in a sterile tissue culture medium (Anitha Karun et al. 2002). 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, seedlings may be monitored for viroids and *Coconut foliar decay virus* in an intermediate (third country) quarantine centre.

10.4 Viroid Diseases

Two viroid diseases, viz. coconut cadang-cadang and tinangaja, are recorded on coconut, and their distribution is limited to the Philippines and Guam, respectively.

10.4.1 Coconut Cadang-Cadang Disease

In the early 1930s, a devastating epidemic of cadang-cadang, the lethal disease of coconut palm, 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 the 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 Calicoan, Guiuan and Eastern Samar.

10.4.1.1 Symptoms

In the early stage, newly developing nuts become more spherical 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 chlorotic look. 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 for 2–4 years, the medium stage for about 2 years and the late stage for about 5 years. Usually, palms become infected only after they have reached the age of flowering. In 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.

10.4.1.2 Aetiology

The detection of 2 small disease-associated RNAs in 1975 provided the initial clue to the aetiology 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 get transmitted independently of any other microorganism (Hanold and Randles 1991).

10.4.1.3 Epidemiology

The mode of natural inoculation in the field is not known. No insect vector has been found (Randles 1987). Positive transmission was obtained through assisted pollination of mother palms with pollen from diseased palms. A small percentage of the progenies, as well as seed nuts collected from cadang-cadang-infected palms, was positive for CCCVd and was also successfully transmitted to palms through contaminated harvesting scythes.

10.4.1.4 Disease Management

At present, there is no direct control measure that can be recommended to manage cadang-cadang, but several possible strategies can be considered (Randles 1987). 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 programme in the Philippines. To minimise 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.

10.5 Future Strategy

Coconut being a perennial crop with a life span of several decades is vulnerable to the vagaries of nature at its various growth stages. Out of the several pathogens infecting coconut, the 3 major pathogens threatening coconut production world over are *Phytoplasma*, *Phytophthora* and *Ganoderma*.

Managing bud rot and immature nut fall caused by *Phytophthora palmivora* and *P. katsurae* is a major challenge owing to the difficulty in taking up curative and prophylactic measures in tall palms. Hence, modern technologies like computer-aided, remote-controlled drones could help in such operations. Remote-sensing technologies can help in regular surveillance for disease incidence, intensity and spread which can aid in planning and executing disease management measures.

Molecular techniques should be increasingly used to help in disease detection and in resistance breeding. Though PCR-based techniques for diagnosis of *Phytoplasma*, viruses and viroids are available, development of field-level, reliable and rapid diagnostic kits is essential.

Region-specific integrated disease management strategies for specific maladies have to be developed for attaining maximum efficacy in disease control. Efficient long- and short-term disease-forecasting measures are to be developed to undertake timely prophylactic measures.

Super palms, both in healthy areas and disease hot spots possessing pre-potency, should be identified on a regular basis to serve as potential donors of disease resistance.

Periodic crop loss surveys and constant monitoring and surveillance are essential to compute the economic loss caused by diseases to guide in policy decisions to combat serious diseases and to check the re-emergence of minor diseases and emergence of new diseases in the prevailing era of climate change. Strict implementation of domestic and international quarantine measures is to be taken for preventing the disease spread. Detailed studies on epidemiology and vectors of transmission are to be given importance.

Integrated management measures are to be popularised and supported by appropriate policy decisions to reduce economic loss to the farmer due to these maladies.

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