

IV.III LEAF ROT AND BUD ROT OF COCONUT

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Coconut (*Cocos nucifera* L.) is one of the most useful crops to mankind, cultivated in humid tropics. India is the principal country in cultivation and production of coconut (currently in 1.91 million hectares, forming a share of 16.02% in global area and 27.57% of the total global production). The productivity of coconut is, however, affected by various constraints -diseases, pests etc. Diseases contribute a major share towards crop losses in coconut. The palm crop is affected by diseases in different countries, caused by various group of pathogens such as fungi, phytoplasma etc. The economic importance of such diseases may also vary from country to country. Certain diseases are distributed in several countries while others are localized to specific country or region. A vast number of species of fungi attack different parts of the palm leading to variable symptoms and level of seriousness. Some of these damages are direct as in the case of bud rot or basal stem rot, where the palm is lost resulting in abrupt losses. Diseases like root (wilt) and stem bleeding cause debility of the palm leading to a gradual yield loss in the beginning which goes unnoticed till the crown symptoms begin to manifest and button shedding becomes apparent. A slow debilitating disease like the root (wilt) has far-reaching effects. The affected palm becomes prey to leaf rot which is a disfiguring disease causing acute yield loss. As a result of the disease, the leaf lamina thickness gets reduced and so does the strength of the other tissues. Diseases like bud rot can be prevented and yield losses can be minimized and in many other diseases by integrated management practices. This chapter attempts to give a comprehensive picture about various aspects of leaf rot disease of coconut and bud rot (including its fruit rot/nut fall phase) of coconut.

1. LEAF ROT DISEASE OF COCONUT

1.1. Introduction

Leaf rot disease (LRD) is widely prevalent in the southern districts of Kerala state, where root wilt disease (RWD) is endemic. Leaf rot generally appear on palms affected by RWD and the palms having latent infection of RWD might be also infected by LRD. Destruction of photosynthetic area of leaves due to LRD is

quite clear and hence RWD affected palms draw attention instantly with the onset of LRD. LRD incidence can be seen on palms of all ages and especially in palms below the age of 25 year. Severe incidence of the disease in young palms, seedlings can be also seen. Currently, the disease is rampantly distributed in eight southern districts of the state (Thiruvananthapuram, Kollam, Alappuzha, Pathanamthitta, Kottaym, Ernakulam, Idukki and Thrissur). Leaf rot along with RWD is also observed sporadically in northern districts of Kerala and also in certain districts of Tamil Nadu bordering Kerala - as for example heavy incidence of the RWD-LRD complex in Cumbum Valley in Theni, Dindigul districts and in Tirunelveli district of Tamil Nadu.

1.2. History of the disease

The RWD was first noticed in 1882 after the great floods in three isolated pockets of the former princely state of Travancore. The disease spread in all directions and then occurred in a contiguous manner in eight southern districts of Kerala and in isolated pockets in the northern districts of the state. It also spread to districts of Tamil Nadu, bordering Kerala. Leaf rot disease also probably existed in the erstwhile princely states of Travancore and Cochin since 1880's (Varghese, 1934; Menon and Nair, 1951). Scientific investigations on LRD were initiated in early 20th century, almost in parallel with the research on RWD (Narayanaswamy Iyer, Bourdillon *cf.* Varghese, 1934; Butler, 1908). Varying intensities of LRD (14-65%) were reported by different workers (CCRS, 1970, 1971; Radha and Lal, 1968; Srinivasan, 1991). Earlier, boundaries of the disease zone in the north were between Thodupuzha and Oachenthuruthu and in the south between Punalur and Kollam (Menon and Nair, 1948). Menon and Nair (1948) and Menon and Pandalai (1958) reported incidence of the disease on palms of all ages and it flourished especially in palms below the age of 25 year. The disease incidence was variable irrespective of hills, the plains and littoral sandy tracts. Radha and Lal (1968) reported LRD on palms both without RWD and with RWD symptoms, but palms of the former group invariably developed the root (wilt) symptoms in due course (latent infection).

Srinivasan (1991) showed general confinement of LRD to RWD affected palms and on an average of 65% RWD affected palms super infected with LRD; incidence also related to severity of RWD. Susceptibility of young palms with RWD symptoms to ready attack of LRD, irrespective of soil types, was also spelt out. Heavy incidence of RWD in palms of different ages in Cumbum Valley (Theni and Dindigul districts) of Tamil Nadu has been reported and severe incidence of LRD in RWD affected palms seen (Srinivasan, 2001a; Srinivasan *et al.*, 2000; Srinivasan and Solomon, 2001; Srinivasan and Sasikala, 2001). Sporadic occurrence of LRD with RWD has

been also found in northern districts of Kerala (Solomon, et al., 2001). A strong relationship of LRD with RWD incidence has been evidenced and there exists a correlation between them. The crop plants affected by phytoplasma are systemically infected and become weakened (Srinivasan, 1982, 1999). The coconut palms weakened by RWD pathogen (Phytoplasma) thus become susceptible to LRD. The occurrence of LRD with RWD is a distinct Phytoplasma- fungal disease complex. Hence LRD forms a part of RWD complex. With spread of RWD, the occurrence of leaf rot has also increased. Leaf rot in combination with root (wilt) is a disease complex of more concern to coconut growers as leaf rot brings about rapid decline of the affected palms and also the affected leaves become unsuitable for use (Solomon et al., 1998,1999; Srinivasan, 1999; Srinivasan et al., 1998).

1.3. Economic importance of the disease (Crop loss)

The annual loss due to RWD in 1950s was considered to the level of Rs. 5.6 million and average yield loss as 20% (Menon and Nair, 1948). Subsequently, it was felt that LRD incidence bring about rapid deterioration in condition of palms and on an average of 70% yield loss perceived as compared to wide range of nut loss due to RWD alone (43 to 82%) (Radha et al., 1962). The survey of 1984-85 put the loss due to RWD as 968 million nuts annually and the overall disease incidence as 26.44% out of 9.15 crore palms in the disease endemic region of Kerala. The intensity of the disease ranged from 1.5% (in Thiruvananthapuram district) to 75.6% (in Kottayam district). The estimated monetary loss (total loss in account of losses in husk, copra yield, leaf number and quality of leaves) on the basis of 1984 price index was about Rs. 3000 million (CPCRI, 1985a).

The sample survey of 1996-97 revealed the disease incidence as 24.05% out of 9.15 crore palms in the disease endemic region. The disease incidence ranged from 2.1% in Thiruvananthapuram district to 48.0% in Alappuzha district (Department of Agriculture, Government of Kerala, 1997). The precise computation of loss in yield due to LRD is not easy as it is difficult to separate the damages caused by RWD and LRD. However, LRD in combination with RWD is a disease complex of more economic concern to coconut growers. Leaf rot does contribute to the loss in nut yield significantly. In addition to loss in yield the loss in quality and quantity of leaves is also to be reckoned. Leaf rot distinctly brings about rapid decline of the affected palms and also the affected leaves are unsuitable for thatching etc. In the disease endemic region, an average of 65% of RWD affected palms are super infected with LRD that points out the level of seriousness of the disease (Srinivasan, 1991; Srinivasan et al., 1998).

1.4. Symptoms of the disease

The earliest mention of symptoms of LRD was by Butler (1908), the Father of Indian Plant Pathology. Patel (1938) described later a shoot rot disease of coconut. Subsequently, several reports have described the symptoms of leaf rot (e.g., Varghese, 1934; Menon and Nair, 1951; Radha and Lal, 1968; Srinivasan and Gunasekaran, 1992):

The RWD affected palm systemically suffers and the deterioration is observed in this form in spindle leaf also (Plate 1, page 390). Thus the emerging spindle in RWD affected palm is weak and it remains white and soft for a relatively longer period as compared to the spindles of healthy palms (Dwivedi *et al.*, 1979). This tender spindle is an ideal infection court and the weakened spindle play a critical role in the LRD incidence. Leaf rot initially appears as minute, water-soaked lesions on the emerging spindle with different shapes and colours, and occasionally on other young tender leaves. These lesions enlarge, coalesce leading to extensive rotting especially under favourable environmental conditions (high rainfall, r.h. and low maximum temperature etc). Mould growth (spore masses and mycelium of fungi) is also seen on the surface of affected leaflets (Srinivasan *et al.*, 1995). The rotting may progress into the interior of the spindle also. The infected spindle gradually decays, emanate a fowl smell and many insects-ants, earwigs, maggots etc. are attracted towards the decaying tissues. It may also increase the activities of certain major insect pests of coconut. Tips of leaflets and midribs also often become blackish and shrivelled. The tips of rotten spindle leaflets some times stick together while the base of the leaflets are still open. The rotten tissues dry up, turn black and fall-off (Plates 2 - 4, page 390).

With maturity of leaflets, the progress of rotting is slowed down in certain palms due to hardening of tissues and the basal portions of leaflets remain without symptoms, thus giving a fan- like appearance of leaves in the crown. Even though leaf rot is non-systemic, successive infection of emerging spindles does occur and rotting occur varying extents (successive infection leads to vertical spread of the disease, aided by close proximity of emerging spindles) ultimately resulting in appearance of the LRD symptoms in most or even in all leaves of the crown. Horizontal spread of LRD occurs from palm to palm with increase in incidence of RWD. The pattern of LRD symptoms in whorls of infected palms is such that the symptoms expressed in entire crown in 24-43% of palms (in different soil types) implying the linear progression/vertical spread of the disease in individual palms. The younger leaves (more specifically the tender unopened spindle leaf) play a critical role in LRD incidence. The appearance of the disease symptoms on petiole, mid-

rib/mid-veins of leaflets, in natural conditions as well as upon artificial inoculations, has been also found (CPCRI, 1996; Srinivasan and Gunasekaran, 1992, 1998a, 2000a; Srinivasan et al., 1998). In certain palms, the digital ends of leaves break due to the disease lesions and become yellow –hang, dry and fall-off (Srinivasan, 2002a,d). In mid-whorl yellow leaves, which appear in certain RWD palms (Department of Agriculture and Fisheries, Travancore, 1938,1939,1940), lesions/spots does appear in leaflets that coalesce resulting in severe blighting of lamina. In such mid-whorl yellowed leaves also severe leaf rot may appear. Palms undergoing chronic infection due to the disease decline steadily and might even succumb. For indexing and quantifying leaf rot disease grading system has been evolved (Radha, et al., 1961; Mathai, 1980; Srinivasan and Gunasekaran, 1996a).

1.5. Causal organisms

McRae (1916) made the earliest record of isolation of a fungus (Penicillium -like) from LRD affected palms; considered fungus as the cause of the disease (Sundararaman 1925,1929;Varghese, 1934). Later, association of *Helminthosporium (Bipolaris)*, *Gloeosporium*, *Gliocladium*, *Pestalozzia (Pestalotiopsis)* and *Fusarium* with LRD was reported; rotting of tissues by various fungi (except *Fusarium*) observed; *Helminthosporium* considered as the most virulent (Menon and Nair, 1951). *H.halodes* (= *B. halodes*) received emphasis as the pathogen of LRD- developed maximum spots on most tender leaf (Lily, 1963, 1981). The fungus was reportedly isolated from coconut leaves in Kasargod in north Kerala (Radha and Lal, 1968) but in such period further concrete evidences were lacking.

Detailed study of the LRD etiology was conducted. Srinivasan and Gunasekaran (1993) made Isolations from infected spindles - the association of a number of fungal species was found and LRD has been established as a disease of fungal complex (Plate 5, page 390). The fungi were identified to an International Standard as *Colletotrichum gloeosporioides*, *Exserohilum rostratum*, *Gliocladium vermoeseni* (Srinivasan and Gunasekaran, 1994a), *Cylindrocladium scoparium* (Srinivasan and Gunasekaran, 1995a), *Fusarium solani*, *F.moniliforne* var. *intermedium* (Srinivasan and Gunasekaran, 1999a), *Thielaviopsis paradoxa*, *Rhizoctonia solani*, *Mortierella elongata*, *Curvularia* sp., *Acremonium* sp. *Thielavia microspora*, *T. terricola* and *Chaetomium brasiliense* (Srinivasan and Gunasekaran, 1994b).

From pathogenicity tests conducted in the laboratory and field it has been proved that the first ten fungi are pathogenic. Isolations from disease graded spindles also revealed predominant species of fungi. *C.gloeosporioides* was more frequently isolated irrespective of grades (Srinivasan and Gunasekaran, 1995b, 1996b). Presence of fungi singly or in combinations in individual palms was noticed. However,

association of more than one fungus in greater number of diseased palms confirmed fungal complex nature of LRD (Srinivasan, 2002a,d; Srinivasan et al., 2002). The profuse mycelia growth and spore masses in diseased spindle tissues also revealed several among these species and *P. palmarum* (the grey leaf spot pathogen) was observed only in older leaves (Srinivasan et al., 1995). The predominant fungi (*in vitro* interactions) are associative-bear etiological significance to fungal complex nature of LRD (Srinivasan and Gunasekaran, 1995c). In healthy plants (free from RWD), LRD fungi induced restricted lesions but in RWD infected plants severe rotting appeared (Srinivasan and Gunasekaran, 1996c). While all other pathogens induced wet rotting of tissues, the fusarial fungi induced a dry rot symptom. *C. gloeosporioides* and *E. rostratum* are the main pathogens of LRD (Plate 6, page 391). The disease symptoms are reproducible in field planted seedlings having RWD by artificial inoculation of the pathogens singly and in combination (Srinivasan and Gunasekaran, 1998a, 2000a). Disease lesions on petiole, mid-veins, and midribs have been developed besides developing the disease symptoms in successively emerging spindles similar to natural conditions (Srinivasan and Gunasekaran, 2000a; Srinivasan et al., 1998).

Certain nematodes (*Panagrolaimus* sp., *Aphelenchoides aligarhiensis*, *Rhabditis* sp. and *Panagrolaimus rigidus* etc.) especially from rotten spindle tissues were reported (Nadakal, 1965; CPCRI, 1997). These nematodes have no role in LRD (Sosamma, 2000, Koshy et al., 2001,2002). Nadakal (1965) and Weischer (1967) firmly ruled then itself that nematodes have no role, respectively in leaf rot and root (wilt) disease of coconut. The foul smelling - decaying tissues of LRD affected spindles attracting scavengers to such tissues are within expectation and that have no biological relationship with the disease.

Leaf rot is an air-borne foliar disease and no biological agent is involved in the pathogen(s) spread within the palm and from palm to palm. This should be noted especially in the situation where the spread of RWD pathogen (phytoplasma) is spread/transmitted by insect vectors from infected to healthy palms.

From the evidences accrued in recent years it is found beyond doubt that LRD is a disease of fungal complex and two fungi- *C.gloeosporioides* and *E. rostratum* are the main pathogens (Srinivasan, 1998, 2002a,d; Srinivasan et al., 2002); also, RWD affected palms are vulnerable to leaf rot pathogens. Irrespective of soils, the species composition of leaf rot fungi seemed to be similar in Kerala and Tamil Nadu (Srinivasan, 2001a,b, 2002a,d; Srinivasan and Sasikala, 2001; Solomon et al., 2001). In mid-whorl yellowed leaves also, *C.gloeosporioides*, *Fusarium* etc. are associated with blighted tissues (Srinivasan and Gunasekaran, 1999b). Symptoms like leaf blight/ leaf rot of a number of diseases of coconut palm recorded in other countries may be generally compared with LRD (Ohler, 1984; Nambiar, 1994; Srinivasan, 2001b).

1.6. Biology of the pathogens

Earlier study (Menon and Pandalai, 1958) on a pathogen of LRD (*Helminthosporium*) showed that its spores are air borne; the spores germinate within 12 hours and infect the tissue on landing on susceptible tender leaflets. The infection histology of *H. halodes* was studied (Menon and Nair, 1951) by spore germination test – the fungal spore developed a hyaline germ tube (through either one or both of apical cells). Further, the germ tube produced an appressorium and an infection peg developed made entry in to the parenchymatous cells through cuticle. After entry, the fungus ramified freely inside the leaf and the cells disintegrated leading to rotting of tissues. The conidial/ morphological characters of *Helminthosporium*, *Gliocladium* and *Gloeosporium* were briefly described (identified as *H.halodes*, *G.roseum* and *Colletotrichum paucisetum*, respectively).

Infection histology of *H.halodes* was corroborated (Lily, 1963, 1981). In the tender most leaf maximum spots appeared, but no spots developed on older leaves, despite conidial germination. Thus for infection, *Bipolaris halodes* (*H.halodes*) preferred the tender most leaf. No difference in virulence among isolates of the pathogen was seen (CPCRI, 1979) and also no toxic effect of culture filtrates of the fungus was evident (CPCRI, 1981).

In LRD infected palms, certain patho-physiological changes due to host-parasite interaction were observed. Arginine infiltration (0.05%) into leaflets enhanced the infection by *B.halodes* and content of phenol increased in lesions (CCRS, 1965, 1971; CPCRI, 1975,1982). While silica content was high in the diseased leaves (CPCRI, 1976) variable levels of certain biochemical components (ascorbic acid, total phenol, total sugar etc.) were also observed. A steady increase in the total phenol of spindle leaves inoculated with *B.halodes* was found (Lily and Ramadasan, 1979) Total nitrogen, non-protein and protein nitrogen were found higher in youngest leaf over mature leaves, (Lily, 1981).

Srinivasan et al. (1995) found in infected leaflets of spindles a common profuse mycelia growth and spore masses and recorded the mycoflora of spindles and older leaves by observing the scrapings of leaves and through isolations. *C.gloeosporioides*, *E.rostratum*, *G.vermoeseni*, *F.solani*, *F.moniliforme* var. *intermedium* and *T.paradoxa* were detected frequently. *Pestalotiopsis palmarum* was isolated from the older leaves only and other fungi occurred in very low frequency. The behaviour of the predominant fungi of LRD was associative than antagonistic (Srinivasan and Gunasekaran, 1995c) although the interaction of fungi (in respect of colony merger, over- growing capacity and inhibition zone) variable.

C.gloeosporioides gains access to yellowed leaves of RWD (Department of Agriculture and Fisheries, Travancore, 1939; CPCRI, 1985b) affected palms also and hastens the destruction of the lamina (Srinivasan and Gunasekaran, 1996d, 1999b). *H.halodes* (= *E.rostratum*) grew best in oatmeal agar and *Gloeosporium* (*Colletotrichum*) in Brown's agar. Optimum growth of the fungi took place around neutral pH while high acidity or alkalinity had a depressing effect. Thermal death point of *H.halodes* was 60°C whereas it was 55°C for *Gloeosporium* sp., *P.palmarum* and *G.roseum* (Menon and Nair, 1951). Largest number of fungal spores in the atmosphere was seen during the Southwest monsoon (June-August). Relatively high incidence of *C.gloeosporioides* occurs during monsoons indicating its distinct biological activity in such periods (Srinivasan, 2002a,d; Srinivasan and Gunasekaran, 1996d, 1998a, 2000a).

Cultural and biological diversity of the pathogens, molecular basis of pathogen(s) etc. were to be known. Understanding the molecular basis / techniques like PCR was suggested (Srinivasan, 1999, 2001b; Srinivasan and Gunasekaran, 1999b; Srinivasan et al., 1998) that would add to detection process of the pathogens. Purified cultures of the main pathogens (*C.gloeosporioides* and *E.rostratum*) were subjected to extraction of Genomic DNA (rDNA). The length of PCR amplified products from both these pathogens was estimated at 590 base pairs (bp) and the PCR amplified internal transcribed spacer (4 and 5 regions) of the pathogens was digested with 12 endonucleases. The restriction enzyme digests yielded a pattern of three groups in *C.gloeosporioides* and two groups in *E.rostratum* (Gunasekaran and Ward, 2002).

1.7. Epidemiology of the disease

No correlation between weather factors and incidence of RWD is known. Even though RWD act critically in predisposing to LRD, the incidence, severity, fungal species etc. of leaf rot are significantly influenced by weather factors. The most severe incidence of LRD is observed in the monsoon-rainy seasons (high atmospheric humidity and low temperature) even as the disease incidence can be generally observed irrespective of seasons. Spore population of fungi in the atmosphere is high during the months of monsoon. Conditions such as compactness of spindle foliage, free water, leaf wetness (rain/ dew) etc. even in dry months influence the disease. However, the severity / expression of leaf rot is maximal during rainy seasons (CCRS, 1962, 1965, 1970; Menon and Nair, 1951; Radha et al., 1961; Radha and Lal, 1968; Dwivedi et al., 1979; Mathai, 1980; Srinivasan, 2002a,d).

In the light of its fungal complex etiology and the distinct effect of weather on LRD it is of utmost importance to understand the fluctuations of the disease-associated

fungi. Hence, Srinivasan and Gunasekaran (1996d) studied the population dynamics of LRD pathogens in relation to weather factors by multiple sampling experiments: Maximum population of *C. gloeosporioides* occurred during the months of monsoons (June-November/December; peak detection in June/July). The pathogen was most strongly correlated with rainfall and r. h. and negatively correlated with hours of sunshine and maximum temperature. Further it was isolated from early lesions more frequently as compared to advanced lesions. Hence *C. gloeosporioides* is the principal pathogen of LRD during monsoons.

E. rostratum was less strongly correlated with weather and it occurred less frequently or erratically. In winter, the population level of *C. gloeosporioides* was low, whereas population of *E. rostratum* was high. Populations of *Fusarium* spp. and *R. solani* were more during the dry season (January -May). The incidence of other fungi, though not influenced by specific weather factor, they do play some role in the disease. The fungi dominant during dry months perpetuate the disease. Relatively low incidence of *C. gloeosporioides* in dry period is due to its quiescence- i.e. dormant phase in dry months. But it sports an aggressive re-emergence by June on onset of South -West monsoon with favourable weather conditions. As spores of *C. gloeosporioides* is small and with heavy build-up of its population, the pathogen spread fast in the field with cyclic infections. *E. rostratum* may be assigned next in the order of infection rate. Aggressive activity of *C. gloeosporioides* during monsoons and over-all trend of fluctuations in fungal population in young LRD affected palms have been also confirmed (Srinivasan and Gunashekarana, 1998a, 2000a).

Boron deficiency was once thought to be a factor for LRD and the disease affected palms considered to have responded favourably to the application boric acid (George and Samaraj, 1966); but boron application did not ameliorate LRD (Radha, 1965). While high dose of nitrogenous fertilizers was also reported to favour the LRD incidence, the potassic fertilizers offered resistance. Between different soil types incidence of RWD alone and RWD plus LRD ranged between 67% and 78% and LRD super imposition varied between 54% and 76% of RWD affected palms in the disease endemic region (Srinivasan, 1991).

Increase in incidence of RWD (spread by insect vectors such as lace bug and plant hopper) leads to increased occurrence of LRD (primarily air-borne). As RWD spreads irrespective of seasons and leaf rot inoculums (one or the pathogen) available in the field palms throughout the year, vertical (within the already infected palm, onto emerging spindles) and horizontal (from infected palm to other palms) spread of LRD happens. Cyclic incidence of RWD followed by infection of LRD therefore leads to development of the disease complex (RWD plus LRD) in more palms in the field (Srinivasan, 2001b, 2002a,d).

1.8. Varietal resistance

The need for evolving a resistant variety against the "Coconut palm disease of Travancore" was stressed as early as 1900s (Butler, 1908). Out of 26 varieties from various parts of the world, none was found to be resistant to shoot rot (Patel, 1938; Patel and Nair, 1936). The varieties such as Andaman Ordinary and New Guinea were considered as more resistant (as compared to other varieties) to LRD (Radha, 1961; CCRS, 1962). In indigenous Chowghat dwarf green (CGD) variety, the natural incidence of LRD was low as compared to tall varieties (CCRS, 1965, 1971). In West Coast Tall X Laccadive dwarf and West Coast Tall (WCT) X Nyior Gading, lowest disease was found. Further, LRD intensity was also low in such varieties where RWD incidence was low; significantly lower levels of incidence of RWD and LRD were recorded in the variety Andaman Ordinary followed by SSG and Cochin China; San Ramon showed lower disease intensity followed by Guam, St. Vincent and Kenya (Mathai, 1980; Mathai *et al.*, 1985, 1988). CGD/hybrids of CGD X WCT are reportedly tolerant to RWD (Nair *et al.*, 1996). As such, varieties/cultivars resistant/tolerant to both RWD and LRD are more important.

1.9. Management of the disease

Control of LRD has received special importance in view of its destructive potential to lamina. Control efforts by spraying fungicides have been in practice (Varghese, 1934). *In vitro* assay of chemicals, fungicides etc. were reported (Menon and Nair, 1951; Prasannakumari *et al.*, 1960; Srinivasan and Gunasekaran, 1998b). Beneficial effect of spraying Bordeaux mixture (thrice a year) in the disease control was observed (Menon and Nair, 1952).

However, LRD control was variable even as some reductions observed in the intensity of the disease (Joseph and Rawther, 1991). The disease was reportedly checked by regular manuring and spraying operations with copper fungicides (Nair and Radha, 1959; Radha, 1961). Aerial spraying of oil based copper fungicides was thought to be useful in the disease control, but no consistent results were seen (Samraj *et al.*, 1966). LRD recurrences were common even though spraying with Bordeaux mixture 1%, Dithane M-45 0.3% and Fytolan 0.5% sequentially -thrice a year- on diseased palms reportedly reduce the disease level (CPCRI, 1985b); prevention of the disease was also not successful by basal application of certain fungicides (CPCRI, 1983). No significant effect of certain other treatments (NPK + Ca + Mg + Zn + Dasanit + Agrimycin -100 + Benlate) in reducing LRD was also observed (Mathai *et al.*, 1984).

Srinivasan and Gunasekaran (1996a) determined methods of application of

fungicides on to palms; simple measure of pouring the chemical into the spindle axil standardized. Beneficial effects of the fungicides - pouring of Calixin (systemic fungicide) and spraying of Indofil-M-45 (contact fungicide) - in the LRD control was found and the measures satisfactorily suppressed the disease recurrences in newly emerged leaves. Further, importance of phytosanitary measures in LRD control was highlighted. Contaf (hexaconazole), a systemic fungicide, has been assessed to be strongly inhibitory to the main incitants of LRD (Srinivasan and Gunasekharan, 1998b) - its possible utility in the disease control marked. Phytosanitation plus pouring of contaf on to the spindle axil significantly reduced leaf rot and elaborated into prophylactic control as well (Srinivasan, 2002a,b,c,d; Srinivasan and Gunasekaran, 1999b, 2000b,2002). Similar work has been repeated (Koshy et al., 2001,2002).

Antagonistic potential of *Bacillus subtilis* (Lily et al., 1952,1955), *Pseudomonas fluorescens* (Srinivasan, 2001c, 2002a,d; Gunasekaran et al., 2001) and such other bacteria from coconut phylloplane / rhizosphere (CPCRI, 1998; Gupta Alka et al., 2000; Srinivasan, 2002a,b,d) against LRD pathogens have been also discerned for possible utility. Both cultures and culture filtrates of certain of these bacterial species were found to inhibit the pathogens of leaf rot. Four bacterial antagonists (two *Pseudomonas* spp. and two *Bacillus* spp.), isolated from native coconut and short listed in the fore-mentioned studies, were further considered (Alka Gupta, 2002) to explain the basis of biological activity underlying certain known mechanisms of biocontrol processes such as production of secondary metabolites by the biotic agents (antibiotics, siderophores, hydrogen cyanide, ammonia etc.) and their extra-cellular elaboration of lytic enzymes (Chinase, beta-1, 3 glucanases) affecting the pathogens.

Control of LRD is a relief to the affected coconut as RWD infected palms is vulnerable to fungal infection (Solomon et al., 1998,1999; Srinivasan et al., 2001) and hence management of LRD mainly with fungicides forms an integral part in the management of RWD complex (Koshy, 1999, 2000). In endemic region, eradication of RWD palms (especially in advanced stage) is necessary. As young leaves (specially the emerging spindles) are vulnerable to LRD, protection of particularly such a part is needed. Therefore, pruning of infected spindle and a few near by leaves (phytosanitation) in the early stage / disease onset itself is useful. Keeping the palms in vigour by agronomic, protection measures etc. (prevention) would also help to reduce the disease incidence. RWD cum LRD resistant / tolerant variety is preferable. For effective management of the disease adoption of a systems approach (integrated measures) is very much necessary (Srinivasan, 1998, 1999, 2001b,c, 2002a,b,c,d; Srinivasan and Gunasekaran, 1999b, 2000b, 2002; Srinivasan et al., 1998, 2001;

Srinivasan and Sasikala, 1999).

Based on current knowledge on leaf rot, various parameters are integrated and a system evolved (phytosanitation, fungicides/chemicals, cultural/agronomic practices, disease resistance/tolerance, biological control etc.) for the disease endemic region for effective management of the disease:

- ❖ Removal of disease advanced/senile/uneconomic palms followed by replanting, if necessary, with healthy seedlings from elite palms/varieties or hybrids resistant-tolerant to the disease complex.
- ❖ Adopting all recommended package (agronomic, cultural etc. practices) as applicable to RWD endemic region (as described elsewhere) – such practices would aid the palm to grow in sound vigour, countering the state of plant sickness.
- ❖ Regularly monitoring the individual palms. If spindle and other young leaves are found infected, the affected portions should be promptly pruned and disposed - off by burning. As a follow-up, effective fungicide like Contaf should be poured onto spindle region (0.2 – 0.4% conc.; up to one litre/palm; 2 – 3 times/year).
- ❖ Treating the palms at initial stage itself would be more useful; in moderate cases, treatment need be prolonged.
- ❖ Keeping all treated palms also under regular observations to counter any recurrence of the disease.
- ❖ General cleaning of the crown in pre-monsoon period would help reducing LRD inoculum and palms should be further monitored with a thrust during monsoons.

Application of pesticides in the spindle region should be exercised cautiously, because the spindle is very soft, tender and sensitive. Indiscriminate use of chemicals may result in phytotoxicity and in environmental pollution. This is especially relevant in situations of combining coconut pest control with LRD (as for example, it has been noticed that phorate application in spindles region of coconut exert phytotoxicity to these soft tender tissues). Biological control of LRD is being evaluated with a direction of eco-friendly measure of disease control and its potentiality could be effectively combined in the integrated management measures. The integrated management practices would go a long way in control of the RWD-LRD complex, enabling coconut cultivation in a sustainable manner.

1.10. Other information(Summary)

Incidence of LRD is correlated to the incidence of RWD. Root (wilt) is caused by phytoplasma (Solomon et al., 1998, 1999). The phytoplasma affected crop plants usually suffer systemically (Srinivasan, 1982, 1999). Hence, RWD affected palms also suffer systemically. The RWD is not lethal (as compared to phytoplasma diseases of coconut in other countries that are lethal) but only a debilitating disease (i.e. a slow decline) and such affected palm becomes further susceptible to fungal infection – a unique case. The vulnerability of RWD affected palms to LRD is distinct and thus LRD is a part of RWD complex. It has been confirmed beyond doubt that LRD is a pathological syndrome and due to a complex of fungi. More than one fungus is associated in greater number of diseased palms. *C. gloeosporioides* and *E. rostratum* are the main pathogens of the disease. Emerging spindle is the most vulnerable part. The influence of weather factors on the epidemiology of LRD is distinct. Leaf rot is severe during monsoon months even as the disease incidence can be generally seen irrespective of seasons. Activity of *C. gloeosporioides* is significantly higher during monsoon months (principal pathogen), followed by *E. rostratum*. The pathogens viz., *Fusarium* spp. and *R. solani* in addition are common during dry seasons. The phytoplasma-induced root (wilt) is not amenable to conventional crop protection measures. However, leaf rot control has been demonstrated possible (Srinivasan, 2002a,d). Strategies have been evolved for integrated management of RWD complex in mildly affected areas and in disease endemic region. Leaf rot control (various measures as described earlier in this chapter) forms an integral and essential part in the management of the RWD complex in the disease endemic region. Current understanding on various aspects of leaf rot including control measures could be strategically utilized for effective management of the disease complex with environmental consideration.

2. Bud rot of coconut

2.1. Introduction

Bud rot is a fatal disease of the coconut palm, characterized by the rotting of the terminal bud and surrounding tissues. Even though it affects palms of all ages, young palms in low lying and moist situations are more susceptible to the disease. It is generally a sporadic disease, but some times epidemics appear. The disease commonly occurs in West and East coasts of India (Menon and Pandalai, 1958; Ohler, 1984; Srinivasan, 2001b). Occasional heavy incidence of bud rot occur in plantations of Kerala, Karnataka and Tamil Nadu

2.2. History of the disease

Bud rot is a common disease of coconut in several countries. The earliest report of bud rot incidence was from Grand Cayman, an island of British West Indies (south of Cuba) in 1834. Since that time the disease has been reported from every coconut producing region of the world. The distribution of the disease occurs in both hemispheres of the world. Butler (1906) reported bud rot disease from India. *Phytophthora* attack on coconut is reported from Sri Lanka, India, Papua New Guinea, Philippines, Vanuatu, Fiji, French Polynesia, Indonesia, The Dominican Republic, Colombia, Cote –de – Ivoire (Quilic and Renard, 1984).

In India, as mentioned earlier bud rot is quite common especially on the West and East Coast tracts. Radha and Joseph (1974) reported a disease incidence of 1.2 to 10.9 per cent in Kerala and observed the infection in 35 to 40 per cent of palms in certain gardens having large number of palms. Ghosh and Yadav (1993) reported the incidence of bud rot varying from 1.45 to 3.65 % in Tamil Nadu, 0.90 to 10.00% in Andhra Pradesh (West Godavari, Krishna and Chittoor districts) and 6.5% in Kerala. Bud rot incidence was also noticed in other states like Karnataka, Maharashtra and Goa in large scale, particularly in the newly planted gardens. In non-traditional coconut growing areas of Madhya Pradesh like Bastar, Raipur and Durg also bud rot is a problem of serious concern (Dantre et al.1997).

2.3. Economic importance of the disease (Crop loss)

The production of coconut and copra is an extremely important activity in Indonesia and across South East Asian countries with production of copra being 2,34,200 tonnes pa (26% of the world production) from 3,41,800 acres (32% of world coconut area). Bud rot and premature nut fall caused by *Phytophthora* species, (mainly *P.palmivora*) are the major disease problems affecting Indonesia and accounts for extensive losses both stands and nut production. In some areas, stand losses of 43% can occur due to bud rot. Bud rot due to *Phytophthora* is irregularly distributed on hybrids in Indonesia. However the number of diseased palms was high in the wettest areas. In Indonesia, where *Phytophthora* diseases pose serious problems especially on the PB121 Hybrid and on the Nias Yellow Dwarf (Bennet et al., 1985).

Bud rot also causes considerable damage in Costa Rica, Mexico, British Honduras, Trinidad, British Guyana and Haiti. According to Renard and Darwis (1993) there has been a resurgence of symptoms in almost all coconut growing zones. In certain regions in the Ivory Coast up to 50% of the coconut initially planted are killed by bud rot and up to 25% nut fall can be recorded each year. The extent of damage depends upon climatic conditions and on the type of planting material

(Quilic et al., 1984). Bud rot disease is very serious in areas with high and well distributed rain fall.

Coconut bud rot is endemic to Jamaica where the disease occurs island wide and attacks coconut palms of all ages, thereby causing considerable loss of income to coconut farmers. Bud rot disease was first reported in Jamaica in 1870 (Anonymous, 1905). In Jamaica bud rot incidence appears on less than 1% palms per year. However the disease incidence varies between plots and in few isolated areas it exceeds 10%. Damage to coconuts caused by the bud rot disease began to be reported in North Sumatra from 1984 onwards, although apparently similar symptoms has been described in Indonesia (Brahmana and Kelana, 1988). In Cot – de –Ivoire, bud rot due to *Phytophthora* was reported for the first time in 1977 on West African Tall. The First attack in Dwarf x Tall hybrids occurred in 1981.

2.4. Symptoms of the disease

The early externally visible, diagnostic symptoms of bud rot is the withering of the youngest spear leaf which subsequently turns pale brown and bends (Plates 7 - 9, page 391). In due course, the younger leaves and leaf sheaths closer to the spindle develop water soaked lesions and show similar symptoms. By the time the disease is visible, it is already at an advanced stage. The internal tissues show discoloration assuming a pale pink with a brown border (Quilic and Renard, 1984). The base of spindle tissue rots, emits a foul smell and such leaves may gently be pulled out. The spindle hang down; other leaves in the inner whorl also fall away one by one, leaving only the matured leaves of the crown in the lower whorl at the trunk The older leaves may retain with green colour for long period; the nuts may also remain with still maturity. The palm ultimately succumbs to the disease (Briton-Jones, 1940; Menon and Pandalai, 1958; Ohler, 1984; Srinivasan, 2001b).

2.5. Causal organisms

The causal agent of bud rot was first suspected to be *Pestalotia palmarum* by Busck (1902). Later, *Bacillus coli* (*Escherichia mingula*) was considered to be responsible for the disease (Petch, 1906; Stockdale, 1907; Fredholm, 1909; Johnston, 1910, 1911). Subsequent investigations revealed that bud rot is of fungal origin (Shaw and Sundararaman, 1914; Mc Rae, 1923; Tucker, 1926).

E.J. Butler described bud rot in palmyra and coconut palms and isolated a fungus, named as *Pythium palmivorum* in 1906. Later he placed it in the genus *Phytophthora* in 1928, and reproduced the disease symptoms by inoculation. Mc. Rae (1923) published the results of successful inoculations on both *Borassus flabellifer* L. and *Cocos nucifera* L. Certain other species of *Phytophthora* such as *P.*

faberi Maubl. (Philippines), *P.heveae* (Ivory Coast, French Polynesia), *P.parasitica* (Costa Rica), *P.nicotianae* (Indonesia) have been also reported by different workers in various countries as the cause of the disease (Plate 10, page 392).

In 1919 Reinking produced bud rot in Philippines using pure cultures of *Phytophthora faberi* isolated from rotten Cacao pods and found that *Phytophthora* strains isolated from coconut and Cacao were identical both morphologically and physiologically (Briton-Joñes, 1940; Ohler, 1984).

Following isolations of *Phytophthora palmivora* from bud rot affected palms from India and confirmation from Butler, Ashby (1920) reported *Phytophthora* to be the sole cause of the disease in Jamaica. Resurgence of the bud rot disease coincided with the introduction of two lethal yellowing resistant coconut cultivars 'Malayan Dwarf' and Malayan Dwarf x Panama Tall hybrid. These cultivars are highly susceptible to *Phytophthora* attack.

Phytophthora katsurae (initially identified as *Phytophthora heveae*) causes significant damage in Cote –de –Ivoire, either through coconut bud rot, which inevitably leads to tree death, or through immature nut fall which can lead to harvest losses of around 30- 40 %. Quillec and Renard (1984) Franquillee et al (1989) and Steer and Coates– Beckford (1990) reported *P.heveae* in the Ivory Coast, Vanuatu and French Polynesia, and *P. parasitica* in Costa Rica as causal agents of the disease. Schieber (1970) reported *P.palmivora* from the Dominican Republic.

Joseph and Radha (1975) found that *P. palmivora* causes dry rot in coconut prior to wet rot due to the secondary invaders such as *Fusarium* sp. and bacteria. Steer and Coates–Beckford (1990) observed in green house trials in Jamaica that *Phytophthora* caused bud rot only when wound inoculated and also that *P.palmivora* isolates varied in virulence. The bacterium *Enterobacter* sp., caused tissue necrosis but when inoculated, the seedlings did not develop bud rot. Studies in Jamaica (Anonymous, 1989) indicated that *T.paradoxa* always produced lesions and nut fall in the laboratory tests but its association with nut fall affected fruits was very low.

2.6. Biology of the pathogens

The genus *Phytophthora* was erected by Anton de Bary in 1876. In this genus both heterothallic and homothallic species are reported. Both sexual and asexual reproductions are present in *Phytophthora*. Studies have clearly shown the existence of two dominant *Phytophthora* species viz., *P. palmivora* and *P. katsurae* in relation to bud rot of coconut. The occurrence of *P. Katsurae* causing bud rot has been reported from Hawaii and also Ivory Coast. Veena et al., (1997) reported *P. katsurae*

from Kuttiadi area in Kerala. *P. katsurae* was first isolated from Chestnut in Japan. It was named as *P. castaneae* and later renamed as *P. katsurae*. The causal organism in Ivory Coast was called *P. heveae*; it is now believed misidentified. The Ivory Coast *Phytophthora* has oogonial protuberances like the coconut pathogen in Hawaii. *P. heveae* does not have oogonial protuberances like the coconut pathogen in Hawaii.

Studies have shown the existence of two dominant species of *Phytophthora*, *P. palmivora* in Indonesia and Philippines, and *P. katsurae* in the Ivory Coast. Both species are found in Jamaica. *P. arecae* and *P. nicotianae* are also found in Indonesia (Renard and Darwis, 1993). Uchida et al. (1992) reported a new *Phytophthora* fruit and heart rot of coconut in Hawaii, the pathogen of which resembled *P. katsurae*. *P. palmivora* develop sporangia (ellipsoidal or ovoid) abundantly. The sporangia releases zoospores (biflagellate and motile) on germination that in turn germinate and infect the host. The pathogen produce chlamydospores and oospores, and also exists in morphological/pathological forms. Presence of both "Plus" and "Minus" strains of the fungus are also seen and coconut isolates are considered as A₂ mating type (Kumar and Hegde, 1987).

2.7. Epidemiology of the disease

Bud rot disease is favoured by high humidity such as conditions found in low lying badly drained lands, in plantations with a very dense stand and under conditions of extensive rainfall. Rillo and Paloma (1988) observed higher incidence of *Phytophthora* in Philippines was always preceded by high rainfall during the previous months. Palms of all ages were susceptible to the disease, but it was rampant in palms below 20 years of age. The period elapsing between the infection of the pathogen and withering of heart leaf depends on a number of factors, the more important of which are, relative humidity and the point of infection. The period may vary from three to nine months or even higher. With return of dry weather, the infection becomes less severe and the pathogen remains dormant. The fungus was found to survive in the frond base or basal part of the crown (Menon and Pandalai, 1958; Radha and Joseph, 1974) or in roots (Harris et al., 1984).

Seasonal factors influence the incidence and spread of the disease. The incidence is found to be severe during monsoon when the relative humidity is high. Studies conducted in Kerala (Radha and Joseph, 1976) revealed that the favourable period of infection is when relative humidity is above 94% and temperature is below 24°C. An infection cycle of *Phytophthora palmivora* in coconut is completed within 6 days under favourable conditions of humidity (98-100%) and temperature (22-24°C). Hence, the rainfall pattern in a given year may play critically on the development of bud rot and the disease can spread over long distances also. During

dry conditions the pathogen can remain dormant in leaf bases and can also survive in roots of healthy palm. It would emerge with arrival of favourable climate. The fungus has also a wide host range. Epidemiological models applied to plots affected by *Phytophthora* in the Ivory Coast suggest the existence of two propagation phases (an aggressive phase where contamination occurs from tree to tree) and a regular phase (during which new cases appear some distance away from the initial foci). The disease is primarily spread by wind and wind blown rain (Renard and Darwis, 1993).

2.8. Varietal resistance

Resistant sources is characterized by assessing the field performance of planted varieties. The performance of coconut varieties depends on the regions where they are planted. Quillec *et al.*, (1984) observed that hybrids were less sensitive than West African Tall. However, Brahmana and Kelna (1988) found that dwarf palms are more sensitive and others are more tolerant. In Dwarf varieties, Nias Yellow Dwarf (NYD) seems to be the most susceptible one (Bennet *et al.*, 1986). Bud rot is also observed predominantly in areas where PB 121 (MYD x WAT) coconut hybrids are planted. Exotic Talls such as Rennel Tall and local Indonesian Talls are more tolerant. Dwarf X WAT hybrids are highly susceptible to bud rot. Hybrids of Malaysian Yellow Dwarf x Rennel Tall or Local Tall were found to be less affected than PB 121. The NIWA hybrids obtained by crossing NYD x WAT are susceptible to bud rot. Rillo and Paloma (1989) found that *Phytophthora* incidence was higher in Cameroon Red Dwarf and MYD as compared to Catigan. Coffe *et al.*, (1990) reported that in Indonesian 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 to *P. palmivora* than introduced ecotypes, 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 Cote-de-Ivoire and is also susceptible to the same damage in Jamaica, where as Red Dwarf x Tall coconut hybrids are more tolerant of 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 hardly susceptible or even highly tolerant in Jamaica. In Philippines, MAWA hybrid (Malayan Yellow Dwarf x West African Tall) was found to be susceptible to bud rot. For avoiding high economic loss, it is recommended that the choice of planting material should be done by taking into account the environmental conditions (Mangindaan. *et al.*, 1992).

2.9. Management of the disease

Control measures are more effective only when they are adopted in the initial stage of the disease. If the disease is detected when the central shoot just withers, chemical control by the application of 10 per cent 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 so as to prevent washing off of the paste during rain. Badly affected palms should be cut and removed from the field as they are not recoverable. Strict phytosanitation should also be enforced. As a prophylactic measure, adjacent healthy palms should be sprayed with 1% Bordeaux mixture. Other fungicides advocated earlier against bud rot disease include Phenyl mercuric urea applied in the form of powder or pellets in the leaf axils, (Peiris, 1962) or stem injection or root infusion of Aliette or Ridomil or Phosphorus acid (Brahmana *et al.*, 1992). Regular spraying with copper fungicides at 40-day intervals especially before and after monsoon is an effective preventive measure. In copper sensitive palms, keeping perforated sachets containing Dithane M-45 in the leaf axils during rainy season is useful (Nambiar and Rawther, 1993).

Antagonistic fungi like *Trichoderma* sp., *Myrothecium roridum* and *M. verrucaria* (Tuset. *et al.*, 1992) are reported to suppress the growth of *P. palmivora* and *P. katsurae*. It has been reported that antibodies produced by these fungi may have a role in the suppression of the pathogen in the soil.

Proper spacing between the palms is important for the management of the disease. Too close planting encourages spread of the disease. A good spacing between palms favours air movement and dissipation of the excess humidity that can build up in the gardens. When grown without proper drainage the coconut palms are highly susceptible to bud rot disease. The low lands with generally high humidity are very favourable for the development of the disease, especially if there is no drainage. Organic matter application favours the growth of a variety of microbes including antagonists such as *Trichoderma* and *Gliocladium* sp. which multiply on the organic matter and help in reducing the population of *Phytophthora*. Biological control is geared towards identifying microorganisms effective against *Phytophthora*. Constant use of site specific fungicide is not desirable because strains of *Phytophthora* sp. resistant to fungicide may appear with their continued use. The control of bud rot thus involves integration of resistant varieties, improved cultural practices, systemic fungicides, biological control methods etc.

2.10. Other information

Another important aspect of the causal agent of bud rot is its ability to infect coconut nuts causing fruit rot or nut fall disease (Srinivasan, 2001b). Nut fall does not kill the palm but substantially reduces the production. The disease is characterized by rotting of immature nuts especially during rainy season, resulting in nut fall (Plates 11-12, page 392). Nuts, two to five months, are attacked. Symptoms begin as water soaked lesions usually appearing near the perianth. These lesions turn brown and become irregular in shape and may spread into the husk and may reach the endosperm (Concibido Manohar, 1990; Quillec and Renard, 1984). Premature nut fall is an important disease because nuts of various ages may be affected. A decrease in the production due to nut fall is observed in Indonesia, where ever the hybrid PB121 and local Dwarfs are planted (production up to 50% reduced). According to Bennet et al., (1985) the loss of production during rainy season can reach up to 75%.

In Indonesia premature nut fall is particularly severe on Yellow Dwarf (Renard and Darwis, 1993) and mainly concerned with immature bunches, particularly during the rainy season. Pathogen propagation is either horizontal, by contact between bunches, during the rainy season. The role of rain water and insects in the disease progress was demonstrated. The infection starts around the floral parts, some times in the equatorial parts, and extends towards the apex of the nut and inside towards the shell. The disease is characterized by the presence of irregular lesions spreading from the surface of the nut to the endosperm. The rate of lesion development is inversely proportional to age of the nut (Bennet et al., 1986). In young nuts, lesion penetrates to the soft shell and the infected testa becomes grey and sticky. The adjacent infested endosperm is more translucent and softer than the usual. In older nuts with hard shell the fungus penetrates through the germ-pore. Bennet et al., (1986) suggest that a dipteran, *Thalostylinus* sp. probably feeding in sugary exudates of young nuts leak on the husk surface of healthy and diseased nuts could act as a vector of the fungus in Indonesia.

Two species of *Phytophthora*, namely *P. palmivora* and *P. katsurae* are important in relation to the nut fall disease (Ooka and Uchida, 1984; Steer and Coates-Beckford, 1990). Climatic factors are found to be associated with the incidence and spread of the disease. The disease incidence is higher when rainfall and the humidity are high. The disease is most active during wet weather. Spores are dispersed by rain splash and air currents. Resistant chlamydospores can survive in soil and in coconut debris including nut tissue. Dissemination of the disease is by simple contact between fruits or via dissemination agents.

The disease can be effectively prevented by prophylactic spray with 1% Bordeaux mixture during pre-monsoon period. Test trials conducted reveal that fungicides like Fosetyl-AI are very effective against nut fall and bud rot. To avoid the spread of the disease, the diseased nuts should be separated and burnt. Proper spacing of palms in garden is important to avoid high humidity which is favourable to the pathogen. Genetic control is through the identification of resistant sources. The performance of coconut varieties depends on the regions where they are planted (Mangindaan *et al.*, 1992). In the Philippines, the disease was first observed on Cameroon Red Dwarf plantation and later on the Malayan Red Dwarf and Malayan Yellow Dwarf plots. Rillo and Paloma (1988) claimed that red and yellow coloured nut populations are more susceptible to nut fall as compared to green coloured cultivars. A decrease in production due to nut fall is observed in Indonesia, where ever the hybrids of PB 121 (MAWA) and local dwarf coconuts are planted.

Observations have shown that the resistance of coconuts to nut fall is variable. In North Sumatra, premature nut fall has been found on hybrids and on West African Tall coconuts, but other varieties such as Bali Tall, Tahiti and Rennel Tall are not affected by nut fall. In Cote-d' Ivoire the West African Tall was highly susceptible to bud rot, whereas its nuts remained unaffected; on the other hand, the MYD x WAT hybrids (PB 121) were not much affected by bud rot but nut fall is considerable. On the whole, the Malayan Yellow Dwarf remained less affected by either bud rot or nut fall. The cultivars most tolerant for nut rot include the Cameroon Red Dwarf (CRD), the Malayan Red Dwarf (MRD) Malayan Yellow Dwarf (MYD), and Vanuatu Tall (VTT). The hybrids with WAT (West African Tall) suffer with more losses. The dwarf varieties are most susceptible to immature nut fall in Indonesia and severe nut fall is seen particularly in Nias Yellow Dwarf (NYD) in certain regions of the country.

In summary, the heavy incidence of nut fall can be checked by planting of coconut hybrids tolerant to nut fall, keeping good spacing between palms, providing proper drainage and by judicious use of fungicides

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