PLANT PATHOLOGY RESEARCH AT CPCRI

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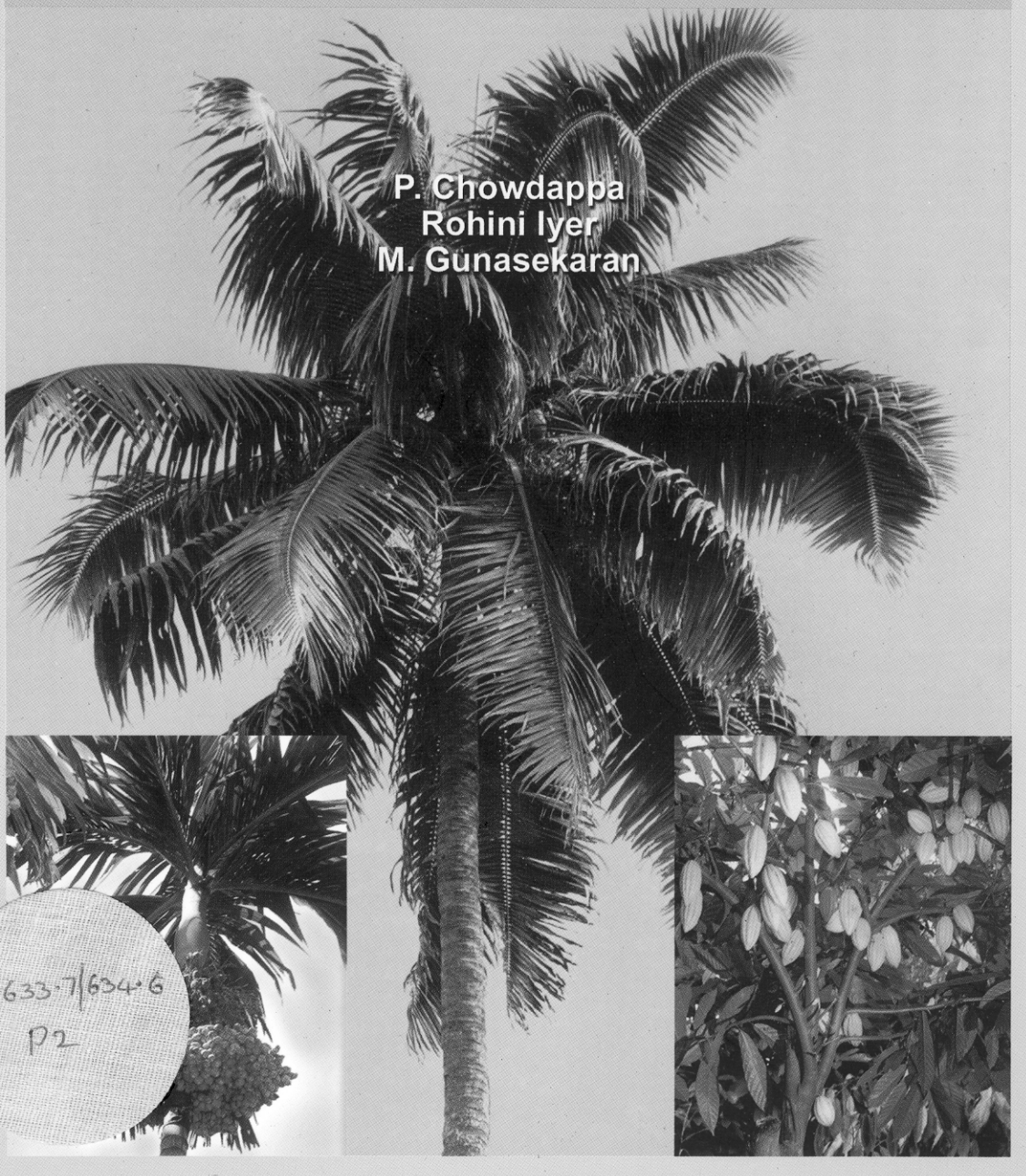

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Complied and edited by

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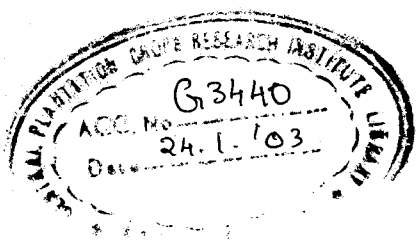

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1. INTRODUCTION

Central Plantation Crops Research Institute (CPCRI) was established in 1970 by merging the erstwhile Central Coconut Research Station, Kasaragod, Central Coconut Research Station, Kayangulam, and the Central Arecanut Research Station, Vittal along with its Sub-stations at Palode and Kannara (Kerala), Hirehalli (Karnataka), Mohitnagar (West Bengal) and Kahikuchi (Assam).

The mandate of the institute includes the following:

- Crop improvement and developing appropriate Production, Protection and Processing technologies for coconut, arecanut and cocoa
- Acting as National repository for the genetic resources of the mandate crops
- To produce parental lines and elite planting materials of coconut, arecanut and cocoa
- Co-ordinating the research under the All India Co-ordinated Research projects on palms.
- Transferring appropriate technologies developed on coconut, arecanut and cocoa to the farmers by establishing linkages with development departments

CPCRI has made significant contributions in the different areas of Plant Pathological research on the mandate crops coconut, cocoa and arecanut. The major research achievements include identification of the pathogens responsible for the diseases of mandate crops, development of disease diagnostics, development of molecular data base for rapid identification of Pathogenic organisms, understanding survival and nature of spread of pathogens in the plantations and development of integrated disease management practices with a focus on ecofriendly apporaches to maintain sustainable production and productivity. These crop health management technologies have made a remarkable impact among the farming community. The progress made on Plant Pathology research on mandate crops is summarized in this monograph.

2. COCONUT DISEASES

Coconut *(Cocos nucifera* L.) is grown over an area of 17,95,500 ha. accounting to the production of 1,39,67,900 nuts. In India, major coconut area is confined to West Coast and East Coast regions. Coconut palm, despite its hardy nature, is affected by a number of diseases. A wide range of fungi attack different parts of coconut namely, crown, stem and root (Table 1). Among the 173 fungal species reported on coconut (Joseph and Radha, 1979), only a few cause serious disease problems and are difficult to control effectively; some of which not only reduce yield but also kill the palms. Root (wilt) disease, bud rot, basal stem rot (Ganoderma wilt or Thanjavur wilt), stem bleeding and leaf rot are the major diseases causing heavy crop losses in India.

Table 1. Diseases of coconut

Name of disease	Causal organism	Parts mostly affected
Phytoplasmal diseases		
Root (wilt)	*Phytoplasma*	Leaves, inflorescence and roots
Tatipaka	*Phytoplasma*	Leaves, roots, trunk, nuts, spathes
Major fungal diseases		
Basal stem rot	*Ganoderma lucidum* *G. applanatum* *G. boninense*	Leaves and trunk
Stem bleeding	*Thielaviopsis paradoxa*	Trunk
Leaf rot	*Colletotrichum gioeosporioides* *Exserohiium rostratum*	Spindle leaves
Bud rot	*Phytophthora palmivora*	Apical bud
Minor fungal diseases		
Grey leaf spot	*Pestalotiopsis palmarum*	Leaves
Alternaria leaf spot	*Alternaria alternata*	Leaves
Anthracnose	*Colletotrichum gloeosporioides*	Nuts

Leaf spot	*Curvularia inequalis* *C. palmarum* *C. maculens* *Botryosphaeria rhodina* *Lasiodiplodia theobromae*	
Petiole rot	*Anthostomella cylindrospora*	Petiole
Spear rot	*Fusarium* sp. *Diplodia* sp.	Spear leaves
Other leaf spots	*Cladosporium cladosporioides* *Botryosphaeria rhodina* *Simatosporium falcatum* *Phomopsis cocos* *Asochyta cocoina* *Periconia saraswatipurensis* *Leiosphaerella longispora*	Senescing leaves Fronds and seed nuts
Nutritional Disorders Crown choke	Boron deficiency	Crown
Post —harvest diseases	*Aspergillus niger* *A. glaucas* *A. flavus* *A. fumigatus* *A. ochraceous* *Ceratostomella adiposum* *Botryodiplodia theobromae* *Rhizopus oryzae* *Penicillium frequentas* *P. citrinum* *Endosporostilbe* sp. *Drechslera hawaiiensis* *Bacillus* sp.	
Diseases of uncertain etiolgy Button shedding and immature nut fall	Cause not known	Button and immature nut

2.1. ROOT (WILT)

The work done on root (wilt) disease (RWD) has been reviewed recently by Solomon *et al.* (1999). Root (wilt), also known as Kerala wilt or Kattuveezhcha in Malaylam, is a non-lethal but debilitating disease of coconut. Palms of all age groups are susceptible to infection. The disease was significantly evident after the great floods in 1882 in three independent locations each 50-km apart in the erstwhile Travancore State. A comprehensive survey undertaken by CPCRI in collaboration with the Department of Agriculture, Kerala and other agencies indicated that disease has spread from its original foci and now it is prevalent in 4,10,000 ha in eight out of fourteen districts of Kerala. Isolated disease pockets were also noticed in the northern districts of Kerala and the bordering districts of Tamil Nadu. The disease intensity in the contiguous disease tract ranged from 1.5 per cent in Thiruvananthapuram district to 75.6 per cent in Kottayam district. The annual loss due to disease is estimated to be about 968 million nuts. The recent survey conducted by Department of Agriculture, Kerala showed that the disease incidence has been reduced by 24.0 per cent. The reasons for decline in the disease incidence is attributed to removal of diseased palms, replanting with quality seedlings, replacement of coconut with rubber and adoption of integrated disease management practices developed by CPCRI (Jacob Mathew *et al.*, 1998). Palms remain unproductive when they contract the disease in the pre-bearing age (Ramadasan *et al.*, 1971). Pillai *et al.*, (1973) reported that the disease is prevalent in all major soil types. The spread is faster in sandy loam, sandy alluvial and in heavy texture clayey soils than in laterite. The disease incidence is relatively higher in waterlogged low-lying areas adjacent to rivers and canals. The pattern of spread is erratic and occurs in jumps or leaps (Pillai *et al.*, 1973).

Symptoms : The most consistent and diagnostic symptom of the disease is the characteristic bending or ribbing of leaflets in mid-whorl, termed as 'flaccidity' (Radha and Lal, 1972). The other associated symptoms are foliar yellowing and marginal necrosis (Fig. 1). An index method for quantifying the disease giving due weightage to the three symptoms has been developed on the basis of the quantitative evaluation

Fig. 1. Root (wilt) and leaf rot disease affected palm

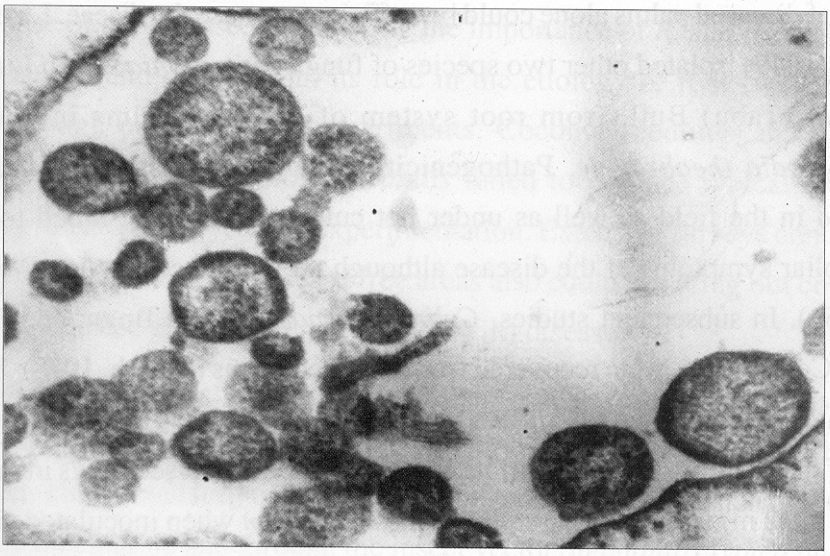


Fig. 2. *Phytoplasma* - the causal organism of RWD

of the foliar symptoms of 7000 palms of varying age (George and Radha, 1973). The formula for calculating disease index (I) is,

$$I = \frac{\Sigma\ F+Y+N \times 10.}{L}$$

where F= flaccidity with 0-5 score, Y= yellowing with 0-3 score, N= Necrosis with 0-2 score and L= total number of leaves.

Rotting of root system, drying of spathe and necrosis of spikelets are observed in certain cases (Menon and Pandalai, 1958). The nuts from diseased palms have thinner husk and fibers are weak and less firm. The kernel is thinner and remains soft and flexible. The oil content is reduced and oil loses its quality as well.

Identity of the pathogen:

The sporadic occurrence and spreading nature of the disease indicated the involvement of a pathogen as the cause of the disease.

Fungi: Butler (1906) reported that *Botryodiplodia theobromae* Pat. isolated from the roots of diseased palms alone could be sufficient to cause the diseae. Later, Menon and Nair (1949) isolated other two species of fungi, *Rhizoctonia solani* Lutu and *R. bataticola* (Tabu) Butl. from root system of diseased palms in addition to *Botryodiplodia theobromae.* Pathogenicity experiments with *R. solani* and *R. bataticola* in the field as well as under pot culture conditions, failed to produce typical foliar symptoms of the disease although they produce root rot (Menon and Nair, 1951). In subsequent studies, *Cylindrocarpon effusum* Bugn. and *Fusarium equiseti* (Corda) Sacc. were recovered from diseased palms (Joseph, 1978). Sosamma and Koshy (1978) isolated *C. effusum* and *C. lucidum* from the burrowing nematode lesions of the roots. Pathogencity trials conducted on coconut seedlings in microplots (1.8 x 1.8 x 1.2 m) with fungi *F. equsetii* and *C. effusum* when inoculated singly and in combination with burrowing nematode, *Radopho/us similis* failed to produce root (wilt) symptoms (Joseph and Lily, 1998). However, these fungi produced toxin in

the culture filtrate and induced root rot in the roots of inoculated seedlings (Lily, 1979, 1981a and b; 1983; Lily and Jayasankar 1974).

Bacteria: Menon and Nair (1951) isolated two bacteria from the roots of root (wilt) affected palms. Srivastava *et al.* (1969) reported the isolation of *Pseudomonas* sp. from the roots of diseased palms. Using an enriched medium, George *et al.* (1976) reported the isolation of *Enterobacter cloacae* (Jordan) Hormaeche and Edwards from the stelar portion of the root tips of the diseased palms. The coconut isolate of *E. cloacae* failed to produce symptoms characteristic of the disease on coconut seedlings in the pathogenicity experiments thereby ruling out its involvement in the incidence of the disease.

Nematodes: Studies on the nematodes present in the coconut roots and the soil around led to the identification of 35 genera of phytonematodes including *Xiphinema, Longidorus* and *Trichodorus,* known virus vectors and the burrowing nematode *Radopholus similis* (Mathen, 1969; Mathen *et al.*, 1970). Weischer (1967) opined that low population density of nematodes, their widespread occurrence and their general distribution pattern could exclude the nematodes from being considered as primary cause of the disease. Considering the importance of *R. similis,* based on its wide occurrence and distribution, its role in the etiology of root (wilt) has been assessed through pathogenicity experiments. Coconut seedlings in field tanks inoculated with upto one million *R. similis* failed to produce typical root (wilt) symptoms even after four years of experimentation. Extensive surveys conducted in root (wilt) diseased areas and disease free areas also could not bring out correlation between its presence and occurrence of root (wilt) disease.

Virus: Based on the systemic nature of the disease and the resemblance of symptoms to other known plant virus diseases, Nagaraj *et al.,* (1954) suspected virus as the causal agent. This gained further significance with positive transmission of the disease through sap inoculation and through the insect vector *Stephanitis typica* under field condition (Nagaraj and Menon, 1956; Shanta and Menon, 1961; Shanta *et al.*, 1964).

Shanta and Menon (1960) reported cow pea as good indicator host for the suspected coconut wilt virus as malformation and crinkling was noticed on trifoliate leaves of cowpea plants, which were inoculated with sap of the diseased palms. The physical properties of virus were determined using cowpea as the test plant (Shanta and Menon, 1961).

Holmes (1965) obtained positive results of cowpea inoculation test and suggested that the sap transmissible agent might be a virus-like organism similar to spirochete of sporozoa, in view of the peculiar nature of symptoms on cowpea and lack of proof on its passage through bacterial filters. The symptoms on cowpea were found highly inconsistent in subsequent studies. Further studies ruled out the use of cowpea plant as a reliable indicator test plant (Sasikala and Pillai, 1978). Summanwar *et al.* (1969) reported the isolation of virus from *Chenopodium amaranticolor* Coste and Reynier, which was infected with purified fraction from diseased coconut leaf. Based on its positive reaction to antisera of three different strains of tobacco mosaic virus (TMV), it was identified as a strain of TMV (Summanwar *et al.*, 1969). However, Solomon and Sasikala (1980) through serological studies, have ruled out the association of TMV with root (wilt) disease of coconut. Maramorosch and Kondo (1977) reported the presence of icosahedral particles of 56 nm diameter in the epidermis and ground parenchyma cells of diseased palms. Later, Parthasarathy (1978) identified these particles as plasmodesmata sectioned in tangential plane. Polyacrylamide gel electrophoretic analysis of isolated nucleic acids from diseased palms did not indicate association of viroid either (Randles and Hatta, 1980). Failure to observe any virus/ viroid particles, associated with disease rules out a virus / viroid as etiological agent for the disease.

***Phytoplasma*:**

Electron microscopy (EM) : Disorganisation and degeneration of vascular tissues in the diseased palms imply a vascular limited pathogen (Govindankutty and Vellaichamy, 1983). Solomon *et al.* (1993) reported the presence of *Phytoplasma* in

the sieve tubes of juvenile tissues such as sub-meristem, petiole of developing leaves, rachilla of unopened inflorescence and root apices of diseased palms. Tissue from healthy palms is devoid of such organisms. Constant association of *Phytoplasma* with the disease has since been established with the finding that the organism is present in tissues of all the seventy diseased palms as against their total absence in an equal number of healthy palms. The phytoplasmas are found in increasing numbers in the sieve tubes in the sink region. Pleomorphic forms ranging from circular to oval and occasionally beaded or filamentous forms were also observed. The coccoid forms are in size range of 200-450 nm; bounded by a triple layered unit membrane and have well defined internal structures such as ribosomes and DNA strands. Generally, they occupy parietal position close to the sieve area (Fig. 2).

Light microscopy: Govindankutty (1981) reported the occurrence of phloem anomalies in both roots and pinnae of palms affected with the disease. In subsequent studies, abnormal bluish coloration in sieve tubes of diseased palms following Diene's staining and increased fluorescing sites in sieve area consequent to 4,6 diamidino- 2 phenylindole-2 HCl (DAPI) staining were observed (Solomon *et al.*, 1987). These histochemical reactions indicative of accumulation of DNA in extra nuclear sites showed the presence of *Phytoplasma*. Such reaction was not evident in healthy palms. The occurrence of the reactions at scattered loci suggests uneven distribution of *Phytoplasma*. Similar observations were made under EM also.

Antibiotic therapy: Since phytoplasmas are not amenable to culture *in vitro,* differential chemotherapy is widely advocated as a tool to establish phytoplasmal etiology of the disease. To ensure that the antibiotic reaches the target site in unaltered form within a reasonable time, a pneumatic pressure injector was developed (Pillai and Raju, 1985). Residue analysis of the antibiotic through bioassay revealed its presence in the foliage within 24 h of application and its retention for more than 12 weeks with concentration reducing to minimum with the passage of time (Chowdappa *et al.*, 1989). A field trial was initiated with four concentrations (1, 2, 3 and 6 g / palm) of oxytetracycline hydrochloride (OTC; Terramycin tree formulation of M/s.

Pfizer India Ltd.) a single concentration each of neomycin, penicillin and distilled water control. Fifty three per cent of palms treated with 3 and 6 g a.i of OTC showed remission of symptoms. Contrastingly, palms in the distilled water and penicillin treatments deteriorated significantly over the pre- treatment condition (Pillai *et al.*, 1991). Thus, the remission of symptoms in OTC treated palms lent additional support to the etiological role of *Phytoplasma* in root (wilt) disease. Although application of OTC cannot be recommended for the control of disease due to possible environmental and health hazards, the results definitely provided an additional supporting evidence to establish the phytoplasmal etiology.

Transmission: The constant presence of *Phytoplasma* in the sieve tubes of diseased palms warranted search for insect vector(s) that transmit the disease in nature. Earlier transmission experiments (Nagaraj and Menon 1956; Shanta *et al.*, 1964) showed the role of lace bug, *Stephanitis typica* Distant being the single major group of insects on coconut, was suspected as a vector of the disease (Mathen, 1985). The report on the association of *Phytoplasma* with the disease necessitated reinvestigation on the vector role of lace bug, as true bugs (heteropteran insects) are not known to be *Phytoplasma* transmitters. Leafhoppers, plant hoppers and rarely psyllids mostly transmit Phytoplasmas. A systematic inventory of all insect visitors, to coconut garden made using various traps, and confirmation of their occurrence in coconut foliage by direct examination over a period of two years, led to the identification of a leaf hopper *Sophonia greenii* Distant, a plant hopper, *Proutista moesta* Westwood and lace bug (Rajan and Mathen, 1984, 1985). There was no disease occurrence independent of all the three insects. The potential of these insects to acquire the organism was verified using EM. *Phytoplasma* was observed in brain and salivary glands of lace bug given an acquisition plus incubation period ranging from 18 to 23 days (Mathen *et al.*, 1987). Phytoplasmas were not observed in lace bugs collected from disease free areas such as Kasaragod and Minicoy in Lakshadweep, and also in bugs offered acquisition plus incubation periods of less than 18 days (Mathen *et al*, 1987). In the light of the detection of phytoplasmas in the tissues of root (wilt) affected

palms and infective lace bugs, the insect transmission experiment was repeated under insect proof condition with improved techniques. Two year old West Coast Tall (WCT) coconut seedlings planted in methyl bromide fumigated soil held in field tanks and protected with insect proof cages were inoculated with field collected and infective lace bugs. Positive serological reaction was observed in three of the four inoculated seedlings nine months after inoculation, and weak reaction in the fourth indicating the disease contraction. Light microscopy of root tissues subjected to Diene's staining; DAPI and Hoechst 33258 fluorochromes also indicated phytoplasmal infection. EM observation also confirmed the presence of phytoplasma in all the four lace bug inoculated seedlings between 9 and 27 months after the inoculation. By the 17th month, two of the seedlings developed 'flaccidity' of leaflets, the diagnostic and decisive symptom of the disease (Mathen *et al.*, 1990). However, in control seedlings there were no symptoms and no phytoplasma was observed either.

Apart from the direct evidences accrued on the vector role of lace bugs, a number of indirect evidences also lent support. Lace bugs were found colonising in increasing number towards the inner leaves (Mathen *et al.*, 1969). This pattern of distribution enhances the chances of the organism being acquired more efficiently by the bug since active forms of *Phytoplasma* in higher concentrations were found in tender tissue. Mathen (1982) also reported that the number of lace bugs in diseased palms was four times that in symptomless palms. There was a direct linear correlation between the number colonising the palms and percentage of fresh incidence of disease. Transverse sections of coconut leaf with lace bug fixed by a cold immobilisation technique revealed the termination of the stylet in phloem, thereby confirming the ability of the Insect to pick up the phloem delimited organism (Mathen *et al.*, 1988). These direct and indirect evidences unequivocally establish the role of lace bug as the vector of the disease. The vector role of the plant hopper was also experimented in two-year-old WCT coconut seedlings, under insect proof condition. *Phytoplasma* was observed in six out of the eight plant hopper inoculated seedlings 5-24 months after inoculation. Five seedlings exhibited the diagnostic symptom of the disease,

thus confirming the vector role of the plant hopper (Solomon, 1994; Solomon *et al*, 1998; 1999).

Dodder transmission: Experimental transmission of the disease to phytoplasmal indicator host, periwinkle (*Catharanthus roseus*), was successful through the dodder laurel, *Cassytha filiformis*. Periwinkle plants grown in nylon net cages bridged to diseased coconut seedlings through the dodder laurel exhibited chlorotic spots in the interveinal areas and at vein endings of fully opened leaves. EM examination of diseased source palm, connecting dodder laurel and periwinkle showed the presence of phytoplasma in all the three samples. Phytoplasmas could also be transmitted from periwinkle to periwinkle (Sasikala *et al*, 1988).

Culturing: Attempts to culture the *Phytoplasma* in cell-free media were not fruitful so far. More than forty different media with various combination of growth factors, nucleic acid precursors, co-factors, vitamins, sera, nutrients, embryonated hen's eggs and vascular sap enriched preparations were used for the culturing of the organism from the tissues of diseased coconut, symptomatic periwinkle and infective lace bugs (Solomon *et al.*, 1998; 1999). None of the media either could support the growth or ensure the long-term maintenance. However, phytoplasmas could be maintained in rachillae explants from diseased juvenile coconut palms for more than 6- 8 weeks in certain plant tissue cuture media.

Non- cultivable nature of *Phytoplasma* is a limiting factor in fulfilling Koch's postulates. Nevertheless, manifestation of disease symptoms through inoculation of insect vector(s) rendered infective and differential chemotherapeutic response of diseased palms to penicillin and tetracycline are considered adequate to offer the best circumstantial evidence to prove the phytoplasmal etiology.

Diagnosis: The coconut root (wilt) diseased palms were mainly identified based on the visual symptoms, considering flaccidity as the primary symptom. As the time lag between infection and manifestation of symptoms vary considerably, there is a need to develop reliable diagnostic tests, which could detect the palms at very early stages

of infection even before the visual symptoms are apparent. Biochemical tests based on differential dehydrogenase activity (Joseph and Shanta, 1963), accumulation of free aminoacids especially arginine (Pillai and Shanta, 1965), tannin (Lal, 1966) and EDTA (Dwivedi *et al*, 1977) have been developed for early detection of root (wilt). But, they did not give consistent results (Rajagopal *et al.*, 1988). Further, all these biochemical tests were based on altered host metabolism perceptible in the form of either accumulation or depletion of substance consequent to differential enzymatic activity, which could be induced under varying conditions. Immuno-format assays based on agar gel double diffusion (Shanta, 1971; Solomon, 1994), Enzyme-linked iniinunosorbent assay (ELISA) (Sasikala *et al.*, 1998) and a physiological test based on stomatal resistance (Rajagopal and Amma, 1989; Rajagopal *et al*, 1986) have been standardised for detecting the disease. Using these techniques, the diseased palms could be detected 6 to 20 months before the expression of symptoms.

Physiology: Earlier studies attributed the root (wilt) disease to certain physiological factors, either the lack of nutrients, excess or inadequate water, disturbing the physiological process of the palms and thus predisposing them to the disease. Subsequent studies indicated that the disease is not due to physiological disorder but fungi, virus and toxic substances produced by micro-organisms living around the root zone of the palms might contribute to the disease (Varghese, 1934). Derangements in the water absorbing capacity of roots in diseased palms, the uptake and transport of water through the trunk (Ramadasan, 1964) and permeability changes in leaf and root tissues resulting in leaching of metabolites have been recorded. Rajagopal *et al* (1986) reported that the mechanism of stomatal regulation is adversely affected in diseased palms. The stomata in diseased palms failed to close in response to soil and atmospheric drought resulting in excessive water loss. The diseased palms also had lower water potential than healthy palms at any given time (Rajagopal *et al.,* 1987). Thus, impairment caused to both the facets of water transport, namely, absorption and transpiration lead to internal water stress. A reduction in photosynthetic rate (Dwivedi *et al,* 1978), higher respiration (Michael, 1978), derangement in

translocation and distribution of sugars (Mathew, 1977), an altered nitrogen metabolism (Varkey *et al.,* 1969) with an increase in aininoacid concentration in leaves particularly arginine (Pillai and Shanta, 1965), an accelerated phenol metabolism with a fall in phenol content and an increase in phenol oxidizing and synthesizing enzymes (Joseph and Jayasankar, 1973,1979; Joseph *et al.,* 1976; Joseph, 1983) are the other changes observed. Such changes encountered in diseased palms are rather suggestive of a pathogen mediated altered host metabolism than of a physiological disorder.

Soil and Nutrition: The effect of soil conditions and associated nutritional factors on the disease incidence has been under investigation since 1939. Based on detailed study of the disease in relation to the soil conditions it was concluded that the soils of disease affected areas are generally poor in major nutrients especially potash and the soils are mostly acidic with less content of exchangeable bases and poor base exchanging capacity (Menon and Nair, 1949; Pandalai *et al.,* 1958 a, b). An accumulation of major nutrients and silica in the leaves of diseased palms was also noticed. However, no perceptible difference in micronutrient level was evident. Khan *et al.* (1985) could not find any relationship between micronutrient content and disease incidence. Cadmium and strontium toxicity was also ruled out (Verghese *et al.*, 1962 a, b). Although higher content of Ba, Cr, Cd, Pb, Sr, and V in the disease affected soils (Biddappa and Khan, 1985), Al, Mn, Cu, Co, Ni and Sr in roots of diseased palms and Cr, Ti, Pb, and Ga in the cabbage tissues (Biddappa and Cecil, 1984; Biddappa, 1985) were noticed, their role in disease causation has not been established.

The possibility of involvement of major nutrients in the incidence of the disease has been ruled out based on detailed analysis of soil and tissue samples from healthy and diseased tract of all the major coconut growing areas (Pillai *et al.,* 1975). Cecil (1975) observed the Ca and Mg contents of healthy palms significantly higher than those of apparently healthy palms and diseased palms. According to him, the palms in the root (wilt) affected areas are in the state of unbalanced nutrition as compared to healthy palms. However, fertilizer field trials with the application of different

levels of major and secondary nutrients, Ca and Mg could neither control the disease nor prevent fresh incidence although a general improvement in yield was observed (Chettiar *et al.*, 1959; John *et al.*, 1959; Lal, 1966). The role of micronutrients in the incidence of the disease has also been ruled out (Davis and Pillai, 1966).

Varietal resistance: As the disease can not be controlled by conventional plant protection measures the development of resistant tolerant variety to root (wilt) is an ideal solution to this malady. Investigations on identifying coconut genotypes resistant tolerant to root (wilt) disease were initiated in 1934 (Varghese, 1934). Trials conducted from 1951 to 1968 have indicated that open pollinated progenies from healthy palms from disease endemic areas had lesser disease incidence (Menon *et al.*, 1981). Field evaluation of 45 cultivars and 62 hybrid combinations were planted in 1972 in the Institute farm at Kayangulam and farmer's garden to identify genotypes resistant tolerant to root (wilt) disease. None of the cultivars/hybrids showed the desired level of resistance tolerance to the disease. However, the hybrid Chowghat Orange Dwarf (COD) x West Coast Tall (WCT) under good management conditions yielded higher nuts (80 nuts / palm / year with 50% disease incidence) compared to WCT (70 nuts / palm / year with 37.5 % disease incidence) palms of identical age in the early years of production (Bavappa *et al.*, 1986). Another field trial involving 27 cultivars, 10 hybrid combinations, F_2 (OP) of D x T and T x D progenies of elite palm, high yielding WCT and prepotent WCT laid out in 1982 at five locations indicated that all the cultivars were susceptible to the disease except Kenthali Dwarf. A large-scale field trial involving 33 tall, six dwarfs and nine hybrid combinations was planted in Kayangulam Kayal farm in 1985. Of these, 12 cultivars and five hybrids combinations have contracted the disease. Among the 10 cultivars and four hybrids planted during 1986, the disease affected Markham Tall and Fiji Tall.

Of the nine elite palms identified in the disease tract (Iyer *et al.*, 1979), three palms (Thazhava, Champakulam and Krishnapuram) have contracted the disease. But these palms showed superiority over the others in yield, ranging from 96 to 156 nut / palm / year. An intensive survey conducted in 1985 in the 'hot spot' disease areas of

Kottayam, Alappuzha, Pathanamthitta and Kollam districts of Kerala initiated resulted in identification of 187 mother palms of WCT and Chowghat Green Dwarf (CGD). Using these palms, breeding for resistance/tolerance to disease initiated in 1987 is showing encouraging results, indicating the evolving of resistant/tolerant high yielding CGD x WCT and WCT x WCT hybrids for combating the root (wilt)) disease (Jacob *et al.,* 1998). Seed gardens one each at CPCRI Research Centre, Kannara and Coconut Development Board farm at Neriamangalam have been established for large scale production of disease tolerant planting materials such as WCT x WCT, WCT x CGD and CGD x WCT.

Disease management: Root (wilt) is not a lethal disease but a debilitating malady and responds to good management. Yield of the palms can be sustained or even improved through the adoption of integrated management practices consisting of balanced fertilizer application, addition of organic matter, raising and incorporation of green manure crops in basin, irrigation during summer months, leaf rot control and adopting inter and mixed cropping (Bavappa *et al.,* 1982). Multiple cropping in diseased gardens involving the raising of fodder crops in the interspaces and maintaining milch cows and recycling of organic wastes has helped in increasing the mean yield of palms by 26 per cent (Sahasranaman *et al.,* 1983). Mixed cropping with cocoa increased the yield by 27 to 35 per cent and reduced the decline of the palms (Nair *et al.,* 1975; Amma *et al,* 1983). Similarly, intercropping with tapioca, elephant foot yam and yams in the interspaces of disease affected coconut gardens for a period of three years increased the nut yield by 5, 15 and 8 per cent respectively (Menon and Nayar, 1978). The yield of the diseased palms increased by growing and incorporation of cover crops, such as *Puereria phaseoloides, Mimosa invisa* and *Calopogonium mucoides,* in the basins (Thomas *et al.,* 1993). Irrigation of diseased palms during summer months is also found beneficial in improving both the health of palms and nut yield (Rajagopal *et al.,* 1987). Removal of diseased palms in mildly affected areas help in arresting further spread of the disease. Removal of diseased palms at two isolated pockets one at Shencottah (Tamil Nadu) and another Nadathara

(Kerala) prevented the recurrence of the disease (Radha *et al., 1985).* Based on these results, a field station was established at Trichur district in 1979 to arrest the fitrther spread of the disease to the north and contain in its geographical limits (Radha *et al.,* 1981). With eradication of 400 diseased palms from 209 gardens in eight villages in Trichur district, 92.4 per cent of the gardens were free of the disease even after four years. The strategy for managing the disease is to eradicate all diseased palms in areas of sporadic incidence, removal of disease advanced in the heavily diseased tract, replanting with high yielding hybrids of CGD x WCT and adoption of recommended package of practices.

2.2. LEAF ROT

Radha (1961) first coined the name leaf rot for foliar necrosis found in the root (wilt) tract of Southern Kerala. Since the beginning of the century, it is well established that palms affected by root (wilt) are generally superimposed by leaf rot disease (Sundararaman, 1925; Varghese, 1934; Nagaraj and Menon, 1956; Radha *et al.,* 1962; Radha and Lal, 1968; Srinivasan, 1991). The palms weakened by *Phytoplasma*, the causal agent of root (wilt) might result in the break down of defence mechanism leading to susceptibility to leaf rot disease.

Symptoms: Leaf rot starts as minute, water soaked lesions on the emerging spindle with different shades of colour and shape. These lesions enlarge, coalesce freely leading to extensive rotting (Fig. 3). The rotted portions dry up, turn black and fall off. Tips of leaflets and midribs often become blackish and shriveled. The progress of rotting slows down with the maturity of the leaflets. The inner whorls of leaves are vulnerable to the disease. Continuous attack of newly emerging spindle leaf 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 the yield. Palms of all ages are susceptible to the infection (Radha and Lal 1968; Srinivasan and Gunasekaran, 1992;

Fig. 3. leaf rot disease

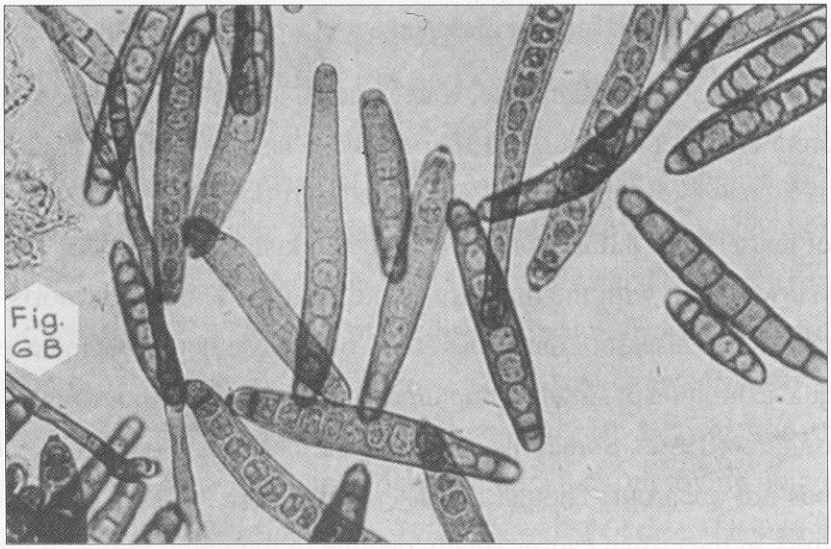
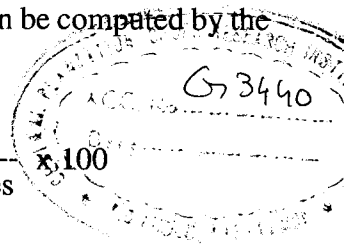


Fig. 4. Conidia of *Exserohilum rostratum*

2000).

Disease indexing: Srinivasan and Gunasekaran (1996 a) developed a formula for leaf rot disease quantification with a four-point scale, 0-(No infection) 1-(Upto 25) 2-(26-50), 3-(51-75) and 4-(above 75 %). The disease index can be computed by the following formula:

$$\text{Disease index (DI)} = \frac{\text{Total numerical ratings}}{\text{No. leaves x Max. No. grades}} \times 100$$



Yield loss: Crop loss due to leaf rot alone is not available as it is superimposed with 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 as it is prevalent in 0.41 million ha (Nambiar and Rawther, 1993).

Identity of pathogen: Fungi like *Bipolaris halodes* (*Helminthosporium halodes), Gloeosporium* sp., *Gliocladium roseum,* and *Pestalotiopsis palmarum* have been isolated from the leaf rot affected palms (Menon and Nair, 1948; Radha and Lal, 1968). Pathogenicity experiments revealed that *B. halodes* caused infection within 12 h and remaining in 48 h (Menon and Nair, 1948). According to them, *B. halodes* was the most virulent pathogen and the rest were a secondary invader, aggravating the rotting initiated by *B. halodes.* Later, Radha and Lal (1968) also confirmed the infectivity of *B. halodes* on coconut. Subsequent studies indicated 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. (Fig. 4), *Gliocladium vermoeseni* (Biourge) Thom, *Cylindrocladium scoparium* Morgan, *Fusarium solani* Martius (Sacc), *F. moniliformae* Sheldon var. *intermedium* Neish and Legget, *Thielaviopsis paradoxa* (Dade) C. Moreau, *Rhizoctonia solani* Kuhn, *Mortierella elongata* Linnem, *Curvularia* sp., *Acremonium* sp., *Thielavia microspora* Mouch., *T. terricola* (J.Gilman and E.V.Abbott) Emmons

and *Chaetomium brasiliense* Batista and Pont (Srinivasan and Gunasekaran, 1993; 1994 a and b; 1995 a and b; 1996 a, b, c and d; 1998 a; Srinivasan *et al.,* 1995). Of these, *C. gloeosporioides* and *E. rostratum* are considered as main pathogens of leaf rot disease based on their frequency of occurrence and pathogenicity (Srinivasan and Gunasekaran 1998).

Epidemiology: The tender leaf was most susceptible (Lily, 1963). The susceptibility of the seedlings decreased with age. Seedlings up to 19 months may get severe infection. Leaf rot infection was more severe during the season when the conditions of high humidity were prevalent (Menon and Nair, 1951). Severity of leaf infection by *H. halodes* was found to be correlated with high temperature and low humidity present during monsoon period (Radha *et al.,* 1961 and Radha and Lal, 1968). The population dynamics of leaf rot disease pathogens in relation to environmental variables was studied by monthly isolations from the spindle leaves of diseased palms (Srinivasan and Gunasekaran, 1996c). The incidence of *C. gloeosporioides* was higher in frequency and population during monsoon with a peak in June-July. Its incidence was positively correlated with rainfall and relative humidity 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* sp. and *R. solani* were isolated most commonly during the dry season of January- May.

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

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 per cent to 7.8 per

cent. Spraying the leaves sequentially with Bordeaux mixture (1%), Dithane M-45 (0.3%) and Fytolan (0.5%) at quarterly intervals after removing severely affected leaves, was found to reduce the further incidence of the disease (Prasanna Kumari *et al.*, 1960). *In vitro* assay of contact (Indofil M-45, Fytolan, Captan and Thiram) and systemic fungicides (Contaf, Calixin and Aureofungin-sol) against *C. gloeosporioides* and *E. rostratum* indicated that Contaf exhibited a broad-spectrum activity inhibiting all the pathogens of leaf rot disease (Srinivasan and Gunasekaran, 1998). A field trial conducted for three years on 20 year old palms revealed that pouring of Calixin (1%) into leaf axil and spraying of Indofil M-45 (0.3%) along with phytosanitary practices reduced the disease intensity (Srinivasan and Gunasekaran, 1998). The bacterial antagonist *Pseudomonas fluorescens* (TNAU isolate) inhibited the growth of *C. gloeosporioides* and *F. rostratum* under *in vivo* conditions and reduced the leaf rot onset. Of the 96 phylloplane and 21 rhizophere isolates from coconut, two isolates from each from phylloplane and rhizophere have been identified as effective native antagonists against both pathogens (Srinivasan and Gunasekaran, 1998; Gunasekaran *et al.*, 2001). Radha (1961) observed that Andaman Ordinary and Papua New Guinea were most resistant than other varieties 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 few leaves close to spindle and use of contaf are the most important in controlling leaf rot (Srinivasan and Gunasekaran, 2000).

2.3. BUD ROT

Bud rot commonly occurs in almost all coconut growing countries. Bud rot was reported from India in 1906 (Butler 1906). The disease is widely prevalent in East and West coasts of India (Menon and Pandalai, 1958). Nut (fruit) rot and premature nut fall was reported to cause considerable damage. (Shaw and Sundararaman, 1914; Sundararaman and Ramakrishnan, 1924). The problem has been reviewed earlier (Radha and Joseph, 1982; Nambiar and Rawther, 1993).

Crop loss: An incidence of 0.1 to 6.5 per cent in Kerala and 0.4 to 6.7 per cent in Tamil Nadu has been reported (Radha and Joseph, 1974). Occasionally, heavy disease incidence about 35-40 per cent has been observed in certain areas of Kerala and Karnataka. During 1992, disease incidence was about 16.4 per cent in Calicut district where about 5000 palms were affected (Nambiar, personal communication).

Symptoms: Unopened tender leaf or spindle is affected leading to rotting of bud and death of palms. The first symptom is pale yellowing and withering of the tender leaf with time it turns brown and bends off. Rotting starts from outer region and gradually extends inwards showing varying degrees of brownish to pink discoloration and rotting (Fig. 5). Affected tissues emit foul smell. Even though the palm may not die immediately, it succumbs finally due to loss of apical bud. The older nuts persist on the crown for some time, while the younger ones may fall off (Radha and Joseph, 1974).

Identity of pathogen: The causal agent of both bud rot as well as fruit rot has been identified as *Phytophthora palmivora* (Butler, 1906; Sundararaman and Ramakrishnan, 1924) and belonged to A2 mating type. Heterothallic nature of *P. palmivora* has been reported earlier (Thomas *et. al.,* 1947) Though primary infection is caused by *P. palmivora,* subsequent colonisation by bacteria enhances rotting (Radha and Joseph, 1974). The hyphae of *P. palmivora* are of uniform diameter (3-8 μm); smooth without hyphal swellings. The sporangia are ellipsoid or ovoid with rounded base, measuring 35-60 x 20-70 μm. the sporangia are borne on simple sympodium. They are caducous and carry a short (< 5 μm) and thick pedicel. Zoospores are released from the sporangia at 22-24 ^{0}C, and are biflagellate and motile in nature. Chlamydospores (30-40 μm) are found in abundance. Sex organs (both antheridia and oogonia) have been reported in nature. Oospores are spherical (16-30 μm) and appeared within 4 weeks in inoculated coconut petioles (Radha and Joseph, 1974). The secondary saprophytic colonisers like *Fusarium, Xanthomonas, Pseudomonas* and *Erwinia* were commonly isolated from bud rot affected palms

Fig. 5. Bud rot disease

Fig. 6. Stem bleeding disease - symptom on trunk

and they enhance the rotting.

Epidemiology: The disease is generally noticed during both South West and North East monsoon periods when wet weather prevails and younger palms are more vulnerable (Menon and Pandalai, 1958). Detailed epidemiological investigations showed that temperature of 21- 24° C and relative humidity of 98-100 per cent were found highly congenial for the disease development and such 'favourable days' determined the disease incidence. Incubation period of 35 days was found essential for manifestation of the disease and micro climatic factors at the axil of the leaves were considered important (Radha and Joseph, 1974; Joseph and Radha, 1975.) Rainfall appears to be an important factor and heavy disease incidence that prevailed during October 1992 was attributed to high rain fall when about 5000 palms of 25-30 years age group were affected in Kuttiady area of Calicut district of Kerala. The fungus was reported to survive at the base of the fronds in the crown during dry months (Menon and Pandalai, 1958). Survival of the fungus up to five months in the affected crown was reported and also formation of oospores when the cultures were contaminated with *Thielaviopsis paradoxa* (Radha and Joseph, 1974). These oospores if found in crown might serve as survival mechanism of the fungus.

Host range: Coconut isolates of *Phytophthora* were pathogenic to palmyrah *(Borassus flabellifer),* oil palm *(Elaeis guineensis),* rubber *(Hevea brasiliensis),* Jack *(Artocarpus integrifolia)* and cocoa *(Theobroma cacao)* (Menon and Pandalai, 1958; Radha and Joseph, 1974; Chowdappa *et al.,* 2000).

Symptoms: The disease is characterised by decaying of immature nuts and their fall during the rainy season. Water-soaked greyish green area develops at the stalk end of the nuts against dark green healthy area around. The lesion development is faster at the stalk end of nuts. The lesions turn brown and become sunken due to decay of underlying tissues. The rot extends into the husk and sometimes deep into the endosperm cavity if the shell has not hardened.

Identity of pathogen: The fungus, *P. palmivora* has been reported as causal agent

of fruit rot (Sarma *et al.*, 2000). *Phytophthora meadii* Mc Rae has also been found to Cause the immature nut fall for the first time in Kodagu district of Karnataka (Chowdappa *et al.*, 2002 a). This culture showed morphological similarities and had identical ITS-RFLP patterns to reference cultures of *P. meadii.* The fungus exhibited chrysanthemum pattern of growth with no aerial mycelium. The growth rate of fungus was 12 mm day. Isolate produced papillate sporangia with round base, ellipsoid, obpyriform and occasionally spherical in shapes. Sporangia were caducous and shed with thin and narrow stalks. The length of the sporangial pedicel was 24 μm. The sporangial dimensions of this isolate was 40 x 25 μm with L / B ratio of 1.61. The isolate belonged to A2 mating type. The size of oogonia was 28.20 μm and oospores were 24.63 μm. The antheridia were amphigynous (13.18 x 12.36 μm in size). The isolate did not produce chlamydospores. The cardinal temperatures were 12, 24-27 and 30 °C respectively. The isolate consistently produced symptoms similar to those observed in natural infections on Chowghat Orange Dwarf coconuts (COD) and the fungus was re- isolated from the inoculated nuts. In cross inoculation studies, it infected arecanut and rubber but not cocoa and black pepper. Additional research is needed to determine the distribution and economic importance of the *P. meadii* on coconut and its relationships with arecanut, cardamom and rubber isolates.

Management: The disease can be effectively controlled by giving prophylactic spray with one per cent Bordeaux mixture during pre-monsoon period.

2.4. FRUIT ROT

The disease, commonly known as ' Mahali' in local languages, has been known in Kerala since 1924 (Sundararaman and Ramakrishnan, 1924).

Crop loss: A severe out break of nut rot was reported from Godavari belt of Andra Pradesh during 1986 causing 3.1 – 18.1 per cent of disease incidence and 7.41 – 53.3 per cent of nut fall (Sarma et al., 2000). Sporadic incidences of fruit rot caused by *Lasiodiplodia* were observed in Alappuzha District, Kerala (Gunasekaran and Srinivasan, 2000).

Symptoms: The disease is characterised by decaying of immature nuts and their fall during the rainy season. Water soaked greyish green area develops at the stalk end of the nuts against dark green healthy area around. The lesion development is faster at the stalk end of nuts. The lesions turn brown and become sunken due to decay of underlying tissues. The rot extends into the husk and sometimes deep into the endosperm cavity if the shell has not hardened. *Lasiodiplodia* fruit rot produce dark grey to brown lesions with wavy to undulated margins, mainly starting from the apex of the nuts (Gunasekaran and Srinivasan, 2000). The affected nuts deform, desiccate, and shrink. In some cases, the contents leak owing to splitting of such nuts. Since the infection mostly originate from the apex, the nuts remain attached to the bunches. Nuts in the age group of 6-8 months are usually affected.

Identity of pathogens: The fungi, *P. palmivora* and *L. theobromae* has been reported as causal agents of fruit rot (Sarma *et al.*, 2000; Gunasekaran and Srinivasan, 2000). *Phytophthora meadii* Mc Raer has also been found to cause the immature nut fall for the first time in Kodagu district of Karnataka (Chowdappa *et al.*, 2002 a). The culture showed morphological similarities and had identical ITS-RFLP patterns to reference culture of *P. meadii.* The fungus exhibited chrysanthemum pattern of growth with no aerial mycelium. The growth rate of the fungus was 12 mm / day. Isolate produced papilate sporangia with round base, ellipsoid, obpyriform and occassionaly spherical in shapes. Sporangia were caducous and shed with thin and narrow stalks. The length of the spoirangial pedicel was 24μm. The sporangial dimensions of this isolate was 40 X 25 μm with L / B ration of 1.61. In cross inoculation studies it infected arecanut and rubber but not cocoa and black pepper.

Management: The disease can be effectively controled by giving prophylactic spray with 1% Bordeaux mixture during the pre-monsoon period.

2.5. STEM BLEEDING

Stem bleeding was reported from Sri Lanka in 1906. At present, it is prevalent in India, Philippines, Malaysia, Trinidad, Papua New Guinea, Indonesia, Bangladesh

and Mexico. It was first reported from India in 1922. Stem bleeding disease is a debilitating disease of coconut, which is prevalent in almost all the coconut growing areas of India.

Crop loss: A preliminary survey conducted in Kasaragod and Kannur districts of Kerala state during 1988-90 indicated disease incidence of 8.8 per cent. The yields of stem bleeding affected trees in these areas were less (37-40 nuts / palm / year) compared to the yields of healthy palms (56 nuts / palm / year).

Symptoms: The typical symptom of stem bleeding is the exudation of reddish brown gummy fluid from growth cracks on the trunk, which becomes black on drying (Fig. 6). In the initial stages of infection, bleeding symptoms appears only one or two longitudinal cracks at the base and later spreads upward on the stem. The longitudinal patches may coalesce to form larger patches. In advanced stages of infection, the bleeding patches may extend up to the crown. The tissue inside the lesions shows discoloration and decay. The internal damage is confined to the hypodermal region. However, in young palms under cooler conditions, the decay may extend into the deeper layers of central cylinder (Menon and Pandalai, 1958; Radha, 1962; Nambiar *et al.,* 1986; Nambiar and Rawther, 1993). As a result of extensive damage in the stem tissue, the outer whorl of the leaves turn yellow, dry and shed prematurely. The trunk tapers and reduces the crown size. In the advanced stages, scoletid beetles like *Diocalandra* and *Xyleborus* infest the plams and further weaken the stem. Jacob Mathew *et al.,* (1989) developed a formula for assessing the disease severity in stem bleeding affected coconut palms.

Identity of pathogen: The etiology of the disease was not established for a long time. Lily (1984 a, b) isolated *Phomopsis cocoina* Cke. (Punith) and *Schizophyllum commune* Fr. from the stem bleeding affected palms but their pathogenicity was not established. Although, Menon and Pandalai (1958) suspected *Thielaviopsis paradoxa* (de Synes) as incitant of stem bleeding. Nambiar *et al.,* (1986) established the etiological nature of *T. paradoxa* in the disease through artificial inoculations. Later,

perithecial stage of the causal agent *Ceratocystis paradoxa* (Dade) Moreau has been isolated from infected trees. *T. paradoxa* produces pale brown to brown hyphae. Conidiophores are slender, arising laterally from the hyphae and produce cylindrical to oval endoconidia; when mature they are hyaline to pale brown, smooth walled (6-24 x2-5.5 μm) Chalmydospores terminal in chains, obovate, thick walled, brown, 10-25x7.5-20 μm. The perithecial stage is *Ceratostomella (=Ceratocystis paradoxa);* perithecia partly immersed, light brown, 190-350 μm dia with numerous appendages; long and black neck, tapering upto 1400 pm, osteolar, hyaline; ascospores ellipsoid, often with unequally curved sides, hyaline, non-septate, smooth, 7-10 x 2.5-4 μm. The optimum temperature for mycelial growth was reported to be 30 ^{0}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 *P. paradoxa* from the diseased tissues. A baiting technique was also developed for the isolation of *P. paradoxa* from the infected soils using sterile frond bits as baits.

Pathogen variability: Gowda (1987) recorded variability among the five isolates derived from five localities in Karnataka and Kerala 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 seven isolates based on their temperature, pH, carbon, nitrogen and vitamin requirements. Some of the isolates exhibited a characteristic fruity smell. Ramanujam and Nambiar (1996) distinguished two sub groups among 12 isolates of *P. paradoxa* based on their pathogenic reaction to detached coconut petioles corresponding electrophoretic sub groups.

Host resistance: All the 26 coconut cultivators (16 talls, six dwarfs and four hybrids), that were tested against to *P. paradoxa* using detached petiole technique were found susceptible with varying degree 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 were more susceptible.

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 attack, and insect attack *(Diocalandra* and *Xyleborus)* are the predisposing factors responsible for disease development as this fungus is a weak soil-borne pathogen. It enters the coconut stem tissue through growth cracks and wounds and multiplies in the host tissue, producing endoconidia and chlamydospores. The chlamydospores survive in the soil during unfavourable conditions. When the conditions are favourable, the chlamydospores in the soil germinate and become capable of infecting coconut. Radha (1962) showed tat fluctuations in soil reaction and 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 could 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 where on the banks of back waters, 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 prevail. Usman (1988) reported that maximum survival of *P. paradoxa* propagules was noticed in red loam followed by laterite soil and the least in the sandy soil. The fungus attacks a variety of hosts like arecanut, palmyra palm, banana, pineapple, sugarcane, papaya and date palm.

Management: Prior to confirmation of etiological agents of the disease, the control measures recommended mainly consisted of phytosanitation 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 condition when Bavistin or Calixin were root fed. Further studies also indicated effectiveness of these chemicals in reducing the disease incidence and increasing the yields (Anil Kumar *et a!.,* 1992; Ramanujam *et al,*

1993 a). Their results also showed the detection of residues of Bavistin 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 chemical respectively.

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 or adopting practices to conserve soil moisture are beneficial in reducing growth cracks. Application of neemcake (5 kg / palm) was found to increase soil microflora including *Trichoderma* population, which were found to inhibit the pathogen *in vitro* (Gowda, 1987) and on detached coconut leaf petiole (Usman, 1988). Indeed, 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 *P. paradoxa* and recorded rice bran: neemcake (1:1) as the best substrate for mass production of this biological control agent. Ramanujam (1997) developed integrated management practices for effective management of stem bleeding of coconut involving root feeding (100 ml at quarterly intervals) and wound dressing (50-200 ml) of Tridemorph (4%) followed by coal tar sealing (100-300 g) and soil application of *G. virens* (1 kg), neem cake (5 kg), farm yard manure (50 kg) and NPK fertilisers (500: 320: 1200 g / palm / year).

2.6. CROWN CHOKE

Crown choke, also known as crown rot, was first noticed in Assam in 1964. At present, this disorder is prevalent in parts of Assam, West Bengal, Kerala and Tamil Nadu.

Crop loss: A survey in Assam showed that about 10 per cent of the palms are affected in the state (George *et al.,* 1990). Palms in the age group of 3-6 years are generally affected.

Symptoms: The first visible symptom is the emergence of shorter leaves with deformed and crinkled leaflets. Marginal tip necrosis of the leaflets is the associated symptom. The number of leaflets decreased progressively when the attack is acute. The affected leaflets do not unfurl and in many cases give a 'choked' appearance to the frond. The tips of the leaves become hooked; the hooks are mostly seen in the terminal pinna, although they may occur in any position on the frond. In the advanced stages of infection, a severely necrotic black, stick- like leaf, stalk devoid of any pinnae emerges and necrosis takes place in the primordial tissues and kills the crown. The outer whorls of the leaves look healthy and remain green. The stem does not show any tapering. The affected palms do not die immediately and take 3-4 years to die. Premature nut fall was also noticed. The inflorescence emergence is hindered resulting in a gradual decline in yield. (Rethinam *et al,* 1990; Baranwal *et al.,* 1989).

Cause: Many attempts to isolate pathogenic fungi or bacteria from the diseased palms were not successful so far. Analysis for soil and leaf samples indicated that the palms are deficient in boron (Cecil and Pillai, 1978; Baranwal *et al.*, 1989). The palms grow normally when a certain balance exists in the intake of Ca and B. Baranwal *et al.,* (1989) showed that the Ca/B ration is significantly low in healthy palms (95) campared to diseased palms (145). They reported the disease palms had 5.4 ppm B as against 7.4 ppm in helathy palms. Pillai *et al.,* (1975) reported that 12 per cent of coconut soils in Kerala are deficient in boronh.

Management: Application of borax (50 g / palm) before monsoon has been found beneficial in improving the condition of the palm (Cecil and Pillai., 1978).

3. ARECANUT DISEASES

Arecanut (*Areca catechu* L.) is an important plantation crop grown in humid tropics of India. Traditionally, the crop is grown in the states of Karnataka, Kerala, Assam, West Bengal, Tamil Nadu and Maharashtra. It is also grown in Andaman, Tripura and Manippur to a certain extent. The nuts of arecanut palms are used for chewing purposes and in socio-religious functions in India. Although arecanut does not have majore alternate use or export potential value other than chewing, the crop is rapidly expanding as fetches more income per unit area. At present, arecanut is grown in 2,64,000 ha with an annual production of 3,13,000 tonnes. Increasing the productivity in exixiting plantations is the need of the hour rather than expanding the area under crop since India has achived self- sufficiency in production even during late 70's. Crop loss due to diseases has been identified as one of the majore production constraints in arecanut. Arecanut diseases, mostly, are fungal origin except one bacterial and a phytoplasmal disease (Table 2.). Among the diseases, fruit rot caused by *Phytophthora* and yellow leaf disease incited by *Phytoplasma* are currently the most important yield limiting factors.

Table 3. Diseases of arecanut

Name of disease	Causal organism	Parts mostly affected
Fruit rot (Koleroga / Mahali)	*Phytophthora meadii*	Nut
Bud rot	*P. meadii*	Bud
Anabe roga / Foot rot	*Ganoderma lucidum*	Root and basal portion of stem
Inflorescence die back	*Colletotrichum gloeosporioides*	Inflorescence
Leaf spot	*Phyllosticta arecae*	Leaf
Yellow leaf disease	*Phytoplasma*	Roots, leaves and nuts
Bacterial leaf spot	*Xanthomonas campestris* var *arecae*	Leaf

Disorders		
Sun scorch	Heat	Stem
Band / Hidimundigi	Soil / Physiological	Leaf
Nut splitting	Physiological	Nut

3.1 YELLOW LEAF DISEASE

Yellow leaf disease (YLD) is the most serious problem affecting arecanut industry. The disease was first reported in 1914 in Moovattupuzha, Meenachil and Chalakkudi areas of central Kerala following a heavy flood (Nambiar, 1949). The disease was also reported from the parts of Karnataka, Tamil Nadu and coastal regions of Maharashtra (Menon, 1963). The disease is commonly known as 'Kattuveezcha' or 'Chovakedu' in Malayalam and 'Chandiroga' or 'Arasinaroga' in Kannada (Nambiar, 1949; Dastagir, 1963). According to Varghese (1934), the disease had some similarities with the root and leaf disease of coconut prevalent in Kerala.

Spread of the disease: A preliminary survey conducted in 1959-60 indicated that the disease had spread to all parts of Kerala with a maximum incidence of 90 per cent in Quilon district. A comprehensive survey undertaken in 1976 in arecanut growing areas of Kerala and Karnataka revealed that the malady is prevalent in almost all the districts of Kerala in varying intensities. The maximum incidence was noticed in Idukki district where 97 per cent of the area under crop was affected. In Kerala State as a whole, 36 per cent of the crop area was affected as against 24.4 per cent in Koppa and Sringeri taluks of Karnataka. A recent survey conducted in Kasaragod and Kannur districts revealed that a total of 4618 palms in Kasaragod and 16,7 1,243 palms in Kannur district were found to be affected by YLD resulting in a loss of 2.8 tonnes and 1365 tonnes of chali respectively (Rawther, 2000). In Hosdurg taluk, 2,508 palms were affected in five villages. The total production of arecanut in the district was estimated at 19,017 tonnes of chali and the estimated loss due to YLD was 3.1 tonnes.

A garden to garden survey conducted in Karnataka involving CPCRI, University of

Agricultural Sciences (UAS), Bangalore, Department of Horticulture and Department of Agriculture, Government of Karnataka during 1989 and1990 revealed that YLD is prevalent in all arecanut growing districts. A total of 12,483 palms in Dakshina Kannada, 544 in Udupi, 2,25,937 in Kodagu, *5,15,260* in Chickmagalur, 1,92,590 in Shimoga and 2,102 in Uttara kannada districts were affected by YLD resulting in a loss of 508.3 tonnes of chali.

Apart from taking a heavy toll of the palms every year, the disease rendered arecanut cultivation uneconomical to the farmers due to reduced yield.

Symptoms: The symptom expression of yellow leaf disease (YLD) is well pronounced soon after the monsoon, when maximum temperature is 30 ^{0}C-32 ^{0}C, nights are cool and wind currents mild to heavy. Paradoxically, with the rise in temperature, the symptom expression is reduced. The intensity of yellowing of the leaves is minimum in May i.e., before the onset of South-West monsoon and maximum in August i.e., mid-monsoon (Nayar, 1976).

Rawther (1976) recorded the characteristic symptoms of the disease as interveinal foliar yellowing starting from the tips of leaflets in two to three leaves of the outermost whorl. Tips of the chlorotic leaves eventually dry up. In advanced stage, leaves are reduced in size, become stiff and pointed, closely bunched and abnormally puckered. Ultimately the crown falls off leaving a bare trunk. The root system of the palm is also affected. The lateral roots are not produced as profusely as in healthy palms. Tips and absorbing regions of young roots turn dark and gradually rot. The endosperm of the diseased nuts becomes blackish and soft, which render them unsuitable for consumption. As the disease advances, the girth of the crown gradually tapers. The internodal length of the affected stem is reduced due to reduction in normal growth. The yield of the affected palms is reduced to the extent of 50 per cent over a period of three years.

A formula for quantifying the disease intensity was developed after studying the association of the various symptoms in more than 2000 palms (George *et al.*, 1980).

Due weightage was given to foliar yellowing, necrosis and reduction in the size of the whole crown.

$$I \text{ (intensity)} = \frac{Y \pm N + R \times 10}{L}$$

Where Y and N are the sum of grade points for yellowing and necrosis, L is 50 per cent of the number of leaves on the crown and R is the grade point for reduction in size of the crown for the whole palm.

Etiology: The etiology of YLD of arecanut was a matter of dispute for a long time. Each of the incitants are dealt with below:

Fungi: Khandige *et al.,* (1957) first recorded the association of fungi with YLD. So far 54 different species of fungi are recorded from different parts of the disease-affected palms. A number of fungi such as *Cercospora arecae, Exosporium arecae, Leptosphaeria* sp., *Diplodia* sp., *Phyllosticta* sp., *Dimersporina* sp., and *Trametes corrugata* were isolated from the diseased leaves (Menon, 1959). Roots of diseased palms yielded *Trichoderma* sp., *Pestalotiopsis* sp., *Aspergillus* sp. *Penicillium* sp., *Fusarium* sp., *Colletotrichum* sp., and *Acremonium* sp., which were not pathogenic on inoculation to seedlings (Srinivasan, 2000). Species of *Pythium* and *Phytophthora* were isolated from the roots of disease affected palms using selective media. All the inoculation experiments with frequently occurring fungi yielded only negative results. None of the species tried was able to produce the typical disease symptoms.

Bacteria: Srivastava *et al.,* (1970) reported bacterial streaming associated with YLD affected roots. Out of two bacterial isolates recovered from roots, one was tentatively identified as *Pseudomonas sp.* Later reports (Srinivasan, 2000) revealed that samples from different regions exhibited bacterial streaming in the order of 70 percent from samples of Palode (diseased) and 20 per cent from samples of Mohitnagar (healthy); but no bacterial streaming from samples of Vittal (healthy). Three genera of bacteria viz., *Bacillus, Xanthomonas* and *Serratia* isolated from areca roots were reported to cause symptoms like discoloration with water soaked areas, when inoculated to areca

seedlings and cowpea. Subsequently, pathogenicity of bacteria could not be proved when inoculated on 18 month old seedlings with isolates from areca roots. According to Bopaiah (1979), gram-positive bacteria appeared to be more (70-80%) than gram negative ones (15-30%) in the root region of healthy areca palms. In diseased gardens, the bacterial population has been reported to be more by 31 percent than in healthy gardens (Srinivasan, 2000). In mixed-cropped garden no consistent bacterial population was recorded and organic recycling in such farming systems did neither increase the yield of arecanut nor reduce the yellow leaf symptoms (Rawther *et al.*, 1979).

Virus: Paper chromatographic studies (Menon, 1961) indicated that some proteins or their subunits were present in diseased areca palms which were absent in the healthy ones. Serological investigation with crude arecanut antigen and disease specific rabbit antiserum showed that there was precipitation reaction, indicating antibody formation. Menon (1960) therefore suggested the possibility of virus or virus-like organisms being involved in the disease. Menon (1963) transmitted yellow leaf disease to indicator plants viz., *Jatropha curcas, Canavalia ensjformis* and *Vinca rosea* using partially clarified sap by mechanical inoculation using carborundum as abrasive.

Mites: Khandige *et al.*, (1957) reported association of mites with the YLD. Menon (1960) distinguished the yellowing caused by mite from the foliar yellowing due to yellow leaf disease.

Nematodes: Extensive surveys, conducted during 1974 to 1979 in the arecanut growing areas of Kerala, Karnataka and Tamil Nadu, indicated the occurrence of twenty eight genera of plant parasitic nematodes in the soils of YLD affected arecanut palms. It was seen that the phytoparasitic nematodes such as *Rotylenchulus reniformis, Helicotylenchus dihystera, Radopholus similis, Hemicriconemoides mangiferae, Caloosia longicaudata* and *Hoplolaimus seinhorsti* were recorded in maximum number of soil samples from all the three states (Sundararaju and Koshy, 1982). The burrowing nematode, *Radopholus similis* was obtained from maximum number of

root samples. Except for *R. similis* the occurrence of nematodes belonging to other genera which were in small numbers in the root zone of healthy and diseased palms may not be of any significance. They may probably be feeding on other herbaceous weeds growing in the root zone of arecanut gardens. It is evident that there is wide spread occurrence of *R. similis* in all soil types (Koshy *et al.,* 1978; Sundararaju and Koshy, 1982).

The burrowing nematode *R. similis* produced lesions, rotting and blackening of root tips (Koshy *et al.*, 1976). The possibility of *R. similis* as an aggravator in the disease complex was brought out. Pathogenicity of *R. similis* in combination with *Cylindrocarpon obtusisporum* on arecanut seedling was established in pot culture experiment. Though inoculated plants showed reduced growth, poor root system and root lesions, no typical YLD symptoms were produced (Sundararaju *et al.*, 2000).

Phytoplasma: Nayar and Selsikar (1978) showed the presence of *Phytoplasma* in the young sieve elements of YLD affected arecanut palms from Kerala and Karnataka. In order to confirm the above reports and to establish the association of the organism with the disease Ponnamma and Solomon (2000) examined 35 diseased palms with varying intensities of YLD symptoms and 29 healthy palms from different areas and observed the presence of *Phytoplasma* in tissues of all the diseased palms. However, such bodies were totally absent in healthy palms studied. Neither protozoan flagellate nor any other sub-microscopic agents were observed in the tissue. The organism was found in increasing numbers in the juvenile tissues in the following order; apical meristem, root tip, rachilla, petioles of developing leaves and spear leaf. *Phytoplasma* in fewer numbers and lacking internal contents, possibly degenerated forms, were observed in the yellowed mature leaves. Only a few sieve tubes in a vascular patch contained the organism.

The organism was bound by triple layered membranes, having less dense fibrillar nuclear area and peripherally distributed ribosomes constituting the electron dense portion. The pleomorphic forms often assumed sites close to the walls of the sieve tube and were also found traversing the sieve pores. Forms ranging from spherical,

oval and elongated budded structures were observed. The size of the organism in general ranged between 250-500 nm. Elementary bodies with electron dense contents were infrequently noticed.

As electron microscopy is laborious and time consuming, light microscopic techniques using certain histological dyes have been standardised. Dienes' staining, Thionine-Acridine orange stain and fluorochromes such as DAPI and Hoechst 33258 were found to be useful in detection of *Phytoplasma* infection in YLD affected areca palms, insect vectors and dodder laurel (Ponnamma and Solomon, 2000).

Attempts were made to develop a reliable and rapid sero-diagnostic method similar to one standardised for detecting root (wilt) disease condition (Solomon, 1991). The antiserum produced (on testing in double diffusion test) gave only a faint reaction against samples from diseased palms. A more sensitive and rapid sero-diagnostic technique such as immuno-osmophoresis was also attempted on microscope slides. The tissue extracts from yellow leaf diseased palms on immuno-osmophorosis produced a single precipitin line, midway between the antigen and antiserum wells, while no such precipitin lines was observed against tissue extracts from healthy areca palms.

The constant association of *Phytoplasma* with the disease warranted search for insect vector(s) that transmits the disease. The involvement of insect in transmission of the disease is evident from an experiment in which areca seedlings, protected from aerial vectors, grown in insect proof cages did not contract the disease as agalnst seedlings grown in open field (Ponnamma, 1994). Similarly, seedlings protected by fortnightly spraying of insecticides were free of the disease. In an observational trial, the reduction in rate of disease incidence by both insecticide treatments and the higher incidence in the control untreated border seedlings revealed that an insect vector transmits YLD pathogen. The decreased rate of spread of the disease among seedlings given foliar treatments of insecticides suggested that a leaf-feeding insect spread the disease. A correlation between the population level of *Proutista moesta* and YLD incidence indicated the probability of P. *moesta* as the vector (Ponnamma *et al.*, 1997).

Inventory of putative vectors through various trapping aids and direct examination of over 300 areca seedlings for a period of two years revealed the consistent presence of P. moesta on areca palms. Survey of representative gardens in Karnataka and Kerala clearly established that the disease does not occur independent of the presence of the insect. EM examination of the ultrathin sections of salivary glands revealed the presence of Phytoplasma in the salivary glands of plant hoppers which were offered acquisition and incubation periods of over 30 days on the foliage of YLD affected areca palms. However, such bodies were observed neither in the salivary glands of the laboratory-reared insects nor in insects with acquisition and incubation period of less than 30 days. The organisms were confined to the acini of the salivary glands. The pleomorphic bodies had triple layered unit membrane and contained ribosomes and reticulated DNA strands. Electron dense elementary bodies were also at times found in the acini. This observation indicated the capability of this insect to acquire the organism, sustain its multiplication and possibly act as a vector in transmitting the disease (Ponnamma *et al,* 1991).

Transmission of YLD from diseased to healthy areca seedlings, using the plant hopper, *Proutista moesta* was done at controlled conditions. In the first set, out of six seedlings, which received infective insects, three showed characteristic foliar yellowing symptom of YLD, twenty-one months after the initiation of inoculation and the rest after 32 months. Root tissues from seedlings showed positive reaction to Diene's stain. Electron microscopic examination of ultra thin sections of root apices revealed the presence of Phytoplasmas in the sieve tube of one of the seedlings. After eleven more months, other three seedlings also developed foliar yellowing of YLD. Phytoplasmas were observed in five out of six *P. moesta* inoculated seedlings between 21-32 months after the start of inoculation. All the control seedlings remained healthy (without any foliar symptoms) and the tissues from the roots showed negative results for LM and EM studies.

In the second set of 15 seedlings foliar yellowing appeared for the first time on five seedlings, 22 months after the initiation of inoculation. After 27 months, a total of

eight seedlings showed foliar yellowing. Phytoplasmas were located under EM in samples from five seedlings. All the control seedlings were free of any foliar symptoms and the 8 samples from these were free of Phytoplasms. The above results are the direct evidences on the role of *P. moesta* as a vector of YLD of arecanut (Ponnamma, 1994). Earlier studies have proved that C*arvalhoia arecae* is not a potential vector (Jacob, 1990).

Experimental transmission from diseased to healthy areca seedlings through dodder *(Cassytha filiformis)* was attempted. Dodder was established on twenty naturally infected (3-4 year old) areca palms. New shoots of the dodder were trailed on to 28 healthy areca seedlings raised under insect-proof conditions. Out of these, four exhibited YLD symptoms. Five more seedlings also showed symptoms like marginal yellowing and reduced size of the crown. EM examination of the recipient palms showed phytoplasmas in three out of four areca seedlings with foliar symptoms. In the fourth seedling, sieve tubes showed structural alterations that are often observed in association with *Phytoplasma* infection. The presence of *Phytoplasma* in the source palm connecting dodder strands and in the recipient palms conclusively established the transmission of the disease (Ponnamma and Solomon, 2000).

Nayar (1971) reported the remission of symptoms of YLD by oxytetracycline treatment (OTC). Two field experiments were conducted, one at Palode and another at Peechi, using OTC, Hostacycline, Ledermycin, Neomycin, Penicillin and Gentamycin. The antibiotics were injected into experimental areca palms using the 'pneumatic pressure injection device'. Injections were performed at the bole region of the palms at a pressure of 4 kg / cm^2. The palms treated with OTC, Hostacycline, Ledermycin, Neomycin and Gentamycin showed improvement over the pretreatment disease condition whereas Penicillin and distilled water treated palms deteriorated, thereby establishing the Phytoplasmal etiology of the disease (Ponnamma and Solomon, 2000).

Physiology: The arecanut palms exhibit very clear symptoms of YLD during the 'wet' season (August-October). In a majority of the diseased palms, the symptoms

begin disappearing well before the onset of 'dry' season and remain symptomless during 'dry' season (December-May). A high evaporative demand in the atmosphere existed during 'dry' (December-May) period as indicated by high PAR, temperature and VPD masking the disease symptoms, while during 'wet' season (June-October), the weather parameters showed differences. There was no soil moisture stress during both the periods as the palms were under irrigation, although atmospheric drought occurred during May. Probably the higher temperature recorded in both locations during 'dry' season may have a detrimental effect on the organisms resulting in a temporary remission of symptoms (Chowdappa *et al.,* 1995).

The effect of water-relations on *Phytoplasma* induced diseases has not been well understood, even though it has a profound effect on the physical environment within the sieve tubes. The leaf samples of the affected palms showed that the moisture touches the lowest level in June, the month of symptom emergence, by 59.1 per cent. The corresponding value in healthy leaves was 70.84 per cent. Root samples of the above groups did not vary much (Yadava *et al.,* 1972).

A hole made at the base of the trunk of affected palms allows a viscous dark liquid to come out and such palms recoup temporarily from this malady. The affected palms showed a high leaf sap acidity (pH) of 3.29 as compared to 4.63 of healthy ones. Chowdappa *et al.,* (1995) reported that the diseased palms had higher stomatal resistance (rs) and lower transpiration (E) than apparently healthy palms in the 'wet' season.

There was significant linear relationship between E and rs. The trend was similar irrespective of the palm's condition and season. The water potential values of apparently healthy palms was -1.11 Mpa, whereas diseased palms showed higher values (-0.81). When determinations on rs were made at different times of the day i.e., from 8 to 16 hour, the leaves of apparently healthy palms showed an increase in rs with increase in light, temperature and VPD Even though, the diseased palms followed similar diurnal patterns, the stomata remained closed through most of the day and thus resulted in accumulation of water in the leaves of diseased palms

irrespective of the hour of the day as compared to apparently healthy palms. The differences in water relation components between apparently healthy and diseased palms were not observed during the 'dry' season. Though the diseased palms maintalned the water balance during the 'dry' season through stomatal regulation, the regulatory mechanism seems to be greatly impaired in the 'wet' season resulting in a higher W and rs, thus indicating that the diseased palms could be identified from the healthy palms only in the 'wet' season.

The dIseased palms exhibited lower photosynthesis (A) and higher internal carbon dioxide (Ci) with significant reduction in stomatal conductance (gs) .The ratio of A to gs was significantly lowered in the diseased palms which implied that mesophyll factors were more affected than stomatal factors. The reductions in the A / E ratio, probably coupled with the lack of strong feed back control of gs by gm (mesophyll conductance) lead to the impairment of stomatal regulation (Chowdappa and Balasimha, 1992). Net photosynthetic rate was reduced with increasing light intensities beyond saturating level. CO_2 assimilation was saturated at PAR of 800 mol m^{-2} s^{-1} in healthy palms compared to 500 mol m^{-2} s^{-1} in the diseased palms. This could be attributed to photoinhibition due to reduction in carotenoid pigments, which could have caused light stress at higher irradiances in the diseased palms.

Fluorescence parameters such as initial fluorescence (Fo), variable fluorescence (Fv), maximum fluorescence (FM) and the ratio of variable to maximum fluorescence (Fv/ FM) were used to study the functional activities of chloroplast in YLD affected palms (Chowdappa and Balasimha, 1994). Fo values were not affected in the leaves of diseased palms compared to apparently healthy palms, presumably reflecting normality at the PS II pigment arrangement. The reduced FM values in induced chlorophyll fluorescence of the leaves of the diseased palms, suggesting the inhibition of Q_A reduction, a primary electron acceptor of PSII. The potential photochemical efficiency of PSII and quantum yield of photosynthetic O_2 evolution was reduced in the leaves of diseased palms as evidenced by decrease in FV/FM ratio. The decrease in fluorescence indices in the leaves of diseased palms was concurrent with a reduction

in chlorophyll and carotenoid pigments. These changes result in reduction of carboxylation efficiency (Chowdappa and Balasimha, 1992). It has been well established that loss of thylakoids and chlorophylls of chloroplasts along with development of plastoglobules containing dissolved carotenoids is the major phenomenon for development of typical yellowing symptom in 'yellows' diseases of phytoplasmal etiology. Srinivasan (1982a) recorded association of a deranged chlorophyllase -chlorophyll system with yellow leaf affected areca palms. In the diseased palm, activity of chlorophyllase was enhanced and concomitantly the pigment chlorophyll declined. Changes occurred in major plant pigments due to the disease and yellow leaf appeared in plant yellows. Changes in foliar-assimilatory pigments have been compared with degree of damage to host tissue. Under such circumstances, the chlorophyll destruction had primary relation with the degree of expression of yellow leaf syndromes. Consequently, the pigment changes were apparently related to the diagnostic symptoms of yellow leaf disease (Srinivasan, 1982 b). Thus, the phenomenon of foliar yellowing was most probably due to loss of chlorophylls and carotenoids mediated by impairment of phloem functions.

Biochemical changes: In YLD affected palms, epicuticular wax content was significantly higher than those of apparently healthy and healthy palms resulting in low transpiration rate and the accunciulation of higher water content (Chowdappa *et al*, 2000a). A higher accumulation of carbohydrates in the leaf tissues of the diseased palms points to the affected carbohydrate metabolism (Yadava *et al.*, 1972). Nair (1976) reported phloem necrosis in YLD affected arecanut palms. Due to this, sugar translocation might have been disrupted resulting in the accumulation of sugars and starch in the diseased palms (Chowdappa *et at.*, 1993). Although total sugars, reducing sugars and starch increased in the diseased palms, no qualitative differences were found in soluble sugar fractions. The impeded removal of these assimilates in the diseased palms following phloem interruption could reduce photosynthesis probably due to an orthophosphate (Pi) limitation. The leaves of diseased palms showed higher water potential and turgor potential, but lower osmotic potential compared to similar

leaves of healthy palms. The observed decrease in osmotic potential at full turgor in leaves of diseased palms is accounted for by increase in sugars and aminoacids. There was no significant difference in leaf DNA and RNA contents as well as DNA / RNA ratio between the healthy and diseased palms, indicating that these quantitative characters may not be useful as diagnostic criteria.

No significant differences in protein content and electrophoretic protein-banding pattern were found between the healthy and yellow leaf diseased palms. The leaves of diseased palms had higher contents of cystine and methionine, while threonine, phenylalanine and alanine were less. In the inflorescence of the diseased palm, cystine, glutamic acid and serine contents were higher whereas lysine, asparagine, arginine, methionine and hydroxyproline were lower. Lysine and arginine contents of leaves progressively increased with advancement of disease. The aminoacids, serine and glumatic acids were absent in leaves while they were present in large quantities in inflorescence tissues. Serine, arginine and threonine declined in stem tissues with advancement of disease. Proline, cystine and histidine totally disappeared from roots on infection. The peroxidase activity as well as isozyme patterns did not differ significantly between healthy and diseased pams at any given season (Chowdappa *et a!.*, 2000).

Biochemical studies in leaf tissue during symptom development indicated that phenol metabolism is altered and lead to an accumulation of ortho dihydroxy phenols during 'wet' season. However, leaf phenolics did not differ qualitatively due to disease contraction. Srinivasan (1982c) reported derangement in activity of oxidative enzymes-polyphenol oxidase; peroxidase, catalase and ascorbic acid oxidase in yellow leaf affected areca palms. Although, the mechanism responsible for non-stomatal limitation of the photosynthesis in YLD affected arecanut palms is not well understood, it is surmised that the inhibition of photosynthesis may be due to either hormonal imbalance or toxins. However, accumulation of cytokinin in the inflorescence of the diseased palms has been reported (Chowdappa *et al.*, 2000a). Sterol content was significantly reduced in leaves of the diseased palms. The lower

content of sterol in diseased palms might be possibly due to the higher rate of utilization by phytoplasma associated with disease for their growth and multiplication. This observation assumes significance in the light of the finding that sterols are important constituents in culture media for culturing of *Spiroplasma citri* associated with citrus stubborn disease.

Histopathology: Nayar (1968) reported multinucleate cells in the diseased leaf tissues, disturbed tissue differentiation, blocking of palisade cells with brown pigments and degeneration of chloroplasts. Diseased palms possessed smaller epidermal cells; stomata and midrib parenchyma cells, while xylem cells in the midrib tissues were larger. Non- turgidity of midrib phloem and blocking xylem vessels with tyloses to the extent of 35 per cent was also recorded. Epidermal cells from diseased palms were mostly devoid of cell contents and turgidity with deeply stalned nuclei. Rapid collapse of stomata and occurrence of tyloses in varying degrees are recorded. Degeneration of inflorescence tissues, phloem bundles of the stems, cortex of roots is also observed. Tyloses occurred in 60 per cent of xylem vessels of roots.

The important deviations noted in the nuts of diseased palms were; degeneration and malformation of embryosac, degeneration and under development of endosperm, discoloration (blackening) and softening of the endosperm and degeneration and blocking of vascular elements. Nayar (1968) while studying the anatomy of YLD affected arecanut roots in comparison with that of the healthy ones, observed lateral and linear proliferation of phloem tubes in diseased palms. Around 60 per cent of the roots showed the presence of spherical or sub-spherical ingrowths of varying sizes within the xylem vessels similar to tyloses. In older roots, these projections were seen completely blocking the lumen of the xylem vessels. Significant accumulation of starch grains indicated impaired translocation of food materials and accumulation of products of photosynthesis. Positive colour reaction with tetrazolium salts were also recorded (Menon, 1960).

Varietal reaction: As management practices did not yield any positive results, the only other practical solution available for controlling this malady is to evolve resistant!

tolerant varieties. The screening of areca varieties against to YLD was initiated from early 1960s.

Screening of varieties and hybrids: The results of multi location trial initiated during 1970's involving six promising cultivars such as VTL-3 (Mangala), VTL-1 1 (Sumangala), VTL-12, VTL-13, VTL-17 (Sreemangala) and Mohitnagar with South Kanara indicated that all of them were susceptible. Nampoothiri (1982) reported that 52 arecanut accessions derived from both exotic and indigenous sources also succumbed to YLD with varying degrees of intensity. Further, large scale screening of germplasm collection! varietal hybrids, hybrids produced from disease escapes, interse/selfed progenies of different collections involving 88 different cross combinations comprising of 2,328 palms during 1976-1993 were undertaken in YLD affected belt. All of them were found highly susceptible and 18 genotypes showed less than 25 per cent of disease incidence.

The 21 diallel cross combinations planted at CPCRI, Palode in 1976 have contracted the disease within a period of three years .The disease incidence varied from 63.9 to 100 per cent. Maximum incidence was noticed in VTL 3 x VTL 13,VTL 11 x VTL13, VTL1 1 x Thirthahalli, VTL 13 x VTL17 and VTL 17 x Thirthahalli (100%) and minimum in VTL 12 X Thirthahalli (63.9 %). The hybrid combinations between Hirehalli dwarf mutant and promising cultivars (VTL 3, VTL 11 VTL 13, Mohitnagar and Thirthahalli) planted in 1976 exhibited certain degree of tolerance in the initial years. However, all succumbed to YLD within a period of 6-8 years. The disease incidence was highest in Thirthahalli x Dwarf (62.9 %) and least in Dwarf x VTL 11 (18.1%).

The most promising results were obtained from the trial laid out in 1981 at CPCRI Research Centre, Palode with field tolerant palms. Even though all the progenies of Saigon x Mangala did not show resistance to the disease, hybrids between two palms No 300 (Saigon) and No.125 (Mangala) exhibited high level of tolerance. The disease index in this combination was only 2.8 per cent (averaged over nine years) with an

average yield of 9.19 kg / palmlyear. One of the palms belonging to this combination did not show any symptom even after 13 years. Since the yield of this palm was also found to be high (14.3 kg), it can be considered as a very promising breeding material and can be successfully used in future breeding programs for YLD tolerance.

A field trial involving nine varietal hybrids and Mangala and South Kanara as control initiated during 1984 at CPCRI Research Centre, Palode indicated that all of them contracted the disease within a period of three years except VTL 12 x South Kanara combination. Later this combination also succumbed to the disease. Another experiment laid out at Kannara in 1986 involving hybrids from disease escapes and Mangala and South Kanara local as check showed that more than 50 per cent of palms in all combinations were susceptible to the disease. The hybrids 96-M x 260 and 172 x 71 M gave satisfactory yield of 5.42 and 6.15-kg nut weight respectively with 217 and 268 mean number of nuts. All other combinations were found to be poor yielding with a mean weight of 2.38 kg and less than 80 nuts / palm.

Among the exotic types and species planted in 1968 at Kannara, only two genotypes (Indonesian II and British Solomon Islands I) have remained disease free. In the experiment in which "true" Mangala and segregants were planted to study the intensity of YLD, 24 per cent segregants contracted the disease in the fourth year after planting compared to four per cent in true Mangala. Even after 18 years of field experimentation, none of the hybrids or varieties exhibited immunity to YLD.

From an intensive survey in hot spot areas conducted during 1985-1987 in 13 districts of Kerala involving 1,32,750 palms. 70 healthy/ disease escapes were identified. They were ftirther subjected to light microscopic examination and six of them were found to be disease free. A recent survey conducted in 1998 led to identification of five disease free elite palms in Thrissur and 10 palms in Ernakulam districts (ChandraMohanan and Nampoothiri, 2000). Further, an arecanut garden consisting of 52 palms, raised by the fanner using the seed nuts collected from a single YLD free high yielding palm of about 30 years old, occurring in middle of all other YLD

affected palms, was also identified in the hot spots of Ernakulam district during the survey. These 52 palms are now more than 20 years old. Though they were raised from open pollinated seed nuts of YLD symptom free elite palm, none of these palms were showing symptoms of YLD except three palms exhibiting kernel discolouration. Thus, there were 49 palms (second generation) without any symptoms of YLD. None of the palms exhibited any YLD symptom so far. All the 67 palms (52 palms of second generation and 15 disease escapes from Ernakulam and Thrissur districts) were subjected to histological staining using Diene's stain. Based on reaction to Diene' s stain, the disease escapes identified in hot spots were categorised into healthy and infected palms. The 10 YLD symptom free elite palms in Ernakulam district were negative in their reaction to Diene's stain. Out of the 5 palms identified in Thrissur district, only one palm was found to be negative to staining reaction. Among the 52 second generation YLD free palms in Ernakulam district, 33 palms exhibited negative reaction to Diene's stain. Of these 33 palms, 24 palms were identified and marked as elite palms based on yield evaluation. Thus, 34 palms in Ernakulam district and one palm in Thrissur district were selected as YLD-free elite palms in Kerala state for production of seed nuts by selfing and interse mating. These palms would be monitored every year for disease expression and disease free palms will be confirmed by light microscopic technique.

Seedlings raised from open pollinated seed nuts of second generation YLD free elite palms identified in Ernakulam district are being evaluated for their reaction to YLD by interplanting them in arecanut gardens with more than 90 per cent of YLD affected palms. Selfing of inflorescence of these 24 second generation elite palms is also in progress.

Soil and Nutrition Management: Among different factors associated with yellow leaf disease of arecanut, soil health and balanced nutrition are profoundly important as they are assumed to influence the disease incidence either directly or indirectly. Menon (1960, 1961) suggested that lack of balanced nutrition and improper cultivation practices made the palms susceptible to the disease. Holmes (1964) observed that

yellow leaf disease was due to deficiency or toxicity of either manganese or iron. Lal *et al.*, (1964) suggested a nutritional approach to the problem in addition to provision of proper drainage.

Trials conducted at Central Plantation Crops Research Institute, Research Centre, and Palode to find out the role of major nutrients on the incidence of yellow leaf disease revealed that yellowing symptoms were not reproduced in nutrient cultures devoid of nutrient element. Another set of pot culture studies to investigate the role of major nutrient deficiencies in the development of the disease did not produce any symptoms typical of this malady (Yadava *et al.*, 1972).

The first report on nutritional aspects of this malady showed the existence of nitrogen and phosphorus deficiency in the soils of disease affected gardens. Studies in YLD affected belt of Kerala (1959-62) indicated that soils were highly acidic with pH as low as 3.8 and were deficient in all three major nutrients. Velappan (1969) observed that the soils of yellow leaf disease affected gardens were found low in pH, organic carbon, available phosphorus and magnesium. Leaves of diseased palms contained lower amount of nitrogen, phosphorus, magnesium and zinc. When seedlings were grown in soils supplied with manganese, calcium, boron and zinc, the toxicity symptoms developed did not resemble those of YLD. Subsequently, leaf analysis of diseased palms at Vittal since 1969 revealed that leaf samples contained more than 3 ppm of dilute acetic acid extractable aluminium, a level, which is considered as dangerous to plants. Similarly, soil in diseased tracts recorded higher contents of exchangeable aluminum (Mohapatra *et al.*, 1975).

In a comprehensive soil survey in disease affected areas in Kerala and Karnataka, the soils were found to be high in organic carbon and medium to low in available phosphorus and potassium. The exchangeable iron, manganese, zinc and copper contents were above the level of sufficiency in these soils. It was also reported that increase in soil acidity and clay content significantly increased the quantity of exchangeable aluminium in Kerala state. A sand culture experiment to investigate

the role of aluminium on yellow leaf disease in 1974 revealed that addition of aluminium at 5, 10, and 20 ppm reduced the leaf size and growth of palms.

The results of trials on micronutrient toxicity were inconsistent. Deficiency of zinc showed some relationship to the disease symptoms (Velappan, 1969). Sam Raj and Pailey (1965) noticed that symptoms similar to those of the YLD were expressed by the application of boron to soil. However, subsequent studies at Vittal showed that yellowing caused by boron was different from the symptoms of yellow leaf disease. Pot culture experiments conducted from 1962 to 1974 indicated that copper, molybdenum; zinc manganese and iron did not produce the characteristic symptoms of the disease. The contents of silica, phosphoric acid and potash were high in diseased samples while percentage of N and CaO were high in healthy palms. Diseased palm tissue contained higher CaO / MgO ratio and less N, K, Ca and Mn contents than healthy palm tissue. Root tissues of diseased palms were also found to contain more aluminium and less NPK. The role of micronutrients on the incidence of yellow leaf disease was not clear as disease symptoms characteristic of yellow leaf disease could not be reproduced. However, spraying of Mn SO_4 (5 g / 5 lit of water) and Mg SO_4 (5 g / 5 lit, of water) had improved the condition of the affected palms with fresh growth of leaves. The application of fertilizer also resulted in more number of non-chlorotic leaves in the disease-affected garden (Menon and Kalayanikutty, 1961).

Pal *et al.*, (1960) reported that water logging is considered as one of the predisposing factors in the spread of yellow leaf disease. It was reported that water table was within the root zone of palms in disease affected garden which finally leads to reduced condition during rainy season (Mohapatra, 1975; Mohapatra *et al.*, 1976). Studies on the effect of submergence on soil pH and exchangeable aluminum indicated that there was increase in pH from 5.01 to 6.08 during first 15 days of submergence and continuous submergence up to 90 days led to considerable decrease of pH (4.27). The exchangeable aluminium was inversely related to changes in soil pH (Mohapatra *et al.*, 1976).

Management: Since the yellow leaf disease is not amenable to control by

conventional plant protection measures, it became imperative to find out other means of containing the disease so as to obtain maximum economic returns from the affected gardens. Soil and nutrient management practices such as drainage, cropping systems, pesticide and fertilizer use are assumed to have greater influence on the disease incidence. A mixed cropping trial involving regular organic recycling in a disease affected garden indicated that there was an increase in yield with cowpea, NB 21 fodder grass and guinea grass as mixed crops in arecanut garden. Application of higher dose of phosphorus (160g / year / palm), dolomite (4 kg) and farmyard manure (12 kg) with irrigation had no apparent effect on disease incidence and yield of palms. However, in another trial at Palode, lime and phosphate application resulted in lowest disease incidence (12.5%) compared to lime (33%) and superphosphate alone (15.4%). An integrated control trial at Palode in Kerala with the application of agrimycin, bavistin and temik alone and in combination revealed that the palms treated with agrimycin + bavistin + temik were free from YLD. In another management trial, the palms which received higher dose of K and Mg recorded minimum disease intensity (Nair, 1994).

Soil application of NPK and lime with or without zinc sulphate resulted in significant reduction in the yellowing of the leaves in Karnataka (Dastagir, 1963). There was also better response to soil application of NPK together with lime, boron and manganese. Addition of dolomite 4 and 8 kg / palm / year had no significant effect on the disease condition of the palm. Mohapatra *et al.,* (1976) also reported that application of lime as dolomite and phosphorus had no ameliorative effect on the disease.

In Kerala and Karnataka the field trials conducted during 1965-69 to find out the effect of macro- and micro- nutrients on disease incidence revealed that there was a general increase in yield ranging from 0.2 to 55.34 per cent in Karnataka. However, the trend was different in Kerala. There was marginal yield increase (0.74 -4.59 %) in eight of the thirteen treatments and there was some increase in yield (2.41 – 10.04%) in the remaining treatments. In contrast, a field experiment at Palode indicated that

application of macro and micro nutrients with or without irrigation was not effective in controlling the disease (Rawther and Abraham, 1972).

A field study conducted at Sullia, Karnataka in 1974 indicated that addition of NPK fertilizers alone and in commbination with dolomite did not help in ameliorating the disease. In a comprehensive package trial in farmers field at Sampaje in Karnataka, application of NPK + lime + boron and NPK + lime + zinc increased the yield by 20 per cent and addition of NPK + dolomite + neem cake reduced the disease intensity.

By taking into all the results from the earlier trials, a detailed experiment was initiated in 1982 at Palode (Kerala) with four management practices on two varieties of arecanut to evaluate their effect on the incidence of YLD. Incidence of disease was least with higher dose of phosphorus application over and above the normal package. Mangala and its segregants were suprerior to South Kanara local. The effect of phosphorus in reducing the incidence of YLD was evident.

The foregoing account indicates that the role of soil and nutrient factors in the causation of YLD is not yet established. However, from the above trials, it is apparent that soil and nutrient management had improved the condition of disease affected palms and increased the yield to some extent or maintained the yield level. Thus, it is essential to follow management recommendations in order to reduce the disease incidence and to realize maximum economic returns from the YLD affected gardens.

Recommendation and future strategies: Though, the role of soil and nutrient factors in the causation of YLD is not established, it is essentialto adopt all the recommended practices, as detailed below, for reducing the disease intensity and sustaining the yield level of affected palms.

- Annual application of 200 g of urea, 200 g Mussoorie phos and 230 g muriate of potash per palm per year in two splits and additional application of 800 g Mussoorie phos in the affected garden.
- Application of 12 kg each of compost and green leaves per palm per year in addition to chemical fertilizers.

- Irrigation during summer months
- Avoiding water stagnation in the garden during monsoon by providing drainage facilities.
- Growing cover crops in the garden.
- When only a few palms are affected in a garden, remove them to prevent further spread of the disease.
- Adopting need based plant protection measures against other pests and diseases.

Eradication of severely affected palms and development of cost- effective and feasible management practices are most important in controlling the disease effectively. Satellite survey of the disease affected arecanut tract using geographic information system (GIS) would be helpful in identifying the extent of spread and intensity of the disease as well as in understanding the incidence in surrounding areas. This could then be coupled with the existing information on the disease to provide more useful information. The intricacies of environmental and edaphic factors on the spread and incidence of the disease are relatively important and need to be investigated as they influence the disease incidence either directly or indirectly.

In the absence of suitable and effective control measures, the main emphasis must be on identifying the tolerant / resistant elite palms in the 'hot spot areas'. Identification of such palms and screening their progenies should be a continuous process in the search for tolerant YLD lines. As breeding for resistance in perennial palms like arecanut is a slow process, early disease detection aids using ELISA would accelerate the identification of resistance. New vistas in biotechnology like PCR based molecular markers can be exploited for determination of genetic diversity in arecanut germplasm and tagging resistant genes. Evolving field tolerant lines coupled with suitable management practices will be the final solution in tackling the YLD of arecanut.

3.2. FRUIT ROT

The fruit rot, commonly known as 'koleroga' or 'Mahali' in local language, is a

major disease. The fruit rot of arecanut was reviewed earlier (Koti Reddy and Anandaraj, 1982; Rawther *et. al.,* 1982). *Phytophthora* disease was first recorded by Butler (1906) in erstwhile Mysore State and later intensively investigated by Coleman (1910).

Crop loss: Annual crop Loss due to fruit rot ranged from 10-90 per cent (Nambiar, 1956: Reddy and Anandaraj 1982) Losses of about 75-350 kg nuts / acre, and death of about 20-50 palms / acre due to bud rot were reported from Uttara Kannada. About 10-15 per cent bud rot was recorded from Dakshina Kannada (Sarma *et al.,* 2000).

Symptoms: Rotting and shedding of the nuts are the major symptoms of fruit rot disease (Sarma *et al.,* 2000). Water soaked lesions appear on the nuts, invariably from the stalk end. The rotting extends from the calyx downwards. The affected nuts show greyish to whitish growth, which supports abundant sporulation. Heavy shedding of nuts at the base of the palm is a clear indication of fruit rot. When infection occurs late in the season, the affected nuts dry and are not shed. Nuts at all stages are prone to infection. Bud rot, unlike fruit rot, is sporadic in its occurrence and generally seen during South-West as well as North-East monsoons. The affected palms show yellowing of the spindle leaf. As the disease progresses the spindle loses its natural lustre, turns yellow, and slumps to a side emitting foul smell and infections spread to the bud. The bud rot occurs in two ways, through the spindle leaf or through infected bunches (Anandaraj, 1982). When the infection occurs through bunches, the lower whorl of leaves show yellowing.

Identity of the Pathogens: The fungus causing fruit rot of arecanut was first described as *P. arecae* (Coleman) Pethybridge (Butler, 1918). However, the species concept for *P. arecae* has been controversial since it was merged with *P. palmivora* (Butl.) Butl. and *P. meadii* McRae due to lack of sufficient diagnostic characters (Tucker, 1931). Later, *P. arecae* and *P. meadii* were separated from *P. palmivora* based on sporangial morphology and lack of chlamydospore production (Waterhouse, 1963;

Newhook *et a!.* 1978; Stamps *et al.,* 1990). Based on isozyme and mt DNA- RFLP patterns, Oudemans and Coffey (1991) and Forster *et a!.* (1990) considered that *P. pa!mivora* and *P. arecae* are synonymous. The delimitation of *P. arecae* and *P. palmivora* remains an area of debate with conflicting conclusions cited in the literature but it has been reported that *P. palmivora* isolated from cocoa and coconut does not infect arecanut. *P. Meadii,* has been reported as the causal agent of fruit rot of arecanut in Uttara Kannada and Shimoga districts of Karnataka (Saraswathy, 1993). and this fungus has also been reported as the etiological agent of abnormal leaf fall of rubber and fruit rot of cardamom (Sarma *et al.*,2000). Based on detailed morphological and ITS- RFLP and AFLP analyses of 63 isolates of *Phytophthora* derived from different localities of arecanut growing areas, Chowdappa *et a?.,* (2001) clearly established that *P. meadii* is the main pathogen associated with fruit rot of arecanut in India and there is no evidence of occurrence of *P. arecae. P. meadii* isolates exhibited typical stellate patterns of growth with low production of aerial mycelium. Sporangia were borne on loose irregular sympodial sporangiophores. Sporangia (30.62-57.39 x 20.94-3 1.42 ~tm in size) were papillate, with mostly rounded base, and sometimes ellipsoid with tapering base. None of the isolates produced chlamydospores. Sporangia were caducous and carried pedicels ranging from [25]-3Skm in length. Restriction digestion of the ITS fragment (900bp) and ALFP analysis revealed no genetic variation amongst the *P. meadii* strains studied. These cultures showed morphological similarities and had identical ITS-RFLP and AFLP patterns to reference cultures of *P. meadii.*

Another species, *Phytophthora heveae* Thompson is also known to cause the fruit rot of arecanut in Dakshina Kannada district of Karnataka in addition to *P. meadii* (Chowdappa *et a!.,* 2002b). The isolate exhibited no distinct colony pattern with moderate aerial mycelium. The mean growth rate of the isolate was 17.6 mm / day. This isolate produced sporangia, oogonia and chlamydospores abundantly on carrot agar. Sporangia were borne on simple sympodium. They were papillate, irregular in shape with round base and non-caducous. The sporangial size was 37.02 ± 5.38 x 25.34 ± 3.94 gm with L/B ratio of 1.48. The chlamydospores were terminal or

intercalary in position with an average diameter of 26.03 ± 2.20 j.tm. Antheridia were amphigynous and of 13.08 x 10.19 jnn in size. Oogonia were globose and form readily in culture. They were broadly funnel shaped at the base and average size was 28 .tm in diameter. Oospores were 25.69 tm in size. This isolate also took up infection on coconut and cocoa but not on black pepper.

Epidemiology: The disease is dependent on monsoon and starts 15-20 days after the onset of South -West monsoon during May-June and extends upto August-September. High humidity, high rainfall and low temperature (20-23 ~ are congenial for the disease development. Alternate sunshine and rainfall favoured the disease development (Coleman 1910, Narasimhan, 1922). Reddy and Anandaraj (1982) reported that severe disease incidence coincided with high rainfall (5086.6 mm) and high relative humidity (80%) during 1978 when the crop loss ranged from *50-90* per cent. Disease spread is mainly though rain splashes and is favoured by heavy winds during the rainy season. Disease also spreads through birds and small insects (Coleman,1910; Narasimhan 1922). Spread of the fungus occurs mainly through rain splashes (Anandaraj, 1985a). Zoospores once released from the sporangia, swim, germinate and enter the nut through stomata. After an incubation period of four days, the mycelium from the affected nut surfaces sporulates abundantly. However a regular pattern of the disease spread could not be observed (Anandaraj and Saraswathy, 1985). Based on the daily average temperature and taking into consideration four days as the incubation period, a linear model for predicting the disease four days in advance was reported (Anandaraj *et a!.*, 1992). However, further refinement of this model for different agroclimatic regions is needed. Disease incidence was reported to be severe in gardens located in valleys and those surrounded by a thick belt of shade trees and this might be due to the build- up of high humidity.The fungus might be surviving in the form of mycelia or chlamydospores in the soil on the dried bunches, and bud rot of affected palms left in the plantation. Saraswathy (1993) observed mycelium, sporangia in the infected dried bunches and oospores in the fruit rot affected nuts.

Disease management: Integrated disease management involving cultural practices, phytosanitation and chemical control is the present strategy adopted, which is fairly effective. The covering the bunches with 'Kotte' or a cover made out of areca leaf sheath or 'Karada' cover made out of grass, was in practice in controlling fruit rot before the formulation of chemical measures. Probably this operation might have helped in reducing the rain splashes and consequent reduction in the disease spread. However, recent studies with modification of the old method by covering the bunches with polythene covers (125 gauge, 75 x 60 cm) before monsoon was found to be highly effective in checking the disease spread (Chowdappa *a a!.*, 1999). The cost of polythene covering of arecanut bunches varied from Rs 5.29 to 8.69. Whereas Bordeaux mixture ranged frm Rs 6.34 to 7.46. The efficacy of Bordeaux mixture for the control of fruit rot was reported earlier (Coleman, 1910). The use of adhesive like washing soda, resin along with Bordeaux mixture has been effective (Coleman, 1910; Narasimhan, 1924). Bordeaux mixture (1%) alone was reported to have the tenacity and was effective without any sticker (Thomas and Marudarajan, 1938). The spray deposit of Bordeaux mixture retained 45 days after spray with or without adhesives was on par, thus indicating that Bordeaux mixture without adhesives was equally effective (Anandaraj, 1985b). Copper oxychloride (0.5%) was found to be toxic and the oil based copper like Fycol 8 and 8E and Oleocap were found to be inferior to Bordeaux mixture in disease reduction. At present, the prophylactic pre-monsoon and second round sprays after 45 days are recommended and a third round would be necessary if the monsoon is prolonged. Among the two systemic fungicides tested, metalaxyl and Fosetyl Al (Aliette at 0.5%), Fosetyl-Al was found to be superior, but was on par with 1% Bordeaux mixture treatment (Anandaraj and Saraswathy, 1986). Just like coconut, bud rot affected areca palms, if detected early, can be saved by removal of infected tissues and application of Bordeaux paste and drenching the crown of the surrounding palms with 1% Bordeaux mixture was effective. Phytosanitary measures like removal of disease affected palms and bunches should be resorted to realize for better protection with Bordeaux mixture treatment (Nambiar,

1956).

Host resistance: In a recent study, out of 49 cultivars of *Areca catechu* and related species screened with *P. meadii,* none were found to be tolerant. However, *A. normabyii. A. concinna* and *Actinorrthytis calapparia* remained unaffected indicating their high degree of resistance (Saraswathy, 1993). The sources of resistance should be exploited in future programmes to induce resistance in *Areca catechu* to *P. meadii* through hybridisation and biotechnological approaches. Studies are warranted on the distribution of different *Phytophthora* species, in monocropping and mixed cropping systems their correct identity on population basis, and their degree of pathogenic potential.

As the disease is spread by rain splashes covering the bunches with polythene sheet protects the fruits against *Phytophthora* infection. Attempts must be made to evolve biodegradable polythene sheets and integrate the same with effective systemic fungicide to protect the palms during the vulnerable monsoon period.

3.3. BASAL STEM ROT

Basal stem rot, also known as foot rot, root rot, betelnut plague or anabe roga, is one of the dreaded diseases of the arecanut palm. Butler (1906) made the earliest reference to this disease while recording the betelnut plague in Sylhet. The disease was first reported from Karnataka on coconut and arecanut. At present, it is prevalent in Karnataka, Tamil Nadu, Kerala, Assam, Maharashtra and West Bengal. It is found severe in ill drained and vercrowded gardens.

Symptoms: The initial visible symptom is yellowing of outer whorl of leaves. The yellowing dually extends to the inner whorls. The affected leaves later droop down. The bearing capacity of the palm is gradually affected. The affected palms exhibit a dull brownish patch at the base of the trunk from which a brownish gummy exudate comes out. The fruiting body of the fungus develops after the death of the palm (Sampath Kumar and Nambiar, 1990).

Crop loss: Naidu *et al.* (1966) recorded 1-2 per cent mortality of palms due to

infection. Later, Sampathkumar and Nambiar (1990) reported that percentage of palms lost due to disease ranged from 15.9 to 20.1.

Identity of pathogen: Venkatarayan (1936) reported the causal organism as *Ganoderrna lucidwn* (Leys) Karst. But later on, the disease could not be reproduced by inoculation of the fungus. Sampath Kumar and Nambiar (1990) isolated the fungus from affected stem pieces and sporophores using Wakeman's agar medium and attempted to prove the pathogenicity by using various methods of inoculation (stump inoculation, planting diseased stump, stem inoculation, stem insertion, root inoculation and soil inoculation). The percentages of positive isolation of *Ganoderma* from freshly infected stem pieces freshly formed sporophores and roots immediately below the zone of sporophore formation was 50, 20 and *75* respectively. Pathogenicity tests were positive with planting diseased stump, stem inoculation and stem insertion methods. Disease symptoms appeared within 6- 8 months from the time of inoculation in healthy arecanut palms. The fungus was re-isolated from inoculated palms, thereby proving pathogenicity of fungus on arecanut. They showed that failure of the reproduction of the symptoms with *Ganoderma by* the subsequent workers was because they used root inoculation technique. Indeed, Sampath Kumar and Nambiar (1990) reported that only one palm was infected out of *55* palms inoculated with root inoculation method. In addition to G. *lucidium, C. boninense* and C. *applanatum* were also known to infect arecanut and coconut in Karnataka, and both were cross-inoculable (Sampath Kumar and Nambiar, 1993). Using serological reactions, they reported that G*anoderma* species affecting arecanut and coconut palms in the maidan parts of Kamataka are inter-related pathotypes.

Physiology of *Ganoderma lucidum:* The factors influencing the growth of the fungus, *Ganoderma lucidum* were analysed by Nambiar and Nair (1973). The fungus grows well on Waksmans' agar medium. Both thick and thin hyphae resembling stag horn and clamp connections are very common. The hyphae remain hyaline for a long time and later turn to light brown colour on storing. The fungus forms both terminal and intercalary chlamydospores. The fungus was able to grow in a wide

range of pH from 2.0 to 9.0; the maximum growth was at pH 5.5. Among the various organic sources studied, peptone supported the best growth. The fungus utilises the organic nitrogen only. Excellent growth was obtained when maltose was the carbon source. Sporophore or bracket formation was noticed when the culture was grown on sand —maize medium and incubated for more than 10-12 weeks (Nambiar and Nair, 1973; Sampath Kumar and Nambiar 1990).

Host range: The pathogen has a wide host range attacking a variety of palms and several forest, avenue and fruit frees. Naidu *et al.*, (1966) reported that hosts belonging to 19 families, 26 genera and 48 species were known to be infected by *Ganoderma.* Crop species like coconut, jackfruit, mango, eucalyptus, tamarind, citrus and grape vine also act as alternate hosts for the pathogen.

Immuno-Diagnosis: Visible symptoms of the G*anoderma* affected trees appear 5-6 months after infection and such palms die subsequently; thus, there is need to detect the disease during the incipient phases of infection to combat it more effectively. As the routine isolation of pathogen from the diseased palms is a tedious process, a simple diagnostic test is needed. The fluorescent antibody technique has been developed for detection of *Ganoderma* in its early stages of infection (Koti Reddy and Ananthanarayanan, 1984).

Disease spread: The fungus is soil borne inhabiting the dead plant materials in the soil, and fungus spreads from plant to plant through contact ot through the debris of the affected stumps left over in the garden. The disease also spreads through water. Extensive cultural operations and excess irrigation in infected gardens will help in the fast spread of the disease.

Disease management: Sampath Kumar and Nambiar (1990) developed a set of integrated disease management practices for effective management of basal stem rot. Isolation of diseased palms from neighbouring healthy palms by digging trenches measuring 30 cm wide and 60cm deep helped in reducing the infection from 17.6 to 2.4 per cent. This indicated that making isolation trenches around the infected palms

thus limiting the root -to- root contact could check lateral spread of the disease. Frequent cultural operations like digging and ploughing in infected gardens should be avoided, as this will disseminate the infected material from infected site to healthy areas. Avoid the planting of susceptible crop species as intercrops in the arecanut gardens. As the disease is severe in ill-drained gardens, drainage of the gardens should be improved by digging drainage channels. Vigour of the palm and organic content of the soil can be enhanced by regular application of recommended dose of chemical fertilisers, 15-20 kg each of FYM and green leaf and 2-2.5kg neem cake. In addition to agronomic practices, palms in the early stages of infection has to be root fed with 125 ml of calixin solution at quarterly intervals coupled with drenching the basins with captan or Bavistin at 0.3 per cent concentration was effective in preventing the spread of the disease. According to them, the basins of the apparently healthy palms surrounding the affected palm should be treated with the chemical to prevent further spread of the disease as a prophylactic measure. However, for effective check of disease spread, an integrated approach comprising phytosanitary, cultural and chemical measures is imperative.

3.4. INFLORESCENCE DIE-BACK

Although no systemic survey has so far been conducted to assess the crop loss due to this disease, Saraswathy *et* al., (1977) reported that this disease affected 60 per cent of the palms.

Symptoms: Die-back starts as yellowing of the rachillae of the male flowers. As the disease progresses, yellowing extends further to the main rachis. The tip of the rachis becomes dark brown and the discolouration spreads from tip towards the base, resulting in shedding of the buttons or the female flowers. In advanced stages, infection causes drying of the rachillae from the tip to base resulting in a condition called as dieback. Infected female flowers and rachis harbour the pathogen as concentric rings of light pink coloured conidial mass. The fungus enters the host mainly either through the scars of shed male flowers or through stigmatic end of female flowers. The disease occurs throughout the year but becomes severe during summer months (February-

May)(Saraswathy *et al.*, 1977).

Identity of pathogen: The possible reasons for die back and button shedding were thought to be nutritional imbalance, physiological factors, lack of proper pollination, failure of fertilisation. High temperatures especially during summer months accelerate yellowing and drying of rachis. Saraswathy *et al* (1977) reported the constant association of *Colletotrichum gloeosporioides* Conidial State of *Glomerella cingulata* (Ston.) Splaud and Shrenk, with more than 70 per cent of the shed buttons and infected inflorescences. Inoculation of rachis and subsequent production of characteristic symptoms of the disease established the pathogenicity of the fungus.

Management: Fungicides such as benomyl (0.1 %), captan (0.25%). thiram 0.25%) and phenyl mercuryurea (0.1%) inhibited the mycelial growth of *Colletotrichum gloeosporioides in vitro.* Removal and destruction of severely affected infloroscences and spraying of Dithane Z-78 or aureofungin- sol + copper sulphate *(50* ppm each) were effective in reducing the disease incidence (Saraswathy *et al.*, 1975). According to them, the first spray should be scheduled at the time of opening of the female flowers and second after a gap of 20-25 days.

3.5. BACTERIAL LEAF STRIPE

The occurrence of bacterial leaf stripe disease was reported from Tumkur district of Karnataka ml 970 (Rao and Mohan, 1970).

Symptoms: Appearance of one to four mm wide dark green, water soaked, translucent, linear lesions alongside and parallel to the mid-rib of the leaflet to its main veins is the initial characteristic symptom of the disease. The lesions may start at any point on the lamina surface. Lesions on the lower surface of the leaflets are covered with creamy white bacterial exudate, which is the distinctive feature of the disease. This bacterial exuadate forms creamy white to yellow flakes on drying. In advanced stages, the lesions may enlarge upto one cm or more wide, often covering the entire leaf lamina. All the leaf- lets in the frond may be affected resulting in complete or partial blighting. In severe cases, the entire crown may be affected.

When infection reaches growing buds it leads to bud rot, resulting in the death of palm. Palms of younger age group (3-5 years) were highly susceptible to the disease than older palms.

Identity of pathogen : Although Rao and Mohan (1970), from IARI, New Delhi, identified the causal agent of the disease as *Xanthomonas campestris* pv *arecae,* further work on this bacterium was carried out at CPCRJ Research Centre, Hirehalli. The pathogen not only produced typical symptoms on arecanut but also produced typical dark green, water- soaked, elongated lesions on coconut and on various ornamental palms (Sampath Kumar, 1991). The toxin responsible for production of characteristic symptoms on arecanut was purified and identified as heteropolymer of glucose, galactose, mannose and small amounts of glucuronic acid. The bacterium produces large amounts of extracellular polysaccharide toxin in highly susceptible arecanut cultivars. Sampath Kumar (1991) reported the presence of an extra phenolic compound in diseased leaf tissue as a result of host- pathogen interaction.

Epidemiology: The disease starts during July- October when the average monthly rainfall is 130 mm or above with more than 10 rainy days per month. Thereafter, the incidence declines. The disease spread slackens when the temperature reaches above 30 ^{0}C or below 17 ^{0}C. The bacterium could not survive in soil for long time, suggesting that soil is not acting as source of inoculum. Close planting, frequent irrigation (every 5-10 days) and application of higher levels of nitrogen and green manure, aggravate the disease development and spread (Sampath Kumar, 1991).

Management: Spraying of tetracycline and its formulations at 500 ppm concentration was found to be highly effective as prophylactic and curative control measures. Stem injections have longer residual effect than foliar application (Sampath Kumar, 1991)

3.6. LEAF SPOT

In recent years, severe incidence of leaf spot disease on arecanut was recorded in several gardens in the coastal regions of Karnataka and Kerala.

Symptoms: The disease was severe in South- West monsoon. Ramanujam *et al.*, (1993b) studied in detail the symptoms of leaf spot disease on arecanut in different localities of Kamataka and Kerala. The leaf spot symptoms were similar irrespective of the locality. The palms of all age groups were susceptible to leaf spot, but the severity of the infection was more in seedlings. Symptoms of leaf spots appear on the leaves of outer and middle whorls. In a palm, one to six leaves were affected. The maximum disease intensity was noticed on the outermost leaf in the outer whorl and that the intensity gradually decreased in the inner leaves. Brown to dark brown or black spots with a broad or narrow halo appear initially on the leaves. These spots enlarged to the size of 20- 40 mm and coalesced to form large blighted areas in the advanced stages of infection. Some of these spots showed a central dried greyish portion with dark pycnidia of the fungus on the upper surface of the leaf. The affected palms showed drying and drooping of leaves in the advanced stages. In the case of severe infections, the entire crown dried up in seedlings.

Identity of pathogen: *Phyllosticta arecae* Hohnel and *Colletotrichum gloeosporioides* (Penz) Sacc were the most commonly isolated fungi from the leaf spots (Ramanujam *et al.*, 1993b). They produced typical leaf spot symptoms individually and in combination. Infection was severe when these two fungi were inoculated together.

Management: *In vitro* evaluation of fungicides was done against *P. arecae* and *C. gloeosporioides* since both these fungi were found to cause leaf spot disease. Among the eight fungicides tested *in vitro,* carbendazim (Bavistin, 0.05 %), Mancozeb (Dithane M-45, 0.3%) Captafol (Foltaf, 02%) inhibited cent percent of vegetative growth.of both the fungi, while Bordeaux mixture inhibited 85.7 per cent growth of *P. arecae* and 81.6 per cent growth of *C. gloeosporioides.* Field trials, involving these four fungicides, were carried out at two locations (Yellapur and Puttur). It was observed that monthly spray of carbendazim (Bavistin, 0.05 %), Mancozeb (Dithane M-45, 0.3%) and Captafol (Foltaf, 0.2%) during June- August were effective in reducing the intensity of leaf spot disease during South- West Monsoon period.

3.7.STORAGE DISEASES

Arecanuts are prone to infection by a number of fungi especially of the Hyphomycetes during storage. Lack of proper drying, improper spreading and turning of nuts, unexpected rains during drying period are the pre-disposing factors for fungal infection of stored nuts. Infection causes discoloration of nuts and reduces the quality and fetches low market price. They are not good for chewing.

Symptoms: Infected kernel exhibits kernel discoloration and disintegration of the white core and exhibits hollow cavity. Colour of the nut depends upon the organism responsible for infection. The fungus first attacks the embryo, spreads to the central white core of the endosperm and causes disintegration and then attacks the adjacent lamella of the rumination.

Identity of the cause: Nambiar and Nair (1970) reported that the nut surface gets mechanically damaged by dropping of the bunches to the ground during harvest, and these damaged areas serve as entry points to the microorganisms. In such nuts, infection was upto 54.7 per cent and the fungi such as *Aspergillus niger, A. flavus, Botryodiplodia theobromae* and *Rhizopus sp.* were responsible for spoilage. Later, a number fungi of other like, *Aspergillus* sp., *Diplodia* sp., *Mucor* sp., *Penicillium* sp., *Thielaviopsis* sp., *Cladosporium* sp., *Fusarium* sp and *Colletotrichum gloeosporioides* have been isolated from spoiled nuts (Saraswathy *et al.,* 1977). Of the fungi recovered from spoiled nuts, *Botryodiplodia theobromae* followed by *Aspergillus* sp caused maximum damage of 19.3 and 6.4 per cent respectively.

Management: Nambiar *et al.* (1971) developed the following technology for management spoilage of arecanuts. No infection was noticed when the nuts were harvested without soil contact and dried in hot air oven at 65 °C for 63 h, whereas in conventional method of harvest followed by drying in mechanical dryer at 62 °C for 72 h, 3.6 per cent of the nuts contracted the disease. Treatment of nuts with fungicides such as blitox or Bordeaux mixture followed by drying on cement floor significantly reduced the percentage of infection. Infection was less when the nuts were stored in

polythene- lined gunny bags compared to those stored in plain gunny bags.

3.8. MUSHROOM PRODUCTION TECHNOLOGY USING ARECANUT WASTES

Oyster mushroom *(Peurotus sajor- caju* (Fr.) Singer is mainly cultivated on steam pasteurized wheat or paddy straw. A number of other agricultural waste materials have been used for the cultivation of several mushroom species. Studies conducted at CPCRI Regional Station, Vittal indicated that leaf sheaths of freshly fallen areca leaves have been found to be a promising substrate for the cultivation of oyster mushroom (ChandraMohanan and Moorthy, 1991; ChandraMohanan, 1993). A simple chemical sterilization method, involving a solution of formalin ± carbendazim for 18 h has been standardised for pasteurization of areca leaf sheath and was found to be better than steam sterilization. The average yield of fresh mushrooms was the highest (418 g / bag containing 3.5 kg wet weight of leaf sheath) after treatment with formalins 500 ppm + carbendazim 25 ppm, with biological efficiency of 49.8 per cent. But the average yield was only 260 g per bag in steam pasteurization method. In one ha of arecanut garden, 5 to 6 tonnes of organic wastes (fallen areca leaves, areca bunches and arecanut husk) are available annually. The substrates after production of mushrooms can be reused for vermicompost production (Chowdappa *et al*, 1999). By utilizing these wastes effectively through oyster mushroom *(Pleurotus sajor- caju)* and vermicompost production gives an additional net profit of Rs.28,173 / ha. Mushrooms are a good source of edible protein. Vermicompost has been found to contain appreciable amount of Nitrogen, Phosphorus, Potassium, micronutrients (Cu, Fe, Zn and Mn) and beneficial micro-organisms. Recycling of organic wastes in an arecanut garden in the form of vermicompost could meet 50% N, 32.5% P and 26% of K requirement of one hectare garden besides supplying considerable organic matter and microcnutrients. These technologies generate employment opportunities for rural youth and women throughout the year.

4. COCOA DISEASES

Cocoa *(Theobroma cacao* L), a native of South America, has been found to be an ideal and profitable mixed crop in the arecanut (*Areca catechu* L.) and coconut *(Cocos nucifera* L.) gardens. At present, cocoa is cultivated in 12,400 ha with an annual production of 6,900 tonnes. The processing industrial units project a demand of 30,000 tonnes by 2005, a four fold increase, based on increased infrastructural facilities to absorb increased production and annual market growth rate. Although programmes have been launched to expand cocoa in non-traditional areas under irrigated coconuts to meet the growing demands, the immediate concern is to increase the productivity in existing plantations as new plantations take considerable amount of time to come to bearing. Pests and diseases are the major production constraints in India causing 30- 40 per cent crop losses every year.

Extensive surveys conducted by ChandraMohanan and Kaveriappa (1981) in the major cocoa growing regions of India indicated the occurrence of 14 diseases on cocoa (Table 2.). Of these, diseases caused by *Phytophthora* species (Ramakrishnan and Thankappan, 1965), *Colletotrichum gloeosporioides* Penz (Sarma and Nambiar, 1976; Koti Reddy and ChandraMohanan, 1976) and *Botryodiplodia theobromae* Pat (Nambiar and Nair, 1972) were considered as major diseases of cocoa based the on extent of damage they cause. The *Colletotrichum* disease and *Phytophthora* pod rot (black pod) were found to occur in all the localities. The Vascular Streak Dieback (VSD) was noticed only in Kottayam and Trichur districts of Kerala. Pink disease, white thread blight and *Cephaleuros* leaf spot were also noticed in few localities of Kerala State. The diseases such as swollen shoot, witches broom, *Monilia* pod rot *C'eratocystis* wilt and green point gall reported in other countries, were not observed in this survey.

Table 2.Diseases of cocoa

Name of disease	Causal organism	Parts mostly affected
Nursery diseases		
Seedling die-back	*Phytophthora palmivora*	Seedling
Pod rots		
Black pod	*P. palmivora* *P. capsici*	Pod
Trunk and branch diseases		
Vascular streak die-back	*Oncobasidium theobromae*	Branch
Stem canker	*P. ahnivora*	Stem
Thread blight	*Marasmius scandens* *Marasmius equicrinis*	Branch
Leaf blight and shot hole	*Colletotrichum loeos orioides*	Leaf
Cushion galls	*Calonectria rigidiuscula*	Flower cushion
Red rust	*Cephaleuros virescens*	Leaf
Nutritional disorder		
Zinc deficiency	*Zinc*	Leaves

4.1. *PHYTOPHTHORA* DISEASES

4.1.1. BLACK POD

Phytophthora is the most important pathogen of cocoa causing black pod (Chowdappa and ChandraMohanan, 1993a), stem canker (ChandraMohanan, 1978) seedling dieback (ChandraMohanan, 1979), chupon blight (ChandraMohanan, 1983 a) and twig dieback (ChandraMohanan, 1981) in India.

Crop loss: Global losses due to black pod have been estimated as 40 per cent of the world's cocoa production. No studies, so far, have been conducted on the losses of

cocoa due to black pod in India. However, from the district- wise distribution of cocoa diseases and the percentage of gardens showing disease incidence in India, black pod has been found to be the major cocoa disease in this country (ChandraMohanan and Kaveriappa, 1981). Based on the disease incidence in five cocoa plantations in Dakshina Kannada district of Karnataka State, Chandra Mohanan (1985) reported that black pod incidence varied from 12.93 to 20.78 per cent and it was 22.83 to 40.84 per cent on nearly mature pods alone. These preliminary studies on the spread and severity of the disease indicated the importance of the disease in India.

Symptoms: Black pod disease occurs in rainy season when higher humidity with constant optimum temperature prevails. In India, this disease is a serious problem during South - West monsoon period (June September). Pods of all ages are susceptible to the disease. The infection appears as one or more small, chocolate brown to dark brown circular lesions anywhere on the pod surface. Within four to seven days, lesion enlarges assuming an elliptical shape. As the lesion advances, a whitish mycelial growth of the fungus consisting of sporangia is produced over the dark brown pod surface. The lesion increases rapidly and covers the whole pod surface. After about 15 days of infection, the whole pod and beans are invaded by the fungus and the pod turns black in colour. The beans in ripe pod may escape partly or wholly from infection as the beans get separated from the pod husk on ripening (Chowdappa, 1995).

Identity of Pathogen: Although the occurrence of black pod was reported in India in 1965 (Ramakrishnan and Thankappan, 1965), fl.xrther studies on this disease were not carried out for about 12 years, probably, because of the fact that cultivation of crop was confined to a small area until 1970. ChandraMohanan *et al.* (1979) first identified the species responsible for disease as *P. palmivora* in Dakshina Kannada of Karnataka. Later, Sreenivasan and ChandraMohanan (1984) reported *P. palmivora* as the sole species associated with black pod in this district. Extensive studies have been conducted to find out the *Phytophthora* species causing black pod in the major

cocoa growing areas in India and the variability within and between species. A total of 133 samples showing typical symptoms of black pod collected in different localities in Kerala and Karnataka states during 1989 and 1990 yielded 130 isolates of *Phytophthora* species and three isolates *of Pythium vexans* de Bary. Of the 133 samples collected 120 (90.22 %) samples yielded *P. palmivora,* five samples each yielded *P. capsici* (3.75%) and *P. citrophthora* (3.75%). *P. palmivora* was found to be widely distributed as the major species causing black pod in all the districts of Kerala and Karnataka states (Chowdappa, 1995). Black pod samples collected from Devikulam and Udumbanchola areas of Idukki district, Ranni in Pathanamthitta district and Kannara in Thrissur district yielded *P. capsici* (Chowdappa *et al,* 1993). *P.citrophthora* was isolated from the samples collected from Udumbanchola areas of Idukki district, Thiruvalla of Pathanamthitta district and Thrissur in Thrissur district (Chowdappa and ChandraMohanan, 1996b). *P. capsici* isolates derived from coconut + cocoa + pepper + cardamom and coconut + cocoa + pepper mixed cropping systems. Natural incidence of black pod caused by *P. citrophthora* was observed in coconut + arecanut + cocoa + offea, coconut + cocoa, arecanut + cocoa and coconut + cocoa + pepper mixed cropping systems.

Morphological characterisation: *P. palmivora* isolates produced smooth combed colonies with sharply defined leading edge while *P. capsici* isolates displayed petaloid pattern of growth with moderate to dense cotton- wool like aerial mycelium. *P. citrophthsora* isolates had no distinct colony pattern. The growth rate *of P. palmivora, P. capsici* and *P. citrophthora* isolates were 11.45, 19.18 and 17.90 mm day respectively. Sporangia of *P. palmivora* were ovoid to ellipsoidal in shape with a rounded base. They were shed with a short, broad and occluded pedicel of less than 4 jim in length. They were formed in sympodial arrangement. The average size of sporangia was 42.35 x 28.46 μm with an average L / B ratio of 1.47. Sporangia *of P. capsici* isolates were ellipsoidal with tapered base. Some sporangia were obovoid with rounded base. The formation of sporangia was in typical umbellate pattern. The mean size of sporangia was 41.93 x 22.80 μm with an average L / B ratio of 1.84.

Isolates of *P. citrophthora* produced non-caducous sporangia. They were highly irregular and highly variable in shape. There was no definite pattern in the formation of sporangia of *P. citrophthora.* The mean sporangia size was 56.35x 31.48 μm with an average L / B ratio of 1.76. *P. palmivora* isolates produced chlamydospores readily and abundantly both on carrot agar and in submerged mycelial mats whereas *P. capsici* and *P. citrophthora* isolates did not produce chlamydospores either on carrot agar or on submerged mycelial mats.

Molecular characterisation: Restriction digestion patterns of total DNA: When total DNA of isolates of *Phytophthora* was digested with restriction enzymes having hexanucleotide recognition sites such as Sal I and Hind III and subjected to electrophoresis, isolates within a species could be identified qualitatively by visual similarity in banding patterns, quantitatively by calculating similarity coefficients and phenograms generated from similarity coefficients using UPGMA cluster analysis (Chowdappa and ChandraMohanan, 1 996a)

Polymerase chain reaction (PCR) based discrimination: Chowdappa *et al.,* (2000b) have shown the usefulness of ITS (Internal Transcribed Spacer) regions of rDNA for rapid identification of *Phytophthora* species associated with black pod disease of cocoa by digesting ITS amplification products by restriction enzymes and comparing electrophoretic pattern of resulting fragments. ITS-.RFLP revealed consistent polymorphisms that correlated to morphological descriptions. The Amplified fragment length polymophisms (AFLP) finger prints of *P. paimtvora* from cocoa and coconut showed similar patterns and existence of two genetic groups within *P. capsici* and they are genetically distinct from isolates of black pepper, supporting the existence of two groups within *P. capsici* from cocoa based on colony morphology, size and shape of sporangia, response to antibiotics and fungicides, serological reactions and pathogencity (Chowdappa, 1995). Pathogenicity studies indicated that *P. paimivora* isolates from cocoa and coconut are cross inoculable. *P. paimivora* has been known on coconut as causal agent of bud rot in India since 1906. When commercial cultivation of cocoa was started as mixed crop in coconut gardens in

1960's, *P. paimivora* isolates might have been moved from coconut to cocoa and resulted in the appearance of black pod for the first time in 1965 (Ramakrishnan and Thankappan, 1965). This lack of genetic diversity is coupled with similar morphological and pathological features, suggesting the presence of clonal population of *P. palmivora* pathogenic to coconut and cocoa in India.

Electrophoretic protein patterns: Polyacrylamide gel electrophoresis of soluble mycelial proteins provided a reproducible and sensitive fingerprint for *P. palmivora, P. capsici* and *P. citrophthora* (Chowdappa and ChandraMohanan, 1995). The three species exhibited distinct protein banding patterns. Isolates within *P.palmivora* and *P. capsici* exhibited largely homogenous banding patterns whereas *P. citrophthora* isolates showed two recognisable banding patterns.

Serological relationships: The three species of *Phytophthora* showed distinct serological differences (Chowdappa and ChandraMohanan, 1997b). The isolates of *P. capsici,* which produced homogenous protein and DNA patterns, formed two distinct serological groups. Isolates of *P. citrophthora,* which were distinguished into two distinct sub groups on the basis of protein banding patterns, formed a single serological group. *P. palmivora* was serologically different from *P. capsici* and *P. citrophthora* tsolates.

Response to fungicides and antibiotics: Isolates belonging to *P. palmivora, P. capsici* and *P. citrophthora* were found to be highly sensitive to demeclocycline hydrochloride, oxytetracycline hydrochloride, tetracycline hydrochloride and streptomycln whereas they were less sensitive to chloramphenicol (Chowdappa, 1995) *P. palmivora, P. capsici* and *P. citrophthora* exhibited significant differences in response to every single antibiotic at lower concentrations. ED_{50} values also demonstrated diversity in response to every antibiotic. *P. palmivora* was generally more sensitive to all antibiotics than isolates belonging to *P. capsict* and *P. citrophthora.* There were two distinct sub groups among isolates of *P. capsici* and *P. citrophthora* that were distinguished by their reactions to tetracycline hydrochloride and chloramphenicol.

The three species of *Phytophthora* also exhibited characteristic response to metalaxyl, phosphorus acid and dimethomorph at lower concentrations. There were no distinct sub groups within *P.capsici* isolates but there was considerable heterogeneity in their response to these fungicides. Two distinct sub groups were distinguished among isolates of *P. citrophthora* based on their reactions to metalaxyl, phosphorous acid and dimethomorph. Thus, sensitivity to antibiotic and fungicide could be used as additional tool in finding out the variability between and within the species of *Phytophthora.*

Mating types: Both Al and A2 mating types were found in *P.palmivora* and *P.capsici* (Chowdappa and ChandraMohanan, 1997 a). A2 mating type was found to be the predominant type in *P. palmivora* population in all the districts of Kerala and Karnataka. In *P. capsici,* Al and A2 mating types occurred in the ratio of 4: 1 indicating the predominance of Al eventhough only few samples collected from Kerala yielded *P. capsici. P. citrophthora* isolates were sexually sterile. Both Al and A2 mating types of *P. palmivora* and *P. capsici* were found occurring in Thrissur, Idukki and Pathanamthitta districts of Kerala State where *P. citrophthora* was also present. The occurrence of two mating types of different species in same locality raises the possibility of intra -and inter- specific hybridization between them in nature. The size of oogonia and oospores of *P. palmivora* and *P. capsici* did not vary much indicating that this character has little diagnostic value in separating these two species.

Cardinal temperature: The cardinal temperatures for growth of the three species were 11-12, 24-30 and 32- 33^{0}C (Chowdappa, 1995). But the three species exhibited variation in growth at different temperatures. Variation in growth response to temperature need not be considered as an important characteristic in differentiating the three species of *Phytophthora.*

Effect of plant extracts: Raghu (1990) studied the effect of different plant extracts and fungicides against *P. palmtvora in vitro* using food poisoned technique. The plant extracts and fungicides, which were found to be promising under *in vitro*

condition, were also tested on cocoa pods to find out their efficacy in inhibiting *P. palmivora* infection. Of the 25 plant extracts tested, the extracts of *Allium sativum* (garlic), *Cinnamomum zeylanicum, Adenocalymna allicea, and Lawsonia inermis (henna) and Allium cepa* (bnion) exhibited complete inhibition of mycelial growth of *P. palmivora.* Among these extracts, garlic extract was superior and its efficacy was comparable with that of captan. Further, the antifungal activity of the extract of *L. inermis* was unaffected by autoclaving as well as storage for a period of 30 days. The leaf extracts of *Pongamia glabra, Strychnos nux vomica, Hyptis suaveolens, Lantana camara* and *Adhatoda vasica* at lower concentrations promoted the growth. The production of sporangia was the highest in the presence of leaf extracts of *L. camaro, Catharanthus roseus* and *Ocimum sanctum.* Such plants should not be used as green manure in cocoa gardens as these may help in to increase the inoculum build up in the soil. Both aqueous and acetone extracts of *L. inermis* and garlic at 1:10 dilution and *A. allicea* at 1:5 dilution inhibited *P. palmivora* infection on cocoa pods completely. Of the 14 fungicides tested, captaf was found to be superior. Captaf even at 0.1 per cent concentration inhibited the growth of *P. palmivora* completely. Ridomil MZ 72, Blitox, Fytolan and Dithane M 45 at 0.2 and 0.3 per cent concentrations, Foltaf (0.3 %) and Hexathir (0.3%) inhibited the growth completely (Raghu and ChandraMohanan, 1993). Out of the 10 fungicides tested, captaf, Ridomil MZ and Foltaf inhibited *P. palmivora* infection on cocoa pods completely. Koti Reddy and ChandraMohanan (1984) tested 24 fungicides in *vitro* and found that Bordeaux mixture (7500 ppm), blue copper-50, fytolan, thiram (each at l000ppm), Ziram and miltox (2000 ppm), duer (500 ppm), panoram (3000 ppm), panolil (7500 ppm), panoctine (500 ppm), and Aureofungin sol (200 ppm) completely inhibited the growth of the fungus. The foregoing results suggest that bulb extract of garlic may be used for field trials to control black pod disease of cocoa.

Varietal Reaction: ChandraMohanan (1982) screened 19 cocoa accessions (C78, P6 xPl lÀ, W5/1(T63/884), P9 xP7, W6/56 (T63/9 10), P7 x P6, C83, P3 x P 4,T86/ 2, P3x P. C76, T30/ 10 x Na 32, P9 xP7, P12 x P2, T85/ 5 x Na 32 T 17/ 11, ICS 1,

C 42) against *P. palmivora* and found that all the cocoa accessions were susceptible to wound infection. But, the rate of infection varied. Cocoa pods of C78 were found to be comparatively less susceptible and C42 and ICS I highly susceptible. The sporulation at the infection site of lesion was very poor on less susceptible cocoa pods and abundant on highly susceptible pods. AccordIng to the above author, the comparative density of sporulation may be considered as one of the criteria in screening cocoa germplasm against black pod disease caused by *P. palmivora.* Based on mean lesion area on pods of 20 cocoa accessions (Redaxil, Landas 364 Landas 356, Landas 358, Landas 365, Jarangan Amel x Pa7, Jarangan Amel x Na 32, Jarangan Pa7 xNa 37 Pa7 x Na 32, Amel x Na 33, Amel x Na 32, P1 x P7, P6 x P4, T85/5 x Na32, P7 x P6, T7/12. T86/2, W 6/56, C79 and C44), Chowdappa and ChandraMohanan (1997c) showed that *P. palmivora* produced highest lesion area followed by *P. citrophthora* and *P. capsici. P. palmivora* had higher composite fitness index, calculated from lesion area, mycelial index and degree of sporulation than that of either *P. citrophthora* or *P. capsici.* Thus, the relative virulence of *P. palmivora* may provide a possible explanation for predominance of this species in India. Based on cluster analysis of lesion on wounded cocoa pods, the isolates of each of *P. capsici* and *P. citrophthora* were differentiated into two phenotypic clusters. Therefore, in future routine screening programmes of cocoa cultivars in India, all the three species and also representative isolates of subgroups within each species may be included. Considering the relative virulence of *P. capsici* and *P. citrophthora* in other cocoa growing countries, it may be expected that these two species may become potential pathogens of cocoa and cause severe epidemics in future, although, at present, *P. palmivora* is the predominant species causing black pod disease of cocoa in India. The cocoa accessions exhibited marked variations in the degree of resistance to *P. palmivora, P. capsici* and *P. citrophthora.* The cocoa accession C44 was found moderately tolerant to all the three species of *Phytophthora* whereas Landas 364 was highly susceptible. The accessions tolerant to *Phytophthora* infection may form the source of resistance in future breeding programmes. The identification of Landas

364 as a highly susceptible cultivar is also an important observation not only because of the reason that this cultivar should not be recommended for planting in black pod disease prone areas but also because of the fact that Landas 364 can be taken as typical susceptible cultivar for comparison in future screening programmes.

Epidemiology: The epidemic of black pod initiates in the first week of June and continue up to September with onset of favourable conditions of high rainfall, high humidity and optimum temperature. Experiments conducted at CPCRI, Vittal indicated that black pod incidence was 43.72 per cent in June and decreased to minimum (3.83%) in September. Black pod incidence was also recorded in relation to the height of the stem to find out the source of inoculum. During June, the disease appeared simultaneously on trunk pods located close to the ground (below O.8m), on pods occurring at the height between 0.9- 1.6-in arid on canopy pods. The disease progression followed a similar pattern, indicating soil as well as non- obvious source of inoculum. The combined results of 270 trees showed that the major source of infection were from flower cushions and running water on plant surfaces (38.21 %) followed by non-obvious sources of infections (21.19%). Infections by rain splashes from sprouting pods were about 18.51 per cent. Soil and litter contributed 6.80 per cent of infections. Ants and rodents contributed 0.01 and 0.84 per cent respectively. Based on detailed studies it is established that epidemics of black pod normally intiates from propagules surving in the soil, plant debris,flower cushions, bark, trunk cankers, pod stalks, cherelles, previously infected pods and leaf petioles. The "omnivorous" nature of the fungus and the occurrence of important "non-obvious sources" of inoculum further contributed to the complexity of the disease. Ants, which build "tents" of mud and debris around the pod peduncle, were implicated as major vectors of fungal spread within and between trees.

Management: The effective control of black pod disease involves three main strategies: phytosanitation, use of chemicals and development of resistance in host. Since infected pods, flower cushion, stem, root and leaves will form the main source of secondary infection, all the diseased parts should be removed from the garden at

frequent intervals. Field trial conducted during 1984, by Koti Reddy and ChandraMohanan (1984), revealed that copper-based fungicides have significantly reduced the black pod infection. Further extensive fungicidal trials conducted for 5 years in Dakshina Kannada district of Karnataka, indicated that spraying of pods with Bordeaux mixture (1%) either at 15 day or 30day intervals and copper oxychloride at monthly (0.3%) and bimonthly (0.6%) intervals along with removal and destruction of infected pods during South-West monsoon season were found to be highly effective in black pod management. Among these treatments, spraying of Bordeaux mixture (1%) at 15 day intervals along with removal and destruction of infected pods was found to be superior. The results of these trials also indicated that proper pruning of cocoa plants and removal of diseased pods at 15 day intervals during rainy season without any fungicidal spray reduce the incidence to 50 per cent compared to control plots (ChandraMohanan and Chowdappa, Unpublished).

4.1.2. STEM CANKER

In India, cocoa canker was first reported in 1978 from Dakshina Kannada district of Karnataka state (ChandraMohanan, 1978). A detailed survey of cocoa gardens in Kerala, Karnataka and Tamil Nadu states conducted in 1980 revealed the occurrence of canker in 22 per cent of the gardens surveyed (ChandraMohanan and Kaveriappa, 1981). Later, a preliminary survey of the cocoa gardens conducted by Ramesh Rao (1989), in four taluks (Sullia, Puttur, Buntwal and Belthangady) of Dakshina Kannada indicated the occurrence of canker in 15 gardens, The highest incidence was noticed in Sullia taluk. The disease incidence varied from locality as well as from garden to garden. It also varied depending on the cropping system and was the highest in cocoa under forest canopy. The high humidity and low temperature prevalent in these plantations due to the heavy overhead shade and high density of forest plant population were the factors responsible for the higher incidence of the disease. The survey also indicated higher incidence of natural infection on trunks, suggesting either black pod diseased pods or soil acting as source of inoculum. Natural incidence of canker was noticed during 1998 only in 12 out of 42 different accessions maintained at

CPCRI Regional Station, Vittal. Among Nigerian collections, canker incidence was observed in two accessions (NC 39 and NC 41) while in Malaysian collections, the highest incidence was in Jarangan Amel. x Pa7 followed by Landas 358, Landas 364 and Amel x Na 32.

The symptoms of canker on the external bark was reported as greyish brown water soaked lesion with a broad dark brown to black margin from many cocoa growing countries. But, Ramesh Rao and ChandraMohanan (1993) noticed different types of symptoms on the external bark such as brownish rusty discoloration, dark brown irregular lesion and infection without any external discoloration. Very close examination of 2-3 year old cocoa plants with yellowing of the leaves revealed the presence of stem infection without any external symptoms on the bark. The stem appeared sunken at the region of cankers without any liquid oozing out. Examination of the internal bark of the sunken area revealed that cankers affected such plants. Ramesh Rao (1989) studied the mycoflora associated with stem canker of cocoa grown under different cropping systems in Dakshina Kannada district of Karnataka. The frequency of fungi isolated, varied with cropping system. Among the fungi isolated from the diseased tissue, *Botryodiplodia theobromae* was the predominant species in the samples collected from coconut + cocoa, arecanut + cocoa and cocoa under forest systems. *B. theobromae* and *Fusarium solani* were most frequent in samples from arecanut+cocoa mixed gardens while *B. theobromae, Tric/oderma hamatum* and *F decemcellulare* were predominant in coconut ± cocoa cropping system. *B. theobromae and* F. *moniliforme* were commonly isolated from samples from cocoa under forest. The fungal population also varied with the samples collected from different heights of the tree. The samples collected from collar cankers had the maximum fungal population followed by trunk cankers. It was the least in jorquette cankers. *B. theobromae* was found in association with canker at all different levels of the tree. Samples from collar cankers exhibited the highest population of *B.theobromae* and it was the least in jorquette cankers. The population of *F. decemcellulare* was the highest in collar canker whereas that of *C. crassipes* was the

highest in branch canker. Frequency of isolation of fungi from canker lesion varied with the severity of infection as well as with the depth of the tissue. The fungal population increased with the severity of infection. The frequency of isolation of *P.palmivora* was the highest from the samples obtained from middle stage of disease. *B. theobromae* followed by *F. solani, F. moniliformae, P. palmivora* and *F. decemcellulare* were represented in greater percentages than the other fungi isolated. *B. theobromae* was isolated from infected external bark, internal bark and wood from three different stages of severity of the disease.

As it was very difficult to isolate *Phytophthora* from canker tissue by plating it on potato dextrose agar medium, inoculation of healthy green mature cocoa pods with infected stem pieces from canker lesion and re-isolation from these pods was found to be the easiest method to isolate *Phytophthora.* Twenty-five isolates of *Phytophthora* collected from different localities in Dakshina Kannada district of Karnataka revealed that *P. palmivora* is the sole causal agent of stem canker. When *P. palmivora, B. theobromae, F. decemcellulare* and C. *crassipes* were inoculated separately on cocoa stem, only *P.palmivora* caused infection. In mixed inoculation trials with different combinations of these fungi, *P. palmivora* and *F. decemcellulare* in combination caused more infection than *P. palmivora* alone. Infection was noticed only when *P. palmivora* was included as inoculum, individually or in combination with other fungi. *F. decemcellulare* plays a role in the severity of the disease though it is a secondary invader and becomes active in the mature stem only in association with *P. palmivora*

4.2. *COLLETOTRICHUM* DISEASES

Colletotrichum infection was reported for the first time in India in 1976 on leaves (Sarma and Narnbiar, 1976) and cherelles and immature pods (Koti Reddy and ChandraMohanan, 1976). ChandraMohanan and Kaveriappa (1983b) reported that, besides cherelle wilt, *Colletotrichum* causes three kinds of symptoms on leaves (blight, shot hole and irregular spot). Among them, leaf blight symptom was the most common and widely distributed. ChandraMohanan (1983b) and ChandraMohanan and Kaveriappa (1983c) made an extensive survey in major cocoa growing areas of India

and collected 312 isolates of *Colletotrichum* to find out variation within and between species. Of the 312 isolates collected, 299 found to be pathogenic and cause foliar symptoms on seedlings. These isolates were classified into three group's (white, dark and light) based on colour of the lesions on potato dextrose agar. Light types were more pathogenic than other two types. Of the 299 pathogenic isolates, 249 were fall within light type indicating the predominance of this group in the natural population. The white, dark and light types were further classified into groups and subgroups based on marked variation in the rate of growth, sporulation, presence of acervuli and conidial size (ChandraMohanan *et alt* 1987b). Out of the 299 isolates, 28 produced acervuli on 10- day old cultures, which belonged to light type. Seven isolates of light type culture group produced perithecia with ascospores. Among these, three isolates representing leaf blight, shot hole and irregular spot were found to be most virulent and they differed in their cultural, morphological, biochemical and serological properties. The isolates of inflorescence dieback of arecanut and cocoa are cross-inoculable. The *C. gloeosporioides* isolates from other plants occurring in cocoa gardens and *C.cassipes* from clove were also found to be pathogenic on cocoa seedlings.

Sarma and Nambiar (1976) recorded the incidence of shot hole symptoms on cocoa leaves throughout the year and the incidence was maximum during October-January when temperature range of 19-33 ^{0}C and RH of 77-79 per cent prevailed, Whereas, Koti Reddy and ChandraMohanan (1976) reported the occurrence of *C. gloeosporioides* infection on cherelles and immature pods during January-February. ChandraMohanan (1983b) reported that the intensities of leaf blight and shot hole gradually increased from the month of July and reached a peak during September-November, decreased thereafter and reached lowest level during April-June. But, critical period for the disease incidence on pods was February- May. Low temperature and constant high rain fall and RH during June-November favoured the increase in the populations of *C. gloeosporioides* resulting in higher incidence of foliar infection. In this period, pod infection was low due to susceptible stages of pods being very

low. When the susceptible stages of pods were plenty, during December-May, the climatic conditions were not favourable for infection resulting low intensity of disease on pods. ChandraMohanan *et al* (1987a) standardised the congenial conditions required for foliar infections of cocoa seedlings inoculated with *C. gloeosporioides.* According to them, the disease development was maximum on 18-day old seedlings when exposed to high RH for 72 h and five day old cultures grown on oat meal agar was used. ChandraMohanan and Kaveriappa (1984) studied relative efficacy of nine fungicides (Bavistin, Kitazin, Calixin, Dithane M *45,* Dithane-78, Foltaf, Iohexene, Liquid cumin and Chlorothalonil) on the mycelial growth of three virulent isolates of *C. gloeosporioides* causing shot hole, blight, and irregular spot symptoms *in vitro.* Of these fungicides, Bavistin (0.05-0.10 %), Dithane M 45(0.20- 0.30 %) and Liquid cumin (0.3%) completely inhibited the growth. The fungicidal trial on seedlings revealed that Bavistin (0.025-0.1%) and Dithane M 45(0.2- 0.3%) were found to be more effective in controlling the disease.

Based on epidemiological data, ChandraMohanan (1983b) recommended that fungicidal spray to control foliar infection should start just before the onset of south-west monsoon in the month of May or June and thereafter continued at regular intervals till November. Similarly, to control the pod rot, spraying should be done during December- May.

4.3. COCOA WILT

Cocoa wilt, first noticed in September 1998 in Hunsur taluk of Mysore district of Karnataka, has become a major impediment in area expansion in non-traditional areas of Karnataka (Chowdappa and Rohini Iyer, 2000). A detailed garden to garden survey indicated that the disease incidence was 6.69 per cent. The cocoa plants of 2-5 years age were found more susceptible for infection under field conditions. The disease appears in September, after South-West monsoon period and reaches maximum incidence during October-March and declines thereafter.

The visible symptoms of the diseased plants are yellowing or browning of the leaves,

wilting of branches and finally death of whole plants. The pods on the dead trees remain green for several days and slowly shrivel. These symptoms are invariably associated with borings on the branches that are about 1mm in diameter and are inhabited by *Xylosandrus* beetles (Chowdappa *et al.*, 2002c). Small coiled plugs or powdery mass protrude from these shot holes. Some trees, which have no visible borings, also show visible symptoms. Removal of bark in the region of the borings reveals a brown discolouration. When the branch is split longitudinally, brown streaks are evident. Cross section of the wood show discrete brown patches scattered over the wood. A total of 600 isolations from infected plants were made using water agar, nutrient agar, carrot agar, potato carrot agar, host stem agar, host leaf agar, and potato dextrose agar and potato sucrose agar. The inoculum material included leaf, petiole, stem bits with streaks, frass, insect bores and beetles. Fungal species like *Fusarium, Pestalotia, Botryodiplodia, Graphium, Mucor, Aspergillus, Penicillium, Chlamydomyces, Monilia, Streptomyces, Melanconium, Helminthosporium, Colletotrichum, Trichophyton, Cephalosporium* and five unknown isolates were recovered from infected plants. Of these, *Graphium basitruncatum and Verticillium luteoalbum* were the predominant fungal species. *Craphium* is the synnematal stage of Ceratocystis while conidial stage of some other species of *Ceratocystis* is placed in the form genus *Cephalosporium.* The suspected causal organisms obtained from various isolations were used for inoculation. The isolates were inoculated onto seedlings under glass house and onto five-year-old plants in the field. However, few plants inoculated with *Graphium* showed positive indication after two months of inoculation. At present, phytosanitary practices such as removal and burning of infected trees and swabbing of pruning knives to avoid carrying inoculum and spraying of propiconazole and monocrotophos to adjacent plants are recommended till the tailor made control strategies are formulated.

4.4. OTHER DISEASES

4.4.1. Pythium pod rot

Natural incidence of *Pythium* pod rot of cocoa was first reported from three localities

namely Mavelikkara, Alappuzha and Kottayam in Kerala state in l993 (Chowdappa and ChandraMohanan, 1993b) .The symptoms of *Pythium* pod rot closely resembled that of black pod caused by *P. palmivora. Pythium* pod rot was less virulent (lesion size 8.91 to 15.50 cm^2 seven days after inoculation) compared to *Phytophthora* pod rot caused by *P.palmivora.* (155 to183 cm). Isolates of *Pythium vexans* exhibited chrysanthemum pattern of colony growth with diffused leading edge. Aerial mycelitun was sparse. They readily produced abundant nonpapillate, spherical to ovate sporangia and sex organs. Mean diameter of spherical sporangia ranged from 16.61 to 18.12 μm. The mean dimensions of ovate sporangia varied from 21.90- 24 x 16.42- 17.60 μm. Oogonia were globose and smooth walled with paragynous antheridia. Oospores were aplerotic. The average diameter of oogonia varied from 19 to 21 μm. *P. vexans* has not been reported as a pathogen of cocoa from any other cocoa growing countries. At present, *P. vexans* is a minor pathogen of cocoa in India.

4.4.2. Charcoal pod rot

Charcoal pod rot caused by *Botryodiplodia theobromae* Pat and *Macrophoma was* first recorded on Criollo variety in 1972 at Vittal (Nambiar and Nair, 1972). Later, it was also recorded on the pods of Forastero variety. Pods of all ages are susceptible to the disease and occur throughout the year. The disease starts as yellow spot and then become black in colour. Later, the infected pod dries up and shrivels and remains on the branch as mummified fruit. The surface of the fruit is covered with large number of black spores giving the appearance of soot. Hence the name charcoal pod rot. Charcoal pod rot caused by *B. theobromae* resembles pod rot caused by *Macrophoma* in all its symptoms. In pathogenicity tests, both the diseases produce symptoms in 2-3 days only on wounded pods indicating that wound is necessary for infection. In the field, both pathogens infect the pods through wounds made by insects, squirrels or birds. ChandraMohanan and Koti Reddy (1979) evaluated the relative efficacy of 17 fungicides against *B. theobromae in vitro,* and found that, besides Benalate, Ceresan wet, Leytosol and vitavax were also effective against flingal growth at low concentrations. They suggested these chemicals might be tried in the field to control

charcoal pod rot.

4.4.3.White thread blight

ChandraMohanan and Kaveriappa (1 983a) reported thread blight disease of cocoa in Areca+cocoa mixed gardens in Dakishna Kannada district of Karnataka and Kottyam district of Kerala. The extensive death of young branches and suspended leaves in rows were the field symptoms of the disease. The affected branches of the plants contained white mycelial threads of fungus, which spread longitudinally and irregularly along the surface of the stem. The fungus is neither sporulating nor fruiting. The disease is caused by *Marasmius scandes* Masse. The disease spreads in the field by wind through mycelial mats and initiate infection under favourable conditions of high humidity and rainfall. The pathogenicity was established on 8-10 year old of forestero variety.

4.4.4.Cushion galls

Of the 150 gardens surveyed in all the districts of Kerala, six districts of Karnataka (Dakshina Kannada, Kodagu, Bangalore, Tumkur Shimoga, and Chikmagalur) and Kanyakumari district in Tamil Nadu, cushion galls were observed only in one garden in Trichur district of Kerala state (ChandraMohanan *et al.,* 1984). Based on their symptomatology, they were designated as fan and knob gall. Neither fungi nor bacteria could be isolated from the abnormal galls or cushions. Of the 500 isolations made on potato dextrose agar, only in two cases, *Calonectria rigidiuscula* (Berk and Br.) (Conidial state: *Fusarium decemceullare* Brick) was obtained from gall. This fungus did not produce any typical symptoms when inoculated on healthy cushions. Further attempts to transmit these disorders were unsuccessful. The 50 per cent of beans from pods collected from fan gall affected tree were soft and thin and failed to germinate. There were no anatomical differences between healthy and flower gall affected trees. However, hypertrophy as well as hyperplasia was noted in cushions affected with knob gall. Biochemical studies indicated hyperauxinity as the possible cause for gall formation.

4.5. NUTRITIONAL DISORDER

4.5.1. Zinc deficiency

Incidence of zinc deficiency has been first observed in many cocoa gardens in Kerala, Karnataka and Tamil Nadu states (ChandraMohanan *et al.,* 1981) Chlorosis of the leaves is the initial symptom of zinc deficiency. Later, leaves exhibit mottling and crinkling with wavy margin. Younger leaves produced subsequently become sickle shaped. They reported that zinc deficiency can be corrected by foliar spraying with a mixture of zinc sulphate (0.3%) and lime (0.15%).

4.6. CURRENT AND FUTURE DIRECTION OF RESEARCHES

As the plantation crops are grown together under high density mullet species cropping systems or in the vicinity of each other, intro and inter specific variation within and between *Phytophthora* species are of utmost importance. *Phytophthora* species associated with black pod of cocoa were identified based on morphological criteria. electrophoretic protein patterns and restriction digestion patterns of total DNA. The interrelationships among various isolates of *P.meadii* causing diseases of rubber, arecanut, cardamom and cocoa have not been established. The recent reports of *P. capsici* from cocoa (Chowdappa *et of.,* 1993) and also on *Piper chaba, P. betel* and *Bauhinia* (Sarma, unpublished), and their relationship to black pepper isolates needs to be clarified. *Pythium vexans* has been found to be associated with pod rot of cocoa (Chowdappa and ChandraMohanan, 1993b) and the same fungus is associated with capsule rot of cardamom (Nambiar and Sarnia, 1976) and patch canker of rubber in association with *P. palmivora.* As morphological and biochemical criteria have limitations in identit~ing intra and inter specific differences, a thorough examination on the basis of molecular data is called for. In recent years, internal transcribed spacer (ITS) regions of ribosomal gene repeat and Amplified restriction fragment length polymorphism (AFLP) have been found to be useful for detailed analysis of genetic variability within and between species of *Phytophthora.* Although black pod has been considered a major disease of cocoa in India based on disease intensity, the

exact losses were not estimated. Limited range of copper fungicides (Bordeaux mixture and copper oxychloride) have been tested experimentally for control of black pod of cocoa so far. It has been well recognized that the important shortcoming in use of copper fungicides is their inability to control canker on the stem. The use of systemic fungicides (metalaxyl, aluminium ethyl phosphonate and phosphorous acid) which have shown promise in other cocoa growing countries should be explored for control of canker and black pod. Identification of high yielding and disease resistant cocoa accessions should receive top priority. As the average productivity (560 kg / ha) is considered to be low in India, one of the recognised priorities is development of economically as well as ecologically sound spraying technologies for control of major diseases of cocoa. In order to resolve these problems, extensive research is being carried out on *Phytophthora* diseases of cocoa under the ICAR funded Adhoc scheme entitled "National Network on *Phytophthora* diseases of horticultural crops" with following programmes:

1 Collection and characterisation of *Phytophthora* associated plantation crops
2. Estimation of crop losses
3 Epidemiological studies on *Phytophthora* diseases in palm based mixed cropping systems.
4. Nature of survival *of Phytophthora* survival
5. Identification of bio-control agents! plant products as components in disease management.
6. Development of integrated disease management practices for *Phytophthora* diseases of plantation crops.

In recent years, cocoa wilt streak dieback has become a major impediment in seedling cocoa establishment in Mysore district of Karnataka. A project, encompassing identification of etiological agents, development of onsite immuno format assay for screening of cocoa seedlings, nature of disease spread and development of integrated disease management practices, is under operation to evolve suitable control measures for this malady.

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