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Diseases of Arecanut

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1. INTRODUCTION

Areca palm (*Areca catechu* L.) is prone to a number of diseases during its different stages of development. Nearly forty fungal species including pathogenic and non-pathogenic fungi were reported to be associated with areca palm. Other than the fungal pathogens, a bacterium and an algal parasite also have been reported. Besides the fungal bacterial and algal diseases, another dreaded disease, 'Yellow Leaf Disease' suspected to be caused by mycoplasma like organism (MLO) also is reported. Of the fungal pathogens, some are causing economic loss, i.e., they kill the palm totally or affect the parts only and few of them cause seedling diseases. In storage also nuts are subjected to infection by a number of fungi which decreases the quality of the stored nuts. Such spoiled nuts fetches reduced market value. Based on the extent and nature of damage, the fungal diseases can be grouped into major and minor diseases. Among the major diseases, Koleroga or Mahali, Anaberoga or foot rot, inflorescence die-back and botton shedding are most important. Minor or the lesser important diseases are bud-rot, bacterial leaf stripe, sun scorch and stem breaking, band or hidimundige, stem bleeding and nut splitting. Seedling diseases gain importance only when it occurs epiphytotically in localised areas and cause mortality of seedlings.

2. DISEASES ON SEEDLINGS

2.1 Collar Rot

This disease is common in secondary nurseries and also known as root rot. Poor drainage predisposes the plants to infection by fungi like *Fusarium* spp. and *Rhizoctonia* spp. (Rao and Bavappa, 1961). Infection can be through roots or collar region. Infection through collar region by bacteria causes rotting of the growing bud, while root rot causes wilting of the seedling. Mortality can be checked by improving drainage and also by drenching the nursery beds with 1 per cent Bordeaux mixture or cheshunt compound.

2.2 Leaf Blight

This disease is characterised by the appearance of reddish brown discoloured spots on leaf lamina which eventually results in blighting. As a result of the disease

the seedlings get stunted. The fungus *Phomopsis palmicola* (Wint) Sacc. has been reported to cause blighting of seedlings at the time of transplanting (Roy, 1965). *Pestalotia palmarum* has also been reported to be associated with seedling blight. Menon (1959) reported the association of a pycnidial fungus.

Application of nitrogen and potash followed by spraying with Dithane Z-78 will minimise the disease (Menon *et al.*, 1962). Providing proper shade and spraying with copper fungicides were suggested as control measure for the blight caused by the pycnidial fungus (Menon, 1959).

2.3 Yellow Leaf Spot

Two different types of yellow leaf spot are reported. In the first type, symptoms appear on the leaf lamina during summer months as yellow specks of 3-10 mm diameter. Seedlings in the age group of 1-2$^1/_2$ years are susceptible to this disease. The spots coalesce to form bigger lesions with a yellow halo around. In the advanced stages of infection, seedlings show stunted growth and in the acute stages cause death of the seedlings. A fungus *Curvularia* sp. was isolated from the affected tissues (Menon, 1962). Fungi like *Colletotrichum* sp., *Phyllosticta* sp., *Helminthosporium* sp. (Rao and Bavappa, 1961) and *Alternaria tenuis* (Agnihotri, 1963) have also been reported to cause similar symptoms.

During recent years, a new leaf spot disease with moderate to severe incidence was noticed in several gardens in Uttara Kannada, parts of Dakshina Kannada and Kasaragod district of Kerala. As against the yellow leaf spot reported earlier, this new disease is severe during South-West monsoon period and the intensity of the disease is more in palms at the age of less than 10 years old. The symptoms are usually restricted to 3-4 leaves of the lower whorl. The spots are brown to dark brown or black in colour and usually round in shape with varied sizes. Spots are characterized by the presence of a yellow halo around and enlarges to form blighted patches (Fig. 1) and in advanced stages cause drying, dropping and shedding of the leaves. *Phyllosticta* sp. (Rao, 1966) and *Colletotrichum gloeosporioides* were the most commonly isolated fungi from the affected leaf tissues and their pathogenicity has been established.

Yellow leaf spot seen in seedlings can be controlled by spraying Dithane Z-78 or 1 per cent Bordeaux mixture and also by improving drainage and providing shade in the nursery (Menon, 1962; Rao, 1962a).

For the disease seen in young palms, the field fungicidal trial conducted at Sirsi and Yellapur areas of North Kanara, and Kavu and Heleneranki of Dakshina Kannada areas revealed that Dithane M-45 at 0.3 per cent or foltaf at 0.2 per cent concentration were effective in reducing the disease intensity. Removal and destruction by burning of the badly affected and dried leaves will minimise the load of inoculum in the field (personal communication, Ramanujam, B. and Chandramohanan, R., 1989).

2.4 Red Rust

The algal parasite *Cephaleuros* sp. has been reported to cause circular spots on the stem and foliage. The spots are characterised by a sunken centre and yellow halo around. Spots are irregular and destroys the epidermis (Paily and Menon, 1960). This disease can be controlled by providing shade and spraying with Bordeaux mixture (Westcott, 1966).

In the recent past, a new but similar disease caused by the fungus *Cladosporium spongiosum* was reported from Andaman and Nicobar Islands. Four to six years old palms are seen affected by the fungus. Small light brown spots are seen on the stem, 2-3 cm above the base of the palm. Spots are 2-6 cm long and 1-3 cm wide with irregular dark zonation. Later, these portions become dark olivaceous brown with slightly raised margin and sunken centre. The outer margin is light yellow with variable width. The disease causes discolouration of the internal tissues up to 5-25 cm depth (Rao, 1988).

3. DISEASES ON ADULT PALMS

3.1 Koleroga

Koleroga is otherwise known as Mahali or fruit rot and is one of the major diseases of arecanut. This occurs as an epidemic especially in the heavy rainfall areas of Karnataka and Kerala. In Karnataka, the disease is known as Koleroga (Kole = rotting; roga = disease). It is also called as Mahali (heavy devastation) or fruit rot as the infection is on the fruit (arecanuts). The disease was first reported from the erstwhile Mysore State in South India (Butler, 1906) and subsequently from areas of present Uttara and Dakshina Kannada of Karnataka and also from certain parts of Malabar and Cochin (Coleman, 1910) and at present seen in all the areca growing areas receiving heavy regular rainfall.

3.1.1 *Losses*

No systematic survey has so far been conducted to assess the crop loss due to this disease but an annual loss of 10-75 per cent in parts of Karnataka, Kerala and Maharashtra or total destruction of crop in individual gardens have been recorded (Coleman, 1910; Coleman and Rao, 1918; Nambiar, 1956; Kamat, 1956; Anony., 1960). Heavy crop loss varying from 50 to 90 per cent was reported in the late seventies, the period when the disease was so rampant. A sampling procedure to assess the yield loss has been developed (Anandaraj and Balakrishnan, 1987; Reddy and Anandaraj, 1980).

3.1.2 *Symptoms*

The first symptom is the appearance of water soaked lesions on the nut surface usually near the calyx (Fig. 2). Butler (1906) described rotting and shedding of immature nuts as the characteristic symptoms. Coleman (1910) studied the disease in detail and

illustrated the different stages of disease development in his pioneering work on Koleroga. The infected nuts loose their natural colour and become dark green. The lesion spread gradually covering the entire surface of the nut before or after shedding. A felt of white mycelial mass develops from the lesion of the shed nuts and it eventually envelops the entire nut surface (Butler, 1906; Coleman, 1910). The fruit stalks and axis of inflorescence are also affected in the advanced stages of the disease (Sundararaman and Ramakrishnan, 1928; Marudarajan, 1950). Infection also causes discolouration of the kernel by formation of brown radial strands. Infected nuts will be lighter in weight and possess large vacuoles. Sometimes, the infected nuts may not shed (Marudarajan, 1950a) and remain mummified in the bunches. Such type of infection is locally called as 'dry mahali' in central Kerala (Fig. 3). Nuts of all stages of development are susceptible to infection. The disease causes both qualitative and quantitative loss by way of inferior quality chali and heavy shedding of nuts.

3.1.3 *Etiology*

The disease is caused by *Phytophthora arecae*. The fungus was first described as *P. omnivora* De Bary (Sydow and Butler, 1907). Coleman (1910) named it as *P.*

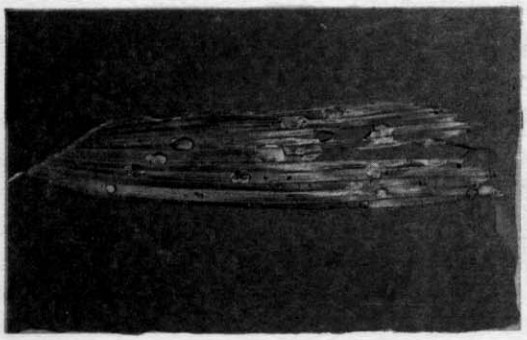

Fig. 1 : Yellow leaf spots on areca seedlings.

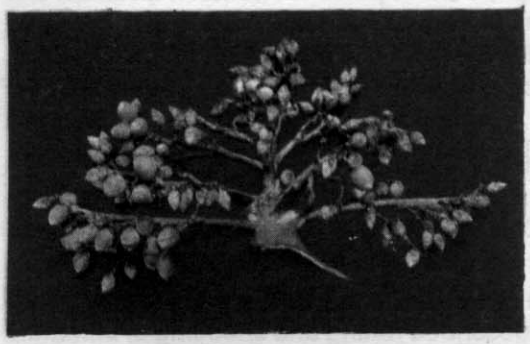

Fig. 3 : Dry Mahali—late infection of *P. arecae*.

Fig. 2 : Fruit rot of arecanut.

omnivora var. *arecae* and later Pethybridge suggested the name *P. arecae* Peth. since the pathogen was quite different from De Bary's *P. omnivora*. Later, Butler (1918) considered it *P. arecae* (Coleman) Pethybridge. Tucker (1931) reported *P. arecae* as synonym of *P. palmivora* (Butler) and later workers considered it a distinct species (Waterhouse, 1963, 1970; Newhook *et al.*, 1978). Sastry and Hegde (1985) studied the sporangial morphology of areca isolates of *Phytophthora* from Sirsi, Siddapur and Yellapur taluks of Uttara Kannada and identified it as *P. meadii* McRae.

The mycelium of the fungus is coenocytic but septate in older cultures. It is inter and intracellular, occasionally branched and the diameter varies 8-9 µ (Coleman, 1910) or 6 µ (Sastry and Hegde, 1987) smooth without hyphal swellings and copiously branched. It grows and sporulates better on steamed cornmeal agar (Tucker, 1931).

The fungus reproduces asexually by production of sporangia and chlamydospores. Sporangia are borne on sporangiophores about 2.5 µ wide occasionally sympodial or mostly sympodial in water (Newhook *et al.*, 1978). Sporangia are spherical, ellipsoid or obturbinate, papillate, occasionally aerial laterally and intercalleraly attached (Newhook *et al.*, 1978). Sporangial diameter ranges from 40-50 × 35-40 µ, the maximum being 70 × 4 µ with an L:B ratio of 1.1-1.4:1. They are caducous with a small pedicel of 1-6 µ (Rawther *et al.*, 1982). Obpyriform with rounded base and hemispherical papilla measuring 25-70 (48) × 15-40 (25) µm with a slender stalk of 11-16 µm (Sastry and Hegde, 1987). Chalmydospores are not produced by all isolates or rare and if produced measures 35-40 µ in diameter (maximum 60 µ) with a wall thickness of 1 µ (Tucker, 1931; Waterhouse, 1963; Newhook *et al.*, 1978) or totally absent (Sastry and Hegde, 1987).

The pathogen is mostly heterothallic. Coleman (1910) could get oospores on inoculated arecanut, *Cereus formosus* and *Clakia elegans*. Desai (1950) reported the production of oospores on fresh bean agar. Homothallic nature of the pathogen was observed by Narasimhan (cf. Anony., 1932), Ramakrishnan (cf. Anony., 1954) and Ramakrishnan and Sethalakshmi (1956). The pathogen was reported as heterothallic (Ashby, 1929; Venkatarayan, 1932; Marudarajan, 1941) and produced oospores in mixed cultures of *P. arecae* × *P. meadii* (Ashby, 1929) and *P. arecae* × *P. infestans* (Gallegly, 1964). Sastry and Hegde (1987) reported oospore production by *P. meadii* McRae when paired with compatible mating type A_2.

Oogonia measures 30-35 µm in diameter (maximum being 40 µm) or 26-45 µm (Rawther *et al.*, 1982; Sastry and Hegde, 1987). Oospores nearly fills the oogonium and the size varies between 25-30 and 30-35 (Newhook *et al.*, 1978) or 15-35 µm (Sastry and Hegde, 1987). Wall thickness is related to diameter and measures 3 µm or 2-4 µm.

Antheridia are always amphigynous, usually broader than its length i.e., 14 µ × 15 µ (Waterhouse, 1963) or 12 µ × 13 µ (Sastry and Hegde, 1987).

3.1.4 *Epidemiology*

Disease is prevalent during South-West monsoon period and rains play a key role in disease development. Usually the disease makes its appearance about 15-20 days after the onset of South West monsoon (May-June) and persist till the end of monsoon (Aug.-Sep.) (Marudarajan, 1950a; Anandaraj and Saraswathy, 1985). High humidity and low temperature are congenial for the growth of the fungus (Coleman, 1910). Heavy rainfall with constant humid conditions and low temperature, alternate sunshine and rain favour the incidence of disease (Coleman, 1910; Narasimhan, 1922; Kamat, 1956). Reddy and Anandaraj (1980) studied the influence of meteorological factors in relation to disease incidence. They studied the data for nine years (1970-1979) and reported that the maximum crop loss of 50-90 per cent was recorded during 1979 when the rainfall was maximum with high humidity of more than 80 per cent throughout the year (Table 1). Besides, the lesser duration of sunshine hours favour the development of the pathogen. Heavy wind and, to a certain extent, birds and small insects help in the spread of the disease and also the rain splashes (Coleman, 1910; Anandaraj, 1985). There is no definite pattern in the spread of the disease (Anandaraj and Saraswathy, 1985). Based on epidemiological data a linear model to predict the disease four days in advance has been developed (Anandaraj *et al.*, 1992). Zoospores released from the sporangia germinate in films of water and penetrate the nut surface through stomatal openings.

The pathogen survives in the form of dormant mycelium or resting spores (Chlamydospores). These propagules may be present on the tree top, i.e., on the infected died bunches or on the upper layers of the soil (Coleman, 1910; Kamat, 1956; Singh, 1973) or the bud rot affected dead palms remaining in the garden may also act as a potent source of primary inoculum.

3.1.5 *Control*

Before the introduction of chemical control method, farmers were practising the method of covering the bunches with 'kotte' or 'karada', i.e., covering with leaf sheaths or grass respectively to fight against the disease. Coleman (1910) studied the possibility of using

Table 1 : Meteorological data for the years 1970-1979 at CPCRI, Research Station Vittal*

Months	Rainfall (mm)	Temperature °C		Humidity (%)	Sunshine (h)
		Max.	Min.		
May	169.1	32.9	24.1	74.1	7.6
June	982.5	29.6	23.0	85.1	3.9
July	1339.7	27.9	22.6	89.2	2.9
August	779.1	28.4	22.7	87.2	3.7
September	294.2	29.9	23.0	81.9	6.1
October	188.0	31.3	22.7	77.5	5.7

*Mean values.

Bordeaux mixture either alone or in combination and reported that Bordeaux mixture (1%) with resin and washing soda can control the disease. Further, the efficacy of adhesives and spreaders with Bordeaux mixture was studied (Narasimhan, 1924; Anony., 1927). Narasimhan (1928a; 1928b) recommended Martin's Bordeaux mixture which is a combination of potash alum and calcium. The usefulness of different vegetable oils was also recommended against Koleroga (Anony., 1932; Thomas and Marudarajan, 1938; Rao, 1960). Later studies revealed that prophylactic spraying with 1 per cent Bordeaux mixture alone is as effective as Bordeaux mixture with any sticker or spreader (Thomas and Marudarajan, 1938; Anandaraj, 1985). Other than Bordeaux mixture, many other chemicals such as mercurised copper oxychlorides and proprietary copper fungicides were also tried. Copper oxychlorides were toxic even at 0.5 per cent concentrations and proprietary fungicides were not effective (Anony., 1969a). Bordeaux mixture possessed good sticking quality and was available on the nut surface for 40-45 days (Anony., 1969b; Anandaraj, 1985). Accordingly, the spraying schedule was recommended. Spraying has to be given first before the onset of monsoon and the second after 40-45 days. A third spray is necessary only if persistent heavy rains prolong. Though Bordeaux mixture is effective in controlling or reducing the incidence of the disease, this method has the disadvantage of repeated sprayings which become impossible during monsoon period. This necessitated the search for an alternate method, i.e., either a better systemic fungicide or mechanical method. Studies carried out in recent years with newer systemic fungicides, viz., Aliette and metalaxyal revealed that both the chemicals were effective at 0.5 per cent concentration (Sastry and Hegde, 1985) or Aliette at different concentrations tested was on par with 1 per cent B. mixture (Table 2) (Anandaraj and Saraswathy, 1986). Of mechanical methods, covering of the bunches with polythene covers (200

Table 2 : Effect of different fungicides on the incidence of fruit rot of arecanut

Treatment	Concentration (%)	Disease incidence (%)
Metalaxyl	0.10	55.81*
Metalaxyl	0.15	51.34*
Hylenec	1.00	43.50*
Chlorothalonil	0.10	65.23
Etridiazol	0.10	59.62
Etridiazol	0.15	53.38
Aluminium tris ethyl phosphonate	0.10	25.04
"	0.15	22.63
"	0.20	29.55
"	0.25	24.97
Bordeaux mixture	1.00	35.31
C.D. (P = 0.05) = 17.94		

Treatment difference significant at 5% level.
*Data for 1991 only.

gauge) gave good control of the disease (Sastry and Hegde, 1985; Saraswathy cf. Anony., 1983, 1984, 1985). This method has the advantage of gradually eradicating the pathogen from the gardens within few years and also repeated climbings for the purpose of spraying can be avoided. Covering has to be done before the onset of heavy monsoon.

Besides the prophylactic methods, sanitation also is equally important to reduce the inoculum potential in the field. Infected shed nuts, dried bunches retained on the palm and dead crowns of bud rot affected palms are to be removed and destroyed by burning.

3.2 Foot Rot or Anabe Roga

'Anabe' literally means a disease caused by bracket forming fungus. The disease is also known as 'foot rot', 'root rot' or 'betelnut plague'. The disease was first reported on arecanut palm from the erstwhile Mysore State (present Karnataka State) by Coleman (1911). Butler (1913) reported its occurrence on coconut palm and called it as *Polyporus lucidus*. This disease was later reported from Tamil Nadu, Kerala, Assam and Bengal (Sharples, 1928).

3.2.1 *Losses*

The disease is more prevalent in neglected gardens. About 7 per cent loss of palm has been estimated due to this disease. The survey conducted recently shows 5-7 per cent of areca palms are affected by the disease. In some endemic areas, the incidence has been found to be as high as 20-25 per cent (Kumar and Nambiar, 1990).

3.2.2 *Symptoms*

Symptoms of the disease is akin to that of drought. The initial visible symptom is the yellowing of outer whorl of leaves, which gradually extends to the inner whorls. The leaves exhibit wilting symptoms and droop down covering the stem (Fig. 4). The development of inflorescence and nuts is arrested and nuts already formed are shed. The leaves are also shed one by one leaving a tuft of one or two leaves along with the spindle at the centre. At later stages, the weakened crown topples off leaving a bare trunk (Fig. 5). The affected palms initially exhibit a dull brownish patch at the base of the trunk, which later enlarges in size. In the advanced stages, a brownish gummy liquid exudes from this patch. The lesions can be seen up to 0.5-1 m height. The bracket or the fruiting body (anabe) of the fungus *Ganoderma* develops at the base immediately after the death of the palm. Often the brackets appear on the stumps only after felling the affected palms (Fig. 6). Occasionally, the brackets develop at the basal portion of the palms in the advanced stages of the disease. The disease also cause damage to the internal tissues of the stem. The tissues will be rotten with brownish discolouration to about one metre height from the ground level. The discolouration extends through the bole to the roots. The infected roots are discoloured, brittle, dry and exhibit a musty smell. The fungal invasion interrupts the uptake of water and nutrients by the palm, leading to yellowing and wilting.

Fig. 4 : Symptoms due to *Ganoderma* wilt.

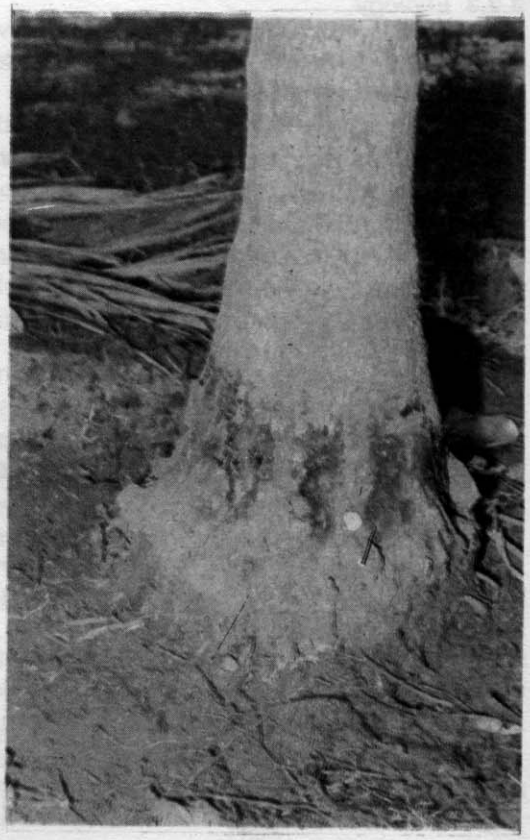

Fig. 6 : Base of *Ganoderma* affected palm.

Fig. 5 : Crown has fallen due to *Ganoderma* infection.

3.2.3 *Etiology*

The disease is caused by a bracket fungus *Ganoderma* spp. The fungus has been isolated and pathogenicity has been established. Venkatarayan (1936) reported *G. lucidum* as the primary pathogen of the disease but could not get infection on inoculation to living trees. Kumar and Nambiar (1990) established the pathogenicity on living palms with 8-9 months after inoculation.The fungus grows well in Waksman's agar medium. There are both thick and thin hyphae resembling stag horn; clamp connections are very common. The hyphae remains hyaline for a fairly long period and later turns to light brown colour on storing. The fungus forms both terminal and intercallery chlamydospores.

The fungus is soil borne inhabiting the dead plant materials in the soil. The fungus spreads from plant to plant through root contact or through the debris of affected stumps left over in the garden.The disease is also disseminated through water. Excessive irrigation, repeated ploughings, digging, etc., in infected gardens will help in quicker spread of the disease.

The pathogen has a wide host range attacking a variety of palms and several forest, avenue and fruit trees. Hosts belonging to 19 families, 36 genera and 48 species have been recorded (Naidu *et al.*, 1966). Some of the economically imponant plant species like *Cocos nucifera, Cassia siamia, Pongamia glabra* (Venkatarayan, 1936), eucalyptus (Bagchee, 1953; Bakshi, 1974), *Casurina equisetifolia* (Bakshi, 1951), *Mangifera indica* (Govindu *et al.*, unpublished) and fruit plants such as *Artocarpus heterophyllus, Citrus* spp., grape vine, etc., are known to be affected by the pathogen. If these plants are planted near arecanut garden, they may act as alternate host for the pathogen.

3.2.4 *Control*

Based on the research findings, a set of agronomic practices have been developed for the integrated management of *Ganoderma.*

Since the infected stumps and roots act as the main foci of infection and spread of the disease, strict phytosanitary measures are to be adopted by removing and destroying the stumps along with the roots.

In irregularly planted arecanut gardens with closer spacings, the intensity of disease is higher compared to regularly planted gardens with a spacing of 2.7 × 2.7 m. The chances of root to root contact is more in closely spaced gardens and hence, higher disease intensity. Diseased palms are to be isolated from the neighbouring healthy palms by digging isolation trenches of 30 cm wide and 60 cm deep around the affected palms so as to avoid root contact between healthy and diseased palms. In a study conducted in Karnataka over a period of 4-5 years showed that the infection could be reduced from 17.6 to 2.4 per cent by digging trenches around affected palms. Repeated ploughing and deep digging in diseased gardens should be avoided as this will help to carry the infected material from diseased site to healthy ones.

The pathogen, *Ganoderma* has a wide host range affecting crop species like coconut, jack, mango, eucalyptus, tamarind, etc., and hence planting these species, especially in gardens or areas where there is history of the disease, is not advisable. Planting of few rows of economically important and resistant plant species is a good practice to help break the continuity of pure plantations so as to check the spread of the disease. Since disease intensity is more in ill-drained gardens, good drainage conditions should be provided by digging drainage channels. To improve the vigour of the palms, regular fertilizer application is a must. The organic matter content in the soil must be increased by addition of 15-20 kg of FYM and 15-20 kg of green leaf. In addition, 2-2.5 kg of neem cake may be applied per palm during September each year.

Besides strictly adopting the above cultural and manurial operations, palms in the early stage of the disease may be root fed with 125 ml of 1.5 per cent calixin solution at quarterly intervals, i.e., during March, July, October and January. Singh (1985) recommended soil drenching with 2 per cent Bordeaux mixture at monthly intervals throughout the monsoon. Recent field fungicidal trials indicated that drenching the base of the palms with captan or bavistin at 0.3 per cent concentrations will prevent the spread of the disease (Table 3) to neighbouring palms (Kumar and Nambiar, 1990).

Table 3 : Effect of fungicides on the control of anabe disease of arecanut

Chemical and concentration	No. of palms affected initially	No. of surrounding palms treated (1978)	No. of palms showing fresh infection at the end of the observation (1982)	Percentage of fresh infection
Captan (0.3%)	4	24	1	4.1
Vitavax (0.2%)	4	26	5	19.2
Thiram (0.3%)	4	20	3	15.0
Difolatan (0.3%)	4	25	3	12.0
Cuman (0.3%)	4	20	5	25.0
Bavistin (0.3%)	4	20	2	10.0
Dithane Z-78 (0.3%)	4	18	4	22.0
Control	4	16	6	37.5

3.2.5 *Fluorescent Antibody Technique for Early Detection of Ganoderma*

By the time, *Ganoderma* disease symptoms externally manifest, the disease would have reached the middle stage and as such adopting control measures at the stage may not pay good dividends. Hence, it is necessary to diagnose the disease in the early stages itself. This is possible by fluorescent antibody technique. This sensitive technique has been standardised at CPCRI and has been very much helpful in detecting the *Ganoderma* infection in its early stages of infection.

3.3 Inflorescence Die-back and Button Shedding

The disease is of complex nature and is responsible for low fruit set in arecanut (Anony., 1971). No systematic survey has so far been conducted to assess the crop loss due to this disease but up to 60 per cent of the areca palms are seen affected by this malady (Saraswathy *et al.*, 1977).

3.3.1 *Symptoms*

Disease first appears as yellowing of the rachille of the male flowers. As the disease increases, yellowing extends further to the main rachis. The tip of the rachile becomes dark brown and the discolouration spreads from tip towards the base of the rachis involving the entire rachis resulting in shedding of the buttons or the female flowers (Fig. 7). The fungus also invades the developing embryo of the female flowers, which eventually shrivels showing a brown discolouration. As the name indicates, in advanced stages, infection cause drying up of the rachille from tip towards the base resulting in a condition called as die-back (Rao, 1965). Infected female flowers and rachis harbour the pathogen as concentric rings of light pink coloured conidial mass (Anony., 1961). The pathogen gains access to the host either through the scars of shed male flowers or through stigmatic end of female flowers. Closer examination of the infected female flowers reveal the presence of mycelium at the stigmatic end. The disease is prevalent throughout the year but becomes severe during summer months, i.e., February-May (Saraswathy *et al.*, 1977; Chandramohanan and Kaveriappa, 1985).

3.3.2 *Etiology*

Nutritional imbalance and physiological factors are reported as the possible causes of die-back and button shedding. Shedding of female flowers due to lack of pollination and failure of fertilization have also been reported (Raghavan and Baruah, 1956). High temperature especially during summer months accelerate yellowing and drying of rachis.

The fungus *Gloeosporium* sp. was reported to be associated with shed female flowers and inflorescence (Anony., 1938). The toxic substance produced by the fungus was reported as the cause of drying of rachille and shedding of buttons (Menon, 1961). A fungus *Colletotrichum gloeosporioides* conidial state of *Glomerella cingulata* (Ston.) Splaud and Shrenk was constantly associated with more than 70 per cent of the shed buttons and infected inflorescence (Saraswathy *et al.*, 1977). The primary pathogenic nature of *C. gloeosporioides* was confirmed through inoculation experiments by production of characteristic symptoms of the diseases, viz., shedding of female flowers and die-back of inflorescence.

3.3.3 *Control*

Since lack of pollination was considered as one of the contributory factors, assisted pollination was recommended to rectify the same. An increase of fruit set from

12 to 26.4 per cent was achieved by this method (Bhat, 1963). Spraying with growth regulators such as GA and 2, 4-D at different concentrations (Yadava *et al.*, 1974) and application of wood ash (Saidalikutty, 1951) were found to be beneficial to certain extent in correcting the malady.

Constant association of the pathogen necessitated the need for the selection of an effective fungicide for the control of the disease. A combination spray of shell copper with endrex (Anony., 1960) or 1 per cent Bordeaux mixture with endrex (Anony., 1963) increased fruit set. Spraying with Dithane Z-78 and heptane antibiotic (aureofungin sol) was effective in reducing the button shedding (Anony., 1971). Fungicides like benomyl (0.1%), captan (0.25%), thiram (0.25%) and phenyl mercury urea at 0.1 per cent inhibited the growth of *C. gloeosporioides in vitro*. In the field DMOC, (0.1%), heptane antibiotic + copper sulphate (50 ppm each) and zineb (0.4%) were effective in reducing the shedding and die-back symptoms due to the pathogen (Saraswathy *et al.*, 1975). First spraying has to be given at the time of opening of the female flowers and second after a gap of 20-25 days. Removal and burning of inflorescence helps in reducing the lead of inoculum in the field, its further spread and intensity of disease.

3.4 Bud-rot

Bud-rot is a fatal disease of areca palms, characterised by the rotting of terminal bud and surrounding tissues. Coleman (1910) observed that the pathogen causing mahali also cause rotting of the base of the growing bud and ultimately kills the palm. The diseases was first recorded in severe form in heavy rain fall tracts of Karnataka (Nambiar, 1949). The disease usually occurs during monsoon months and the fresh infections initiating after monsoon, i.e., from November onwards becomes severe during subsequent dry months unlike the incidence of mahali in heavy monsoon season (Marudarajan, 1950a). Palms of all age groups are prone to this disease but young palms are found to be more vulnerable. An annual crop loss of one per cent (Coleman, 1910) or heavy loss in endemic areas (Doraisami, 1956) have been reported.

3.4.1 *Symptoms*

Initial symptom is the yellowing of the spindle or the central leaf. As a result of infection the spindle loses its natural light green colour which, in the advanced stages, turn to yellow or brown (Fig. 8). Infection spreads to the adjacent younger leaves very rapidly. The spindle slumps to a side and can be drawn out with a gentle pull. Subsequently, the other whorl of leaves also get discoloured or turn yellow, droop and drop off one by one leaving a bare stem. As a result of colonisation of secondary organisms on the rotten spindle the infected palm emits a disagreeable odour (Coleman, 1910).

3.4.2 *Etiology*

The pathogen *Phytophthora* causing mahali may pass on to the growing point and cause bud rot. Coleman (1910) also suggested independent infection of leaf sheath

surrounding the growing point. Association of *Gloeosporium* sp. (Rao, 1962b) and *Thielaviopsis paradoxa* (Anony., 1970; Sarma and Murthy, 1971) causing crown rot disease in Assam also have been reported as agents of bud-rot disease. Naidu and Kumar (1964) reported *Nigrospora sphaerica* causing severe rotting of young leaves paves the way for the other bud-rot organisms resulting in death of palms. Naidu (1960) noticed a sort of bacterial rot of spindle of young palms in many parts of Karnataka. The disease is characterised by discolouration and drying of heart leaf. Lightning injury also causes rotting of the bud in areca palms.

3.4.3 *Control*

Early detection of the disease and prompt removal of the infected tissues will help in the recovery of the palms and also prevents the spread of the disease. Infected tissues may be scooped off by making longitudinal side slit and treated with 10 per cent Bordeaux paste. The treated portions have to be covered with polythene sheet or areca leaf sheath to ward of insects visiting the infected tissues and also avoid washing off of chemical. Removal and destruction of dead areca palms and also bunches affected by mahali and drenching of crowns of the surrounding healthy palms with 1 per cent Bordeaux mixture will minimise the disease incidence (Nambiar, 1956; Anony., 1960; Lingaraj, 1969). Drenching of the crown with mercuric compounds like wet ceresan (0.2%) or leytosol reduces the incidence of crown rot by bacteria (Naidu, 1960). Since high humidity and overcrowding of plants predisposes the palms to infection especially in low lying areas, closer planting and heavy intercropping should be avoided.

3.5 Band or Hidimundige

'Band' is a peculiar condition of the palm rather than a disease caused by physiological disorder within the palm or adverse environment of the particular spot where the palm is standing. The first report of this malady dates back to 1889-1890 from parts of Maharashtra (Joshi and Joshi, 1952).

The disease is reported in a severe from particularly in parts of South Maharashtra, North Kanara in Karnataka and also to a certain extent in plains of Karnataka. 'Band' means 'barren' in Marathi language, as the diseased palm ceases to bear fruits. In Karnataka this diseases is known as 'Hidimundige'—a sort of constriction at the crown region. Similar types of disorder or disease was reported from Sri Lanka as 'pencil point' and as 'rosette' disease from Australia. Affected palms in the advanced stages remain unproductive for a long time or recover itself but it is of rare occurrence.

3.5.1 *Losses*

Loss due to this malady varies from 5-25 per cent depending upon the locality (Joshi and Joshi, 1952) and plantations in the plains are affected more than those situated on hill tops (Kibe *et al.*, 1956). Disease incidence as high as 50 per cent has been reported from Kolaba and Ratnagiri districts of Maharashtra. Recent survey conducted in Konkan

regions of Maharashtra revealed that the disease incidence varies from 0-16.66 per cent (Salvi *et al.*, 1985).

3.5.2 *Symptoms*

The first visual symptom is the production of leaves smaller than normal size characterised by dark green colour which are brittle and crinkled with wavy margin. Gradually, as the disease increases, the other symptoms, such as reduction in the internodal length, formation of small bunches and tapering of stem, etc., appear. In advanced stages, the crown exhibits a 'rosette' shape due to the failure of natural opening of the leaves and tightly binds to the top portion of the palm. (Fig. 9). This condition prevents the normal growth of the bud. Rarely multiple shoots are also seen due to the persistent leaf bases of the lower leaves, sometimes shoots may come out through the side of tightly folded earlier leaves (Joshi and Joshi, 1949). Bunches may or may not form in severe cases and if at all formed they will be small and malformed. Changes are seen in the root system of the diseased palms. Roots are poorly developed (Kibe *et al.*, 1956; Patel and Rao, 1958) and they will be crinkled, short and brittle.

3.5.3 *Etiology*

The exact cause of the disease is still eluding. No biotic agent either alone or in combination are responsible for the disease (Anony., 1951). Infestation by the nematode *Aphelenchus coccophilus* reported as the cause of this disease (Thirumalachar, 1946) was ruled out later (Venkatarayan, 1946). The disease is not transmitted by any means than natural occurrence (Nayar, 1976). Nutritional imbalance of the palm also has been attributed as a possible cause of the disease (Daji, 1948; Joshi and Joshi, 1952; Kibe *et al.*, 1956). Studies on nutrient status of soils of healthy and disease affected areas did not show any significant difference in the major nutrients but low concentrations of zinc was noticed in diseased soils whereas no change was recorded for boron and magnesium (Joshi and Joshi, 1952).

The disease is prevalent in water logged soils. Poor drainage, low soil fertility (Gokhale *et al.*, 1916; Daji, 1948; Nambiar, 1951) or environmental factors (Coleman, 1910) are reported as possible causes or cause of the malady. Presence of lateritic subsoil pan or hard clayey soils are found to be factors associated with the disease (Patel and Rao, 1958).

3.5.4 *Control*

Since biotic agents are not involved in the occurrence of the disease spraying of plant protection chemicals is of no value. Good soil management and improvement of drainage are important operations to reduce the disease incidence. Palms in well managed gardens respond more to manurial treatments against the disease than those in neglected gardens (Kibe *et al.*, 1956). Removal of hard soil strata, if present, will increase the soil aeration and thereby improve the formation of better root system and

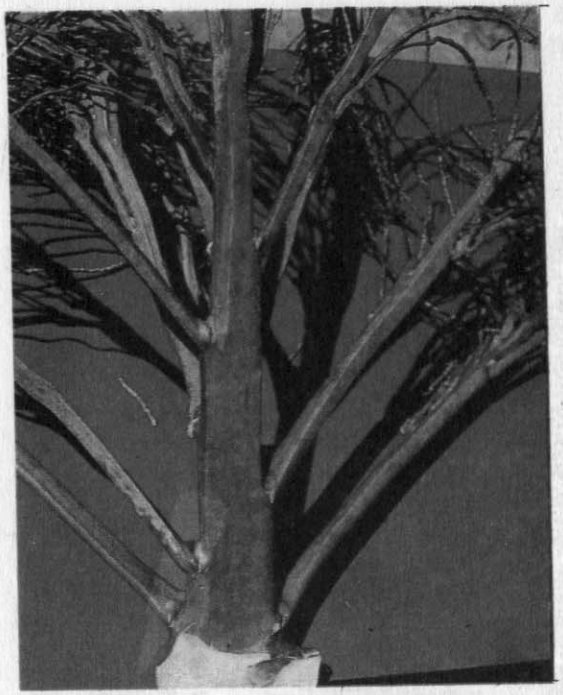

Fig. 7 : Inflorescence die-back—shedding of buttons.

Fig. 8 : Bud rot disease—crown affected.

Fig. 9 : Band disease—Rosette crown.

also foliar application of micronutrients minimise the disease intensity (Patel and Rao, 1958). Better soil management and incorporation of a mixture of equal quantities of copper sulphate and lime to the soil could check the disease effectively (Rao, 1960).

3.6 Sun Scorch and Stem Breaking

Stem breaking is another disorder of areca palms which is the resultant of constant exposure of the palms to severe solar radiation. Arecanut palms exposed to south-western sun are more prone to stem breaking. This disease was reported decades ago from parts of Dakshina Kannada district of Karnataka.

3.6.1 *Symptoms*

Symptoms appear on palms as golden yellow spots on the exposed side. These spots later turn to dark brown and gradually longitudinal fissures of 1-3 cm deep develop on these portions (Fig. 10). Further colonisation by saprophytic microorganisms weakens the stem and accelerate the damage and finally the stem breaks during monsoon and heavy wind (Seshadri and Rawther, 1968).

3.6.2 *Etiology*

Number of fungi, viz., *Ceratostomella paradoxa, Lenzitus* sp., *Acrothecium* sp., *Polyporus* sp., *Nigrospora* sp., *Pestalotia palmarum, Fusarium* sp. and *Ganoderma lucidum* were reported from infected palms. Of these, *G. lucidum, C. paradoxa, Lenzitus* and *Polyporus* could cause infection of stems of young palms when they were wound inoculated (Coleman and Rao, 1918; Patel and Rao, 1958).

3.6.3 *Control*

Trailing pepper wines on the stem will minimise the incidence of scorching and stem breaking. Raising fast growing trees on the south-western side of the garden (Govindakutty Kurup, 1955) or protecting the stem with dry areca leaves (Shama Bhat *et al.*, 1956) will also reduce the incidence of the disease. Adapting a suitable alignment of planting also is suggested to minimise the disease (Bhat, 1965).

3.7 Nut Splitting

Nut splitting is a physiological disorder than a pathological problem. Locally, it is known as 'Anduadakke' in Karnataka and 'Achikeral' in certain parts of Malabar in Kerala (Nambiar, 1949). Arecanut palms of younger age group, i.e., 10-25 years only show this abnormality and the disease is generally seen during heavy monsoon.

3.7.1 *Symptoms*

Symptoms are noticed on nuts as yellowing when they are half to three-fourth mature (Fig. 11). Splitting may occur near the perianth or the base or from both ends (Fig. 12). As a result of splitting of the husk, kernel also gets split and exposed (Bavappa and Sahadevan, 1952). All the nuts in a bunch may be shed due to this disorder.

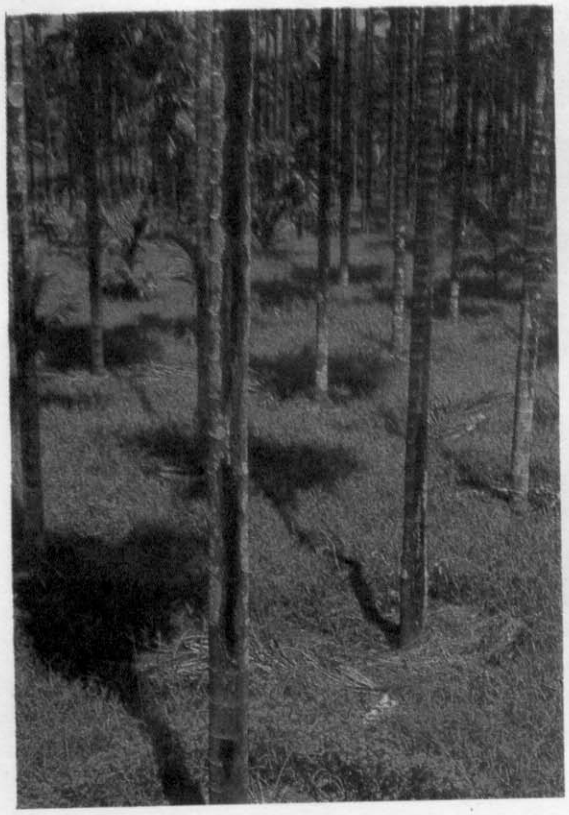

Fig. 10 : Sun-scorch on areca palm.

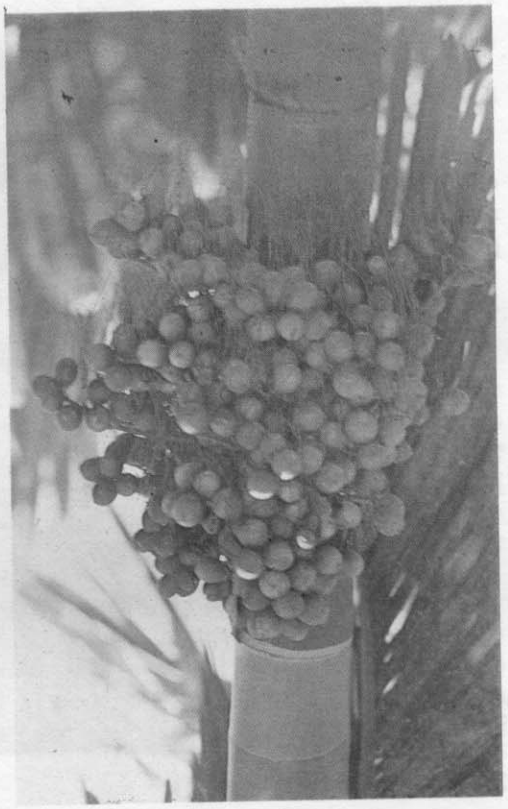

Fig. 11 : Pre-mature yellowing of areca fruits.

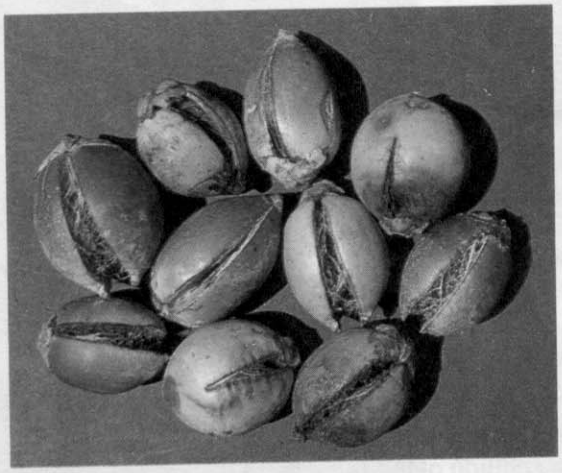

Fig. 12 : Nutsplits in arecanut.

3.7.2 *Etiology*

Sudden flush of water after a period of drought may cause this abnormality. Sometimes, inadequate moisture in soil upsets the rhythm of development of pericarp and kernel inside. Potash deficiency is also reported as a probable cause of this malady (Ishwara Bhat, 1961).

3.7.3 *Control*

The splitting can be reduced by checking the flow of excess sap to the developing nuts through making longitudinal side slit just at the base of the spadix (Bavappa and Sahadevan, 1952). This method is effective if done when the nuts are half mature. Improvement of drainage in areas of high water table is known to help in minimising the incidence. Application of potash fertilizers (Ishwara Bhat, 1961) and spraying of borax at 2 g per litre of water during the early stages of the disease also reduce splitting.

3.8 Bacterial Leaf Stripe

The first report of bacterial disease of arecanut is that of Orion who reported the natural infection of arecanut and other palm species by *Xanthomonas vasculorum* (Cobb) Dawson, the incitant of gumming disease of sugarcane. Rao and Mohan (1970) reported the occurrence of the bacterial leaf stripe disease of arecanut in Tumkur district of Karnataka State.

3.8.1 *Symptoms*

The leaf stripe disease on arecanut differs from the bacterial disease reported so far in symptomatology being purely parenchymatous in nature. The initial characteristic symptoms are appearance of one to four mm wide dark green, water soaked, transluscent, leniar lesions or stripes alongside and parallel to the midrib of the leaflet to its main veins (Fig. 13). They may originate at any point on the lamina. The lesions margins are straight and well defined; but at places may appear wavy due to the lateral spread of the lesion. The lesions are covered with abundant bacterial exudate (which is a striking feature of the disease) on the lower surface of the leaflets. The exudate is creamy white, dispersed all over the lesions surface making it strikingly slimy when wet. On drying, it forms a waxy film or creamy white to yellowish flakes or fine granules or irregular masses. In advanced stages, the lesions may be one cm or more wide and several cm long often involving the entire length of the leaflet. Portions of midrib and veins of leaflets also get discoloured. All the leaflets in a frond may be affected resulting in complete or partial blighting and severe cases entire crown may be affected (Fig. 14); when growing buds are affected 'bud rot' ensues often resulting in the death of the palm (Fig. 15).

3.8.2 *Etiology*

Microscopic examination of the affected leaf tissue shows profuse bacterial streaming throughout the cut surface indicating the parenchymatous nature of the disease

Fig. 13 : Bacterial leaf stripe.

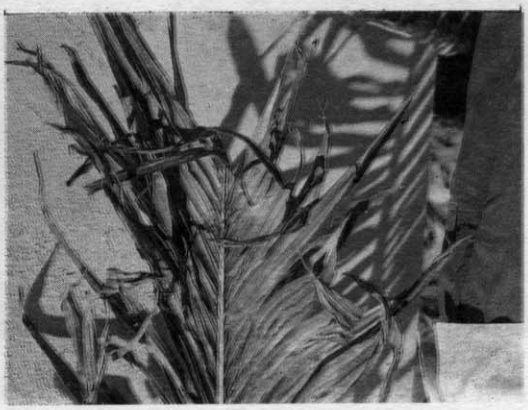

Fig. 14 : Severely blighted frond.

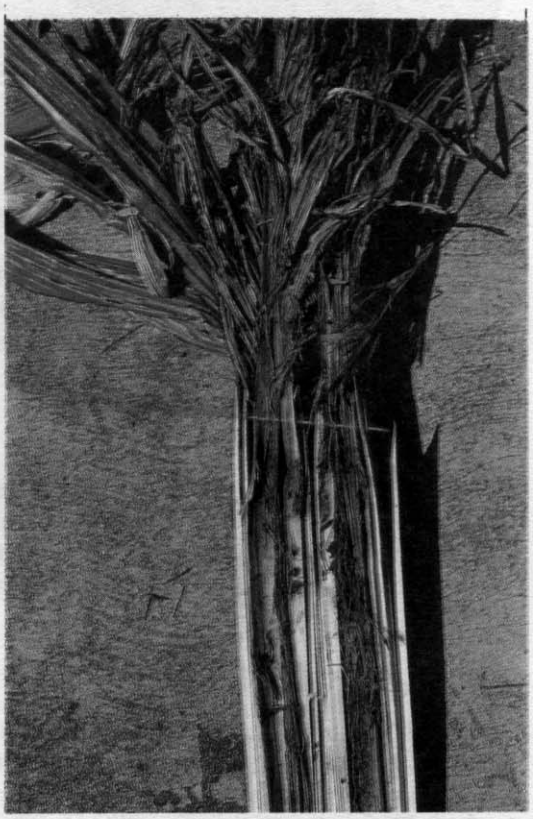

Fig. 15 : Bacterial leaf stripe leading to infection of growing bud.

(Rao and Mohan, 1970). On the basis of the cultural and morphological characters, the pathogen was identified as *Xanthomonas compestris* pv. *arecae* (Rao and Mohan, 1970). The pathogen produced typical symptoms on artificial inoculations (Rao and Mohan, 1970; Kumar, 1981). The organism also produced dark green, water soaked elongated lesions on coconut and on ornamental palms (Kumar, 1981).

The bacterium produces large quantities of extracellular polysaccharide toxin. The ability to produce polysaccharides by phytopathogenic bacteria has been linked to the virulence of the pathogen. In highly susceptible arecanut cultivars, the proliferation of the pathogen result in copious amounts of gummy exudate, chlorosis and localized water soaking. The purified polysaccharide toxin produced characteristic symptoms on detached arecanut leaves. The toxin is a heteropolymer of glucose, galactose, mannose and small amounts of glucuronic acid (Kumar, 1981). Most of the phenolic compounds produced were common to both healthy and diseased leaf tissues of arecanut. The

diseased leaf tissues contained an extra phenolic compound formed as a result of host pathogen interaction (Kumar, 1981).

3.8.3 *Epidemiology*

The disease remains aggressive during the active monsoon season (July-October) when the average monthly rainfall is 130 mm or above with more than 10 rainy days per month. Afterwards, there is a decline in disease incidence. Temperature above 30°C and below 17°C was found to slacken the disease spread (Kumar, 1981). The organism does not survive in soil for long, indicating that the soil may not be a primary source of inoculum. Palms of younger age group (3-5 years) were highly susceptible to the disease than older palms. As age advances, susceptibility character also varied both with age of the palm as also with the age of the leaf (Kumar, 1981).

Close spacings were found to be congenial for disease development and spread. Frequent irrigation (once in 5-10 days) was found to aggravate the disease incidence. Higher levels of nitrogen and green leaf manure were found to favour disease development. Phosphorus and potash did not have any influence. Intercropping with banana in locations with bacterial leaf stripe disease was found to aggravate the disease intensity and was detrimental to arecanut plantations (Kumar, 1981).

3.8.4 *Control*

Antibiotics like tetracycline and its formulations are effective as prophylactic and curative control measures. Spraying of the antibiotics at 500 ppm concentration was effective. Stem injection with antibiotic showed longer residual effect than foliar application (Kumar, 1981).

3.9 Stem Bleeding

The disease is prevalent in isolated pockets in all the arecanut growing states of South India. Nambiar (1949) reported the occurrence of stem bleeding disease from Mettupalyam area in Tamil Nadu. The disease in areca palm closely resembles the stem bleeding in coconut (Sundararaman *el al.*, 1928). Younger and middle aged palms are more susceptible to the disease (Patel and Rao, 1958).

3.9.1 *Symptoms*

Symptoms appear mostly on the lower portions of the stem as small discoloured depressions in the initial stages. These spots later coalesce and cracks develop on the stem. With the progress of the disease, the fibrous layer disintegrates which eventually hallows up to varying depths along the infected portion. Crowns of affected palms get reduced in size followed by reduction in yield. A dark brown gummy exudate oozes out from the cracks (Sundararaman *et al.*, 1928) (Fig. 16).

3.9.2 *Etiology*

The disease is caused by *Thielaviopsis paradoxa*, the causal organism of stem bleeding disease in coconut.

3.9.3 *Control*

The disease has been found to be serious in gardens with poor drainage. Improving the drainage to remove excess moisture from the soil may help in minimising the disease incidence. Scooping out the affected portions and application of coaltar (Sundararaman *et al.*, 1928; Nambiar, 1949) or Bordeaux paste (1:1:10) (Patel and Rao, 1958; Seshadri and Rawther, 1968) is effective in reducing the disease incidence.

3.10 Fungal Infection of Processed Arecanuts

In storage also, arecanuts are prone to infection by a number of fungi especially of the hyphomycetes. Lack of proper drying yard, improper spreading and turning of nuts, unexpected rains during drying period, etc., are predisposing factors for fungal infection of stored arecanuts. Infection causes discolouration of nuts and this, in turn, reduces the quality and fetches only low market value. Infected nuts are not good for chewing.

3.10.1 *Losses*

Extent of damage depends upon the conditions of the drying yard, season of drying, prevailing temperature and humidity. The longer the period of time taken for drying, the greater will be the chances of infection since the nuts are exposed to invading fungi for a long time. Severity of infection varied according to season. The percentage of infection varied 21-62 per cent when dried in February and October respectively. The possible reasons suggested for highest infection in October were the prevailing low temperature, high humidity (up to 91%) and unusual rains, i.e., 157 mm (Table 4).

Table 4 : Fungi involved in the spoilage of stored arecanuts

Fungi	Colour of infected kernel	Infection (%)
Aspergillus niger	Black	6.4
A. chivalieri	Yellow	
A. flavus	Yellowish green	
A. fumigatus	Velvetty green	
Penicillium sp.	Fetty olive green	1.3
Botryodiplodia theobromae	Grey to greyish black	19.3
Rhizopus sp.	Grey	1.8
Mucor sp.	Yellowish grey	0.7
Thielaviopsis paradoxa	Black	0.2

3.10.2 *Symptoms*

Both husk and kernel are affected. Infected kernel shows discolouration and disintegration of the white core and exhibits a hollow cavity. Colour of infected nuts depends upon the organism responsible for the spoilage (Jaleel and Govindarajan, 1969). The invading fungus (fungi) first attack the embryo (Jaleel and Govindarajan, 1969; Nambiar *et al.*, 1971) spread to the central white core of the endosperm and cause disintegration and then attack the adjacent lamella of the rumination (Fig. 17).

3.10.3 *Etiology*

During harvest by dropping the bunches on the ground, nut surface gets mechanical injury by abrasion and these points serve as foci of entry to the microorganisms (Nambiar and Nair, 1970). In such nuts, infection was upto 54.7 per cent and the following fungi were reported, i.e., *Aspergillus niger, A. flavus, Botryodiplodia theobromae* and *Rhizopus*. The infection is more in the initial days of drying and the maximum moisture loss takes place during the first 5-10 days whereas in the case of kernel the loss will be still slower as it is seated inside. The slow process of drying coupled with high nutrient content of the kernel favour the infection by fungi (Nambiar *et al.*, 1971).

⇐ **Fig. 16** : Stem bleeding in arecanut.

Fig. 17 : Infection of the stored arecanuts.

A number of fungi have been reported from the husk and kernel. The common forms recorded were *Aspergillus* sp., *Diplodia* sp., *Fusarium* sp., *Mucor* sp., *Penicillium* sp., *Thielaviopsis* sp. and certain aerobic bacteria (Anony., 1961, 1962b), *Cladosporium* sp., *Fusarium* sp. (Rawther and Nambiar, 197[illegible]), *Phomopsis heteronema* (Butler and Bisby, 1931), and *Colletotrichum gloeosporioides* (Saraswathy *et al.*, 1977). The fungi *A. niger arecae* (Lal and Chandra, 1953), *Subramanella arecae* (Srivastava *et al.*, 1962) and *A. chevalieri* (Anony., 1971) were also recorded on stored nuts. Besides the above, different species of fungi belonging to the imperfect group were recorded from the processed nuts also. The fungi, the damage and discolouration caused, etc., are given in Table 5 (Nambiar *et al.*, 1971; Rawther *et al.*, 1982). Of the fungi recorded from processed nuts *Botryodiplodia theobromae* followed by *Aspergillus* sp. caused maximum spoilage of 19.3 and 6.4 per cent (Table 4) respectively and the 'Sweet areca' showed the highest kernel infection (Nambiar and Edison, 1971). Highest infection in this cultivar was attributed to the availability of larger size of endosperm which serves as a good substratum for the invading organisms.

Table 5 : Infection of processed arecanut during different months

Months	Infection (%)	Range of temperature		Relative humidity (%)	Total rainfall (mm)
		Maximum °C	Minimum °C		
October, 1969	61.5	26.0-35.0	19.1-22.3	56.8-92.4	156.8
November, 1969	52.8	31.0-34.5	15.4-22.6	46.9-81.7	454.4
December, 1969	43.8	31.0-35.0	12.7-22.9	49.6-86.8	0.0
January, 1970	31.5	31.4-35.8	14.9-18.6	33.2-85.7	0.0
February, 1970	21.0	32.5-36.5	16.0-20.9	48.1-91.9	0.0
March, 1970	25.7	33.8-37.8	19.1-24.1	50.7-90.2	1.2

3.10.4 *Control*

Since soil is the primary source of infection eliminating soil contact while harvesting will help in minimising infection. No infection was recorded when nuts were harvested without soil contact and dried in hot air oven at 65°C for 63 h (Nambiar *et al.*, 1971) whereas in the conventional method of harvest followed by drying in mechanical drier (Namboodiri *et al.*, 1963) at 62°C for 72 h, 3.6 per cent of the nuts contacted the infection (Nambiar *et al.*, 1971). Treatment of nuts with fungicides, such as blitox (Anony.,1962) or Bordeaux mixture followed by drying on cement floor significantly reduced the percentage infection. Time required for drying on cement floor was less (Anony., 1972). Studies conducted on the methods of storage revealed that infection was very much less when nuts were stored in polythene lined gunny bags compared to that stored in plain gunny bags (Nambiar *et al.*, 1971). The percentage infection recorded was 17.7 and 32.3 per cent in nuts stored in air tight bins and in gunny bags respectively. Storage in polythene lined gunny bag reduces infection.

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