7

DISEASES

T. S. S. RAWTHER
Central Plantation Crops Research Institute,
Regional Station, Krishnapuram 690 533,
Kayangulam, Kerala, India

R. RADHAKRISHNAN NAIR
Central Plantation Crops Research Institute,
Research Centre, Palode, Pacha 695 562,
Kerala, India

and

N. SARASWATHY
Central Plantation Crops Research Institute,
Regional Station, Vittal 574 243,
Karnataka, India

Arecanut palm is affected by a number of diseases during different stages of its growth and development. About 20 diseases causing varying degrees of damages to the palm have been recorded in India. They are associated with 40 pathogenic and non-pathogenic forms of fungi and one bacterium. Based on the extent of damage and nature of disease, yellow leaf disease, *mahali, anabe,* inflorescence die-back and button shedding are considered to be the major diseases. Among the less serious diseases, the bud rot, bacterial leaf stripe, stem breaking and seedling diseases are important, since some of them at times occur epiphytotically in localised places and cause heavy losses. Etiology of diseases like *mahali,* bud rot and *anabe* is clearly known, while that of others like yellow leaf disease and *band* are uncertain. Microbial infestation of processed nuts is an important post-harvest problem causing considerable deterioration in the quality of the produce.

I. Diseases affecting adult palms

1. Yellow leaf disease

The yellow leaf disease (YLD) remains today as the most serious malady affecting the crop. This disease known in Malayalam as *kattuveezhcha* was reported from Muvattupuzha, Meenachil and Chalakudi areas in Kerala about a century

back (Nambiar and Sreenivasan, 1951). In earlier years, it was felt that YLD was more or less similar to the leaf and root diseases of coconut (Nambiar, 1949). In the *Malnad* areas of Karnataka the disease is known as *chandiroga* (Dastagir, 1963, 1965). The malady does not kill the palm outright but is only debilitating in nature.

i. *Crop loss*

A preliminary survey conducted during 1959–'60 showed that the disease had spread to all parts of Kerala with a maximum incidence of 90% in Quilon district (Anonymous, 1960b). The disease was also reported from the central regions of Maharashtra and Tamil Nadu (Anonymous, 1963b).

A comprehensive survey was undertaken in 1976 in the disease affected areas of Kerala and Karnataka. The results revealed that the malady is prevalent in almost all the districts of Kerala and in parts of Chickamagalur district of Karnataka. The results are presented in Table 7.1 (George, Nayar and Rawther, 1982, unpublished).

Table 7.1. *Spread of yellow leaf disease in Kerala and Karnataka*

State/district	Area under arecanut in '000 ha	Percentage of area affected by YLD
KERALA		
Cannanore	16.58	1.20
Kozhikode	8.10	0.70
Malappuram	15.50	Negligible
Palghat	3.70	Not available
Trichur	15.10	6.30
Idukki	1.70	97.00
Ernakulam	7.80	34.10
Kottayam	5.40	94.30
Alleppey	5.10	Not available
Quilon	9.20	75.40
Trivandrum	4.50	71.80
Total	*92.68*	*35.80*
KARNATAKA		
Chickamagalur (Koppa and Sringeri Taluks)	–	28.4

Thorough and systematic observation was made with respect to the pattern and spread of the disease at the CPCRI Research Centre, Palode located in a predominently affected area. Seedlings planted in 1961 in virgin soil manifested symptoms in 1968 and thereafter within a period of four years 80% of the palms contracted the disease (Rawther and Abraham, 1972). Though the spread was rapid, it did not follow any definite pattern.

Fig. 7.1 Yellow leaf disease affected palms

Studies on YLD affected palms revealed a reduction in yield to the extent of 50% over a period of three years immediately following the disease incidence. An average of 4% reduction in leaf fall was also noticed on account of the disease during the three year period (Anonymous, 1976).

ii. *Symptoms*

Nambiar (1949) observed the symptoms of the disease as yellowing of the leaves and shedding of both mature and immature nuts. The endosperm of the diseased nut has a blackish appearance and is soft to touch, which renders it unsuitable for consumption and fetches only very low price in the market. According to Menon (1963a), the first visible signs are translucent spots, 1–3 mm in diameter on the growing spindle. Brown necrotic streaks running parallel to the lamina are present in the unfolding leaves. As the leaves develop, yellowing starts from the tips of leaflets, gradually extending to the middle of the lamina. This chlorosis could be distinguished from the physiological yellowing by the abrupt demarcation between the green and yellow regions in the diseased leaf. Subsequent studies have shown that the first visible symptom is the yellowing at the tips of leaflets in two or three leaves of the outermost whorl (Rawther, 1976).

One or two leaflets in any part of the crown or the entire foliage may be affected by the disease. Tips of the chlorotic leaves eventually dry up. In the advanced stage, leaves are reduced in size, stiff and pointed, closely bunched and abnormally puckered (Fig. 7.1). 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 affected fruits fall off in large numbers.

Though endosperm discolouration is associated with foliar yellowing in majority of cases, palms exhibiting foliar yellowing sometimes produce normal nuts. Further, all the nuts produced in a bunch of a diseased palm may not show kernel discolouration (Rawther, 1976). Palms with normal green foliage standing among diseased ones were also observed to produce nuts with blackened kernel.

Nayar (1968) observed multi-nucleate cells, deranged tissue differentiation and palisade cells blocked with dark brown pigments in various stages of degeneration in leaves of affected palms. She also observed extensive degeneration in the phloem of the affected stem and leaves. Medullary rays were found disturbed and accumulation of starch grains was observed indicating impaired translocation. Nair (1976) found that the diseased leaves possessed smaller

epidermal cells, stomatal pores and midrib parenchyma cells. Blocking of xylem vessels of the older leaves of diseased palms, degeneration of the cortex and presence of tyloses in the xylem were also noticed in the diseased roots.

A formula for quantifying the disease was evolved after studying the association of the various symptoms in more than 2000 palms (George, Mathew and Nagaraj, 1980). Due weightage was given to foliar yellowing, necrosis and reduction in the size of the whole crown. In the formula

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

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

iii. *Etiology*

a. Fungi

A number of fungi like *Ceriospora arecae, Exosporium arecae, Leptosphaeria* sp., *Diplodia* sp., *Phyllosticta* sp., *Dimerosporina* sp., and *Trametes corrugata* were isolated from the diseased leaves (Menon, 1959a; Anonymous, 1963b). Roots of diseased palms yielded *Trichoderma* sp., *Pestalotia* sp., *Aspergillus sp., Penicillium* sp., *Fusarium* sp., *Colletotrichum* sp., *Acremonium* sp., etc. (Anonymous, 1963b). *Fusarium* sp., *Acremonium* sp. and *Colletotrichum* sp. were not pathogenic on inoculation to seedlings (Anonymous, 1963b, 1976). Species of *Pythium* and *Phytophthora* were isolated from the roots of disease affected palms using selective media (Rawther, T. S. S. 1982, personal communication).

b. Bacteria

Srivastava, Rao and Mohan (1970) reported bacterial streaming associated with YLD affected roots. Out of the two distinct forms of bacteria, one was tentatively identified as *Pseudomonas* sp. One and a half year old arecanut seedlings planted in heat sterilized soil in pots on inoculation with bacteria isolated from disease affected roots for a period of $2\frac{1}{2}$ years did not produce any disease symptoms (Anonymous, 1975).

c. Virus and mycoplasma

Paper chromatographic studies (Menon, 1961a) indicated that some proteins or their sub-units 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 (1960a) therefore suggested the possibility of a virus or virus like organisms being involved in the disease. Menon (1963a) transmitted yellow leaf disease to indicator plants *viz., Jatropha curcas, Canavalia ensiformis* and *Vinca rosea* using partially clarified leafsap.

Electron microscopic tests failed to show the presence of virus like particles (Raychaudhuri, S. P., 1966, personal communication). Nayar (1971, 1976) cultured mycoplasma like organism (MLO) from bits of diseased yellow leaf. Numerous colonies were produced in solid plate transfers from liquid cultures. Cross inoculation with rabbit antiserum using double cell diffusion technique showed that the areca antigen reacted against sandal spike-specific rabbit antiserum (Nayar, 1971).

Further, electron microscopic studies (Nayar and Seliskar, 1978; Seliskar and Wilson, 1981) showed the presence of MLO in the young sieve elements of YLD affected arecanut palms in Kerala and Karnataka. Diseased palms treated with the antibiotics like chlortetracyclin HCl (aureomycin) and tetracycline HCl (achromycin) failed to ameliorate the disease symptoms (Rawther, 1976).

d. Mites

Khandige, Patel and Bavappa (1957) reported association of mites with the yellow leaf. Menon (1960b) distinguished the yellowing caused by mite from the foliar yellowing due to yellow leaf disease.

e. Nematodes

Nair (1964) observed the presence of nematodes *Meloidogyne javanica, Helicotylenchus* sp. and *Tylenchorhynchus* sp. in the root zone of yellow leaf affected palms at Palode. Weischer (1967) recorded seven genera of plant parasitic nematodes from a few soil samples collected from the root zone of healthy and diseased palms.

Among the twenty-two genera of plant parasitic nematodes isolated from the root zone of healthy and disease affected palms, *Radopholus similis* was present in 111 out of 218 root samples. The occurrence of nematodes belonging to genera other than *Radopholus* in small numbers in samples may not be of any significance. Koshy, Sosamma and Sundararaju (1976) could not find any correlation between the presence of *R. similis* and the yellow leaf disease.

In a recent survey conducted in the healthy and yellow leaf affected arecanut gardens in Koppa, Sringeri, Sullia, Thirthahalli, Narasimharajpura and Somavarpet

in Karnataka state, 7/17 (41.2%) root samples from healthy and 16/20 (80%) from disease affected palms yielded *R. similis*. *R. similis* was also recorded in 7/17 (41.2%) and 14/20 (78%) soil samples collected from healthy and disease affected arecanut gardens respectively (Sundararaju and Koshy, 1980, unpublished).

f. Soil and nutritional factors

Water logging was considered to be one of the predisposing factors for the disease incidence (Anonymous, 1960b). Application of fertilisers, improved the condition of diseased palms (Anonymous, 1967). Menon and Kalyanikutty (1961) reported a reduction in the intensity of foliar yellowing when sprayed with salts of magnesium and manganese. Qualitative tests on diseased arecanut leaves recorded low pH, more water and HCl soluble iron. The high CaO/MgO ratio obtained was attributed to low content of magnesium in the diseased tissues (Anonymous, 1967). Diseased leaves had more silica, phosphorus and potash (Anonymous, 1964).

The extensive surveys in the nutrient status of healthy and disease affected gardens of Kerala and Karnataka were carried out in 1969 and 1974 (Mohapatra, Bhat and Harishukumar, 1976). No significant difference was noticed in the nutrient contents in leaves and soil between healthy and diseased samples. However, some difference in nutrient status was noticed between samples of Kerala and Karnataka. Soils of both the states were high in organic matter, low to medium in available P and K and contained adequate levels of exchangeable Fe, Mn, Zn and Cu. Soils from Karnataka were neutral in reaction while soils from Kerala were slightly acidic. The results of analysis of soils from healthy and disease affected areas of Kerala and Karnataka are given in Table 7.2 (Mohapatra et al., 1976).

Velappan (1969) observed that deficiencies of nitrogen, phosphorus and magnesium had some relationship with the disease. In the initial stages of the development of the symptoms, nitrogen, phosphorus and magnesium were found deficient but potassium and calcium were present in normal quantities in the affected palms (Anonymous, 1971). Yadava, Mathai and Vellaichamy, (1972) examined the role of major nutrient deficiencies in the symptom expression of the disease in a pot culture experiment, but could not produce any typical symptoms by this method. Root feeding of iron solution (as ferrous sulphate) did not produce symptoms similar to that of the disease (Mathai, 1976). Leaf tissues of healthy palms in general showed higher accumulation of nutrients (N, P, K and Mg) when compared with the diseased (Yadava, Mathai and Vellaichamy, 1973).

Table 7.2. *Mechanical and fertility constituents of soils from yellow leaf disease affected and healthy areas of Kerala and Karnataka states (mean values expressed on air dry basis)*

Constituents	Kerala (Trivandrum and Quilon)				Karnataka (Chickamagalur and Dakshina Kannada)			
	Category							
	Apparently healthy palm		Diseased palm		Healthy palm		Diseased palm	
	Low lying	High elevation	Low lying	High elevation	Low lying	High elevation	Low lying	High elevation
Sand (%)	72.48	62.60	69.92	65.91	59.68	55.84	56.77	58.45
Silt (%)	8.42	8.92	8.87	8.39	15.52	16.85	16.22	17.86
Clay (%)	19.08	26.34	21.20	25.68	24.79	27.29	27.26	23.68
pH (H_2O)	5.66	5.60	5.58	5.58	6.34	6.36	6.54	6.14
pH (KCl)	4.39	4.27	4.31	4.26	5.13	5.16	5.30	5.06
Organic carbon (%)	0.82	0.91	0.84	0.96	1.27	1.32	1.38	1.10
Available P_2O_5 (ppm)	12.17	6.06	9.23	6.01	21.46	14.84	18.95	9.05
Available K_2O (ppm)	66.52	84.00	75.81	84.90	144.10	163.18	153.18	130.80
Exchangeable Ca (ppm)	179.50	215.00	186.20	185.40	692.90	720.30	834.00	497.50
Exchangeable Mg (ppm)	47.90	68.80	45.20	59.00	173.10	190.90	220.10	152.40
Exchangeable Al (ppm)	54.35	64.53	59.95	68.84	1.59	0.10	0.45	Trace
Extractable Al (ppm)	66.97	97.48	83.30	105.60	41.21	42.10	48.23	37.84
$Fe^2 + Fe^3 +$ (ppm)	19.53	14.77	23.98	16.16	14.84	12.80	12.90	12.36
Exchangeable Mn (ppm)	8.03	8.83	7.65	8.82	12.50	17.47	15.68	16.30
Dithizone extractable Zn (ppm)	1.03	1.08	1.15	0.98	2.32	2.36	2.49	2.22
Exchangeable Cu (ppm)	2.04	1.92	1.99	1.97	3.94	4.02	3.57	3.32

The amino acid content (cystine, aspartic acid and threonine) of diseased leaves decreased in the early and middle stages of the disease and enhanced in advanced stages. On the other hand, lysine and arginine contents of leaves progressively increased with advancement of disease. Glutamic acid was absent in inflorescence and nuts of healthy palms and present in the diseased. Serine, arginine and threonine commonly found in arecanut stems decreased with increase in intensity of disease. Proline, cystine and histidine seen in the roots of healthy palms began to fall with the onset of disease. Thus an impaired amino acid metabolism is the characteristic feature of the disease (Nair, 1969).

iv. *Control*

a. Chemical control

Disease affected palms were given basal treatment of seven chemicals *viz.*, Brassicol, Vitavax, Bavistin, Cupramar, Blue copper–50, Furadan and Metham sodium for a period of five years. None of the treatments had any ameliorative effect on disease symptom, though palms treated with Blue copper–50, Cupramar and Furadan recorded increase in yield over the pretreatment to the tune of 31, 25 and 29 per cent respectively (Chandramohan, 1979). Diseased palms treated with tetracycline by root feeding and stem injection did not have any effect. Basal application of aureofungin solution also gave only negative results (Rawther, 1976).

A field trial conducted in the diseased tracts of Palode to study the effect of major and minor nutrients and irrigation showed that none of the treatments was effective in controlling the disease (Rawther and Abraham, 1972). A comprehensive package plan trial was laid out in Kerala and Karnataka involving all major and minor nutrients (Nagaraj, Mathai and Vellaichamy, 1976). Though the yield of the treated palms registered a decrease in majority of the treatments in Kerala, treatments NPK + lime, NPK + lime + boron and NPK + lime + zinc registered 15–19% increase in yield.

Palms treated with Hogland's solution showed an initial reduction in foliar yellowing but the improvement was not consistent (Anonymous, 1979). Foliar application of urea, diammonium phosphate and manganese sulphate did not show any ameliorative effect on the condition of the diseased palms (Rawther, T. S. S. 1982, personal communication).

b. Disease management

In view of the uncertain etiology, non–lethal nature of the disease and the absence of known remedial measures, it was felt necessary to evolve proper

management practices to contain the disease to the maximum extent possible. A mixed cropping experiment involving regular organic recycling in a disease affected 15-year old arecanut garden was conducted. Cowpea and Guinea grass were mix-cropped with arecanut. The organic manures for recycling were obtained from a dairy maintained. None of the treatments had any specific influence on the expression of disease symptoms. However, there was a general improvement in yield in all the treatments. Further it was noticed that the effect of the treatments on the microbial population was not consistent (Rawther, et al., 1979).

An observational trial to study the effect of the application of high doses of fertilisers (100g N, 160g P_2O_5 and 140g K_2O/ palm/year) along with 4 kg of dolomite, and organic manures and irrigation failed to give positive results (Rawther, T.S.S. 1982, personal communication).

c. Disease reaction of cultivars

Fifty accessions of indigenous and exotic collection of arecanut were planted from 1961 onwards at the CPCRI Research Centre, Palode – a diseased area – in order to assess their field reaction against yellow leaf. All of them except *Areca triandra* began to exhibit disease symptoms after about eight years of planting. Only a few instances of doubtful cases of disease incidence have been noticed in *A. triandra.*

2. *Mahali*

Mahali (heavy devastation) or *koleroga (kole* = rotting, *roga*=disease) is the most dreaded disease occurring in all the arecanut growing regions receiving heavy rainfall. Butler (1906) first recorded the disease in the erstwhile Mysore state. The disease was reported from the present Dakshina Kannada and Uttara Kannada districts of Karnataka as well as in small pockets of Malabar and Cochin areas (Coleman, 1910).

The exact crop loss caused by the disease is not available till now. However, an annual loss of 10–75% 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; Anonymous, 1960a). In the year 1978, the *koleroga* disease was rampant and crop loss ranged from 50–90% (Reddy and Anandaraj, 1980).

i. *Symptoms*

Butler (1906) described the disease with the characteristic symptoms of rotting and excessive shedding of immature nuts. Coleman (1910) described the symptoms with prolific illustrations in his pioneering work on this disease. The first visible symptom is the appearance of water soaked lesions on the surface of affected nuts. The infected nuts lose their lustre. The lesions gradually spread covering the entire nuts which rot and shed from the calyx (Fig. 7.2). A felt of white mycelial mass develops on the fallen nuts (Butler, 1906; Coleman, 1910). As the disease advances the fruit stalks and rachis of inflorescence are also affected (Fig. 7.3A) (Sundararaman and Ramakrishnan, 1928; Marudarajan, 1950a). Affected nuts are lighter in weight and possess large vacuoles and dark brown radial strands internally. Infections occurring later in the season result in drying

Fig. 7.2 *Koleroga* infected bunches

up of nuts without shedding (Marudarajan, 1950a). These nuts are often colonised by saprophytes like *Gloeosporium* sp. and are called 'dry mahali' in central Kerala. Apart from the quantitative loss by shedding of nuts at its various stages of development, the infected nuts are unsuitable for chewing due to deterioration in quality.

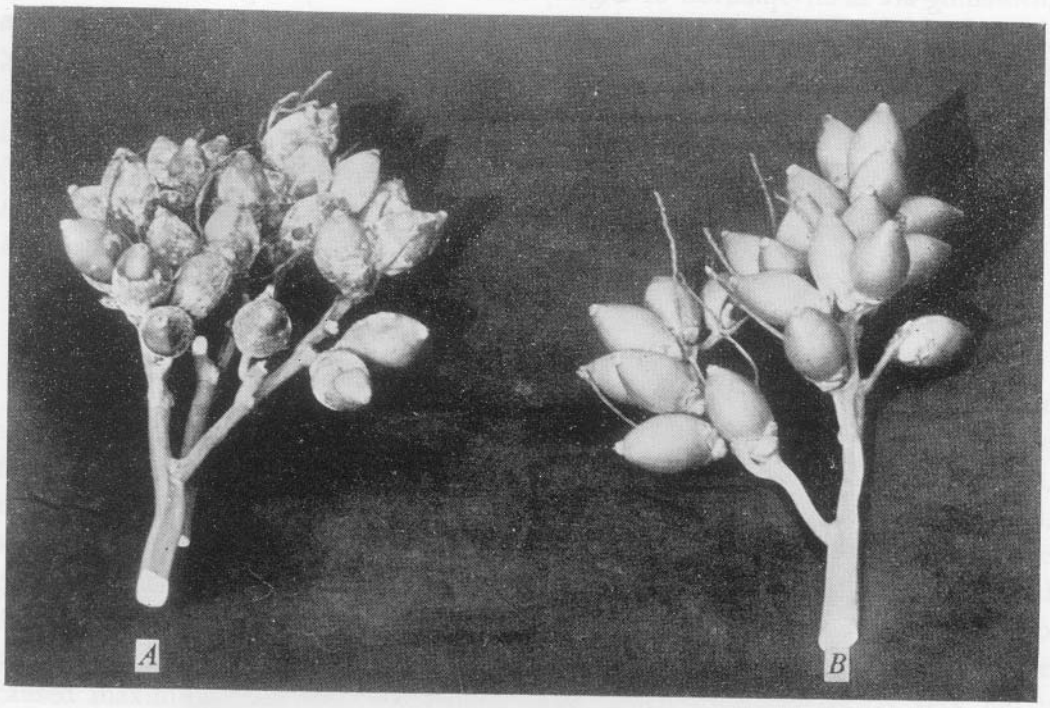

Fig. 7.3 A. Portion of the bunch showing white mycelial growth of *Phytophthora*. B. Portion of a healthy bunch.

ii. *Etiology*

The pathogen was first described as *P. omnivora* De Bary by Sydow and Butler (1907) and as *P. omnivora* var. *arecae* by Coleman (1910). Pethybridge (1913) observed that the fungus was quite different from De Bary's *P. omnivora* and hence named it as *P. arecae* Peth.

The mycelium of the fungus is coenocytic, but sparsely septate in the older stages. It is inter and intra-cellular, occasionally branched with finger like haustoria. The hyphal diameter varies from 8 to 9μ (Coleman, 1910). The fungus grows and sporulates better on steamed corn meal agar (Tucker, 1931).

Asexual reproduction is by production of sporangia and chlamydospores. Sporangiophores are about 2.5μ wide and irregularly sympodial. Sporangia are broadly ellipsoid or obturbinate to nearly spherical, mostly in the range of 40–50μ × 35–40μ, maximum being 70μ × 48μ (l:b ratio=1.1–1.4:1) (Fig.7.4). They are deciduous with a pedicel length of 1–6μ. The papilla and apical thickening are hemi-spherical or slightly less. The chlamydospores are variable in number, sometimes absent or rare and never abundant, the size being 35–40μ in diameter (maximum 60μ) with a wall thickness of 1μ (Tucker, 1931; Waterhouse, 1963; Newhook, Waterhouse and Stamps, 1978).

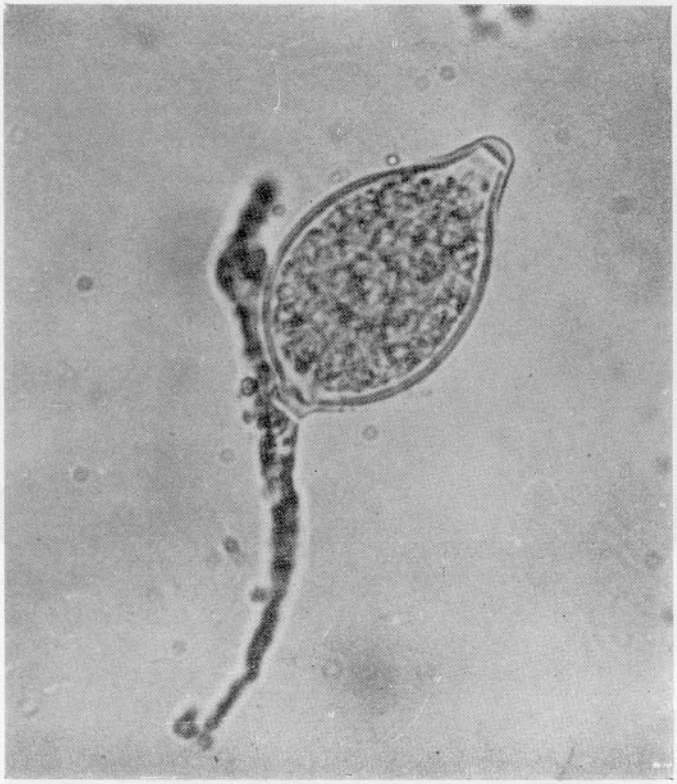

Fig. 7.4 Sporangium of *P. arecae*

Sexual reproduction is oogamous. Oogonia are 30μ in diameter, rarely over 35μ (maximum being 40μ), with smooth wall. Oospore nearly fills the oogonium and have a diameter of 28μ with a 3μ thick wall. Antheridia are always amphigynous, frequently broader than its length, with a size of 14μ × 15μ (Waterhouse, 1963).

Sundararaman and Ramakrishnan (1928) could not observe oospores in nature. This was due to the presence of + and — strains which are localised (Uppal and Desai, 1939). Coleman (1910) observed oospore production on inoculated arecanuts, *Cereus formosus, Clarkia elegans* and Desai (1950) on fresh bean agar. Homothallic nature of the fungus was observed by Narasimhan (cf, Anonymous, 1932) Ramakrishnan (cf, Anonymous, 1954b) and Ramakrishnan and Seethalakshmi (1956), while it was reported as heterothallic by Ashby (1929), Venkatarayan (1932) and Marudarajan (1941). The formation of oospores was observed only in the mixed cultures of *Phytophthora* from arecanut with isolates from coconut, palmyrah and rubber, but not among the isolates from arecanut (Marudarajan, 1941). Oospore formation was also reported in mixed cultures of *P. arecae* and *P. meadii* (Ashby, 1929) and in paired cultures of *P. arecae* and *P. infestans* (Gallegly, 1964).

iii. *Epidemiology*

Rain plays an important role in the initiation and spread of the disease since low temperature and high humidity are favourable for the growth of the fungus (Coleman, 1910). Soon after the South West Monsoon breaks in June, the disease makes its appearance. Heavy rain fall with constant humid condition, wind, low temperature (20–23°C), alternate sunshine and rainfall, favour disease development (Coleman, 1910; Narasimhan, 1922; Kamat, 1956). Reddy and Anandaraj (1980) attempted to correlate the intensity and spread of the disease to the rainfall and temperature for a period of nine years and found that the disease caused maximum crop loss (50.9%) in 1978, when the rainfall was very heavy (5088.6 mm). The intensity of *mahali* is very severe in plantations situated in valleys or those surrounded by thick belts of trees (Kamat, 1953) or with intercrops resulting in high humid conditions.

The zoospores germinate in films of water and penetrate the surface of the nut through stomatal openings. In about four days, the fungal growth emerges out with sporulation. The subsequent spread depends upon the heavy rains and wind, which help the dispersal of the spores through splashing (Coleman, 1910).

The over-summering of the fungus is through resting spores probably present in the diseased parts of the host as well as in the upper layers of the soil (Coleman, 1910). Bud rot affected areca palms remaining in the garden may serve as potent source of primary infection.

iv. *Control*

Attempts to control *mahali* by providing covers to arecanut bunches made of arecanut leaf sheath called *Kotte* in *Malnad* region or covers made of grass called *Karada* in other regions of Karnataka were in vogue, before the scientific plant protection was introduced. These covers neither helped in preventing, nor in eradicating the disease (Coleman, 1910; Anonymous, 1954a; Rao, 1960). Coleman (1910) was the first to recommend spraying 1% Bordeaux mixture with resin and washing soda as adhesive to control the disease. Various workers tested the efficacy of different adhesives and spreaders with Bordeaux mixture with good results (Narasimhan, 1924; Anonymous, 1927). Potash alum with casein called Martin's Bordeaux mixture (Narasimhan, 1928a, 1928b) and vegetable oils from ground nut, gingelly, coconut or safflower (Anonymous, 1932; Thomas and Marudarajan, 1938; Rao, 1960) added to Bordeaux mixture before spraying, protected the palm from *mahali*. However, it was also shown that plain Bordeaux mixture without any adhesive was equally effective in controlling the disease (Thomas and Marudarajan, 1938) and therefore prophylactic spraying with neutral Bordeaux mixture (1:1:100) alone once before the onset of heavy South West Monsoon and a second application 40–45 days after has been recommended. If rain is persistent a third round of application may also be given at the same interval.

Spraying campaign against *mahali* was undertaken in Karnataka on payment basis (Anonymous, 1924) and an improved sprayer called 'Primus' was developed for the purpose (Anonymous, 1938a). A number of other chemicals such as mercurised copper oxychloride and Blitane were tested besides Bordeaux mixture. Spraying the bunches with copper oxychlorides did not check the disease in the field while it produced copper injury symptoms even at 0.5% concentration (Anonymous, 1969). Proprietory copper fungicides sprayed also could not protect the nuts as efficiently as Bordeaux mixture (Table 7.3). It could be seen from the data that the Bordeaux mixture spray gives maximum copper deposit initially, resulting in less disease incidence and maximum retention of copper on the nuts even after 40 days (Anonymous, 1969).

Besides the protective spraying against the disease, it is also necessary to reduce the inoculum potential by adopting phytosanitary measures such as collection and destruction of fallen nuts, removal of diseased bunches, tree tops and other plant parts in the field (Coleman, 1910) and eliminate alternate hosts.

Table 7.3. *Copper deposit on nuts sprayed with different fungicides*

Fungicides	Type of spray	Infection (%)	Copper deposit in μg/ml of washed nut (mean value)	
			1 day after spraying	40 days after spraying
Fycol 8E in water	Low volume	68	7.34	5.59
Fycol 8E in oil	,,	21	5.28	4.80
Oleocap in oil	,,	29	7.48	4.11
Fycol 8 in oil	,,	28	7.36	4.35
Bordeaux mixture	High volume	8	15.52	7.63

3. *Anabe roga* or foot rot

Anabe roga literally means a disease caused by a mushroom. Occurrence of the disease in Karnataka was recorded as early as 1807 by Buchanan (Buchanan, 1807). The same disease was mentioned as betelnut plague from Silhat (Butler, 1906). The *anabe* disease was prevalent in *Maidan* and semi-*Malnad* areas of Karnataka, causing heavy crop loss.

The disease has also been reported from parts of Tamil Nadu especially in Mettupalayam areas, Kerala and Assam (Anonymous, 1960a) Bengal (Sharples, 1928) and Nicobar Islands (Sangal, Mukerji and Singh, 1961).

The disease was observed in severe form in neglected, ill-drained and over crowded gardens (Venkatarayan, 1952). The intensity was more in hard black loamy and acid soils and soils of higher iron and lower calcium contents (Lalithakumari, 1969). Palms over 5-10 years are generally affected (Coleman and Rao, 1918). The disease is primarily soil-borne and spreads secondarily through air-borne spores (Venkatarayan, 1936).

Butler (1906) recorded 94% mortality in a neglected arecanut garden. The death rate of areca palms due to the disease in Karnataka has been estimated from 5% (Venkatarayan, 1936) upto 8% in neglected and water logged gardens (Naidu, Kumar and Sannamarappa, 1966). The incidence of the disease varied from 0.05 to 5.10% in Mettupalayam areas of Tamil Nadu (Anonymous, 1971).

i. *Symptoms*

The slight discolouration of the leaflets in the outer whorl of the leaves is the first observable symptom of the disease. This pale discolouration spreads to the whole leaf and the entire crown becomes progressively yellow with the outer whorl drooping down and covering the stem. Later the leaves in the inner

whorl also become yellow. The development of inflorescence and nuts is arrested. In the advanced stage, the infected palms exhibit the typical symptoms of a pronounced drought. Irrigation of the affected palms do not bring back the palms to normalcy. Subsequently the leaves dry up, droop and fall off leaving the bare stem. The infected brittle stem is easily broken off during heavy wind. The base of the stem shows brown discolouration and oozing of a dark fluid.

The fructification of the fungus (basidiocarp) with the characteristic bracket stage appears at the base of the trunk, and is called *anabe* (Fig. 7.5). The roots of the affected palms are brittle, discoloured and dried (Naidu et al., 1966.)

On cutting open the affected trunk, the infection could be traced upto one meter from the ground level. The central tissues of the affected portion of the

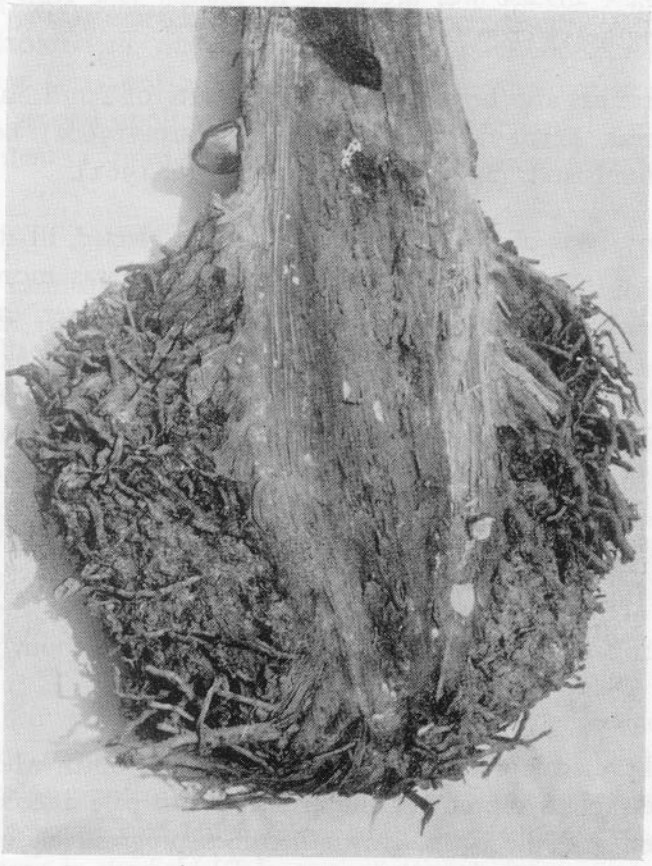

Fig. 7.5 *Anabe* affected stem showing developing sporophores

stem are generally dark brown in colour and emit a musty smell. The xylem and xylem parenchyma are completely broken down due to the invasion of fungus. This impedes the water supply to the top, resulting in pathological drought and death of the infected palm (Venkatarayan, 1936).

ii. *Etiology*

According to Coleman (1911) and Rao (1917) the disease is caused by a fungus *Fomes lucidus*. Venkatarayan (1936) reported that the causal organism as *Ganoderma lucidum* (Leys) Karst. The biology of the fungus was studied in detail by Bose (1930) and Menon (1963b). The fungus is heterothallic and tetrapolar, which reproduces sporophores having three types of spores and gastrospores in addition to chlamydospores (Banerjee and Sarkar, 1958, 1959). Chlamydospores are intercalary and terminal, golden yellow in colour and granular in content measuring $4-8\mu \times 4\mu$ in size (Menon, 1963b).

The hyphae are hyaline, $1-2\mu$ in diameter covered with a deposit of calcium oxalate crystals. Clamp connections occur profusely in older hyphae. The mycelium remains hyaline for about a month when the mats become dotted with drops of colourless fluid containing round, white, thin-walled conidia measuring $14\mu \times 20\mu$ in size. Patches of mycelium turn pale yellow, take up a russet tint very characteristic of the mature sporophores of *Ganoderma* found in nature. Luxuriant growth of the fungus was obtained in malt extract agar. The fungus grows well in a wide range of pH (3-9), the optimum being 4.5-6.5. Maximum mycelial growth of the fungus occurred at soil moisture of 40-80%. Production of fruiting bodies of the fungus was observed on wood pieces of *Mangifera indica* (Bose, 1930) as well as on saw dust medium enriched with 10% malt extract (Nambiar and Nair, 1973). The fungus produced many hydrolytic enzymes in culture (Venkatarayan, 1936).

The fungus has a wide host range infecting coconut (Butler, 1906), oil palm (Sharples, 1928), mango, arecanut, *Delonix regia, Pongamia glabra* and *Casuarina* (Bose, 1930, 1931; Venkatarayan, 1936), *Cassia,* tamarind (Anonymous, 1971) *Phyllanthus* sp. and *Acanthospermum* sp. (Anonymous, 1974). Being a slow growing fungus, *G. lucidum* does not always induce disease incidence on artificial inoculation (Anonymous, 1934).

iii. *Control*

A large number of chemicals have been tried against the disease with varied results. But none of them could eliminate the fungus from the soil or

control the spread of the disease effectively. Though Narasimhan (1940) found sulphur to be effective in controlling the disease, contrary results were obtained by Nair and Rao (1965). Mercurised copper oxychloride (Nair and Rao, 1965), Difolatan, Vitavax and aureofungin sol (Anonymous, 1973) were found to be effective against the pathogen in *in vitro* trials.

It is also known that *Trichoderma* sp., *Bacillus coagulans, Streptomyces* sp. and *Mucor* sp. are antagonistic to *G. lucidum* (Anonymous, 1963a, 1967; Menon, 1963b).

Since the disease is primarily soil–borne, its prevention is considered to be better than cure. Fresh planting of arecanut should be avoided in newly cleared jungle areas containing dead stumps. Fruiting bodies of the fungus and dead stumps of diseased palms should be extracted along with major portion of roots and destroyed by burning (Venkatarayan, 1935; Venkatakrishnaiah, 1956). Improving the drainage condition of the soils, avoiding dense planting of palms and adoption of clean cultivation of gardens help in checking the disease. Planting of susceptible trees such as *Delonix regia, Pongamia glabra, Cassia siamea* etc. in the vicinity of gardens should be avoided. When once the disease appears, its spread to neighbouring palms could be prevented by digging deep trenches all around the affected palm (Anonymous, 1956b).

4. Inflorescence die–back and button shedding

Die–back of inflorescence due to the association of micro–organisms is reported to be associated with the low fruit set in arecanut (Anonymous, 1971). About 60% of the palms in the states of Karnataka and Kerala are infected by this disease causing severe shedding of buttons (Saraswathy, Reddy and Nair, 1977). No systematic survey has been conducted to assess the crop loss caused by this disease.

i. *Symptoms*

Disease appears first on the rachillae of the male flowers, then in the main rachis as brownish patches which soon spread from tip downwards covering the entire rachis causing its wilting. The female flowers of the infected rachis are shed (Fig. 7.6B). The fungus also infects the developing embryo inside the female flowers, which eventually shrivels up showing a brown discolouration. Under severe condition the fungal infection proceeds from tip downwards producing the condition known as die–back (Rao, 1965). Concentric rings of light pink coloured conidial mass of the pathogen appear on the discoloured portions

of the infected inflorescence (Anonymous, 1961). On closer examination, a tuft of mycelial growth of the fungus could be observed on the stigmatic end of the fallen buttons. The disease is present throughout the year, but is most serious during the dry period (February–May).

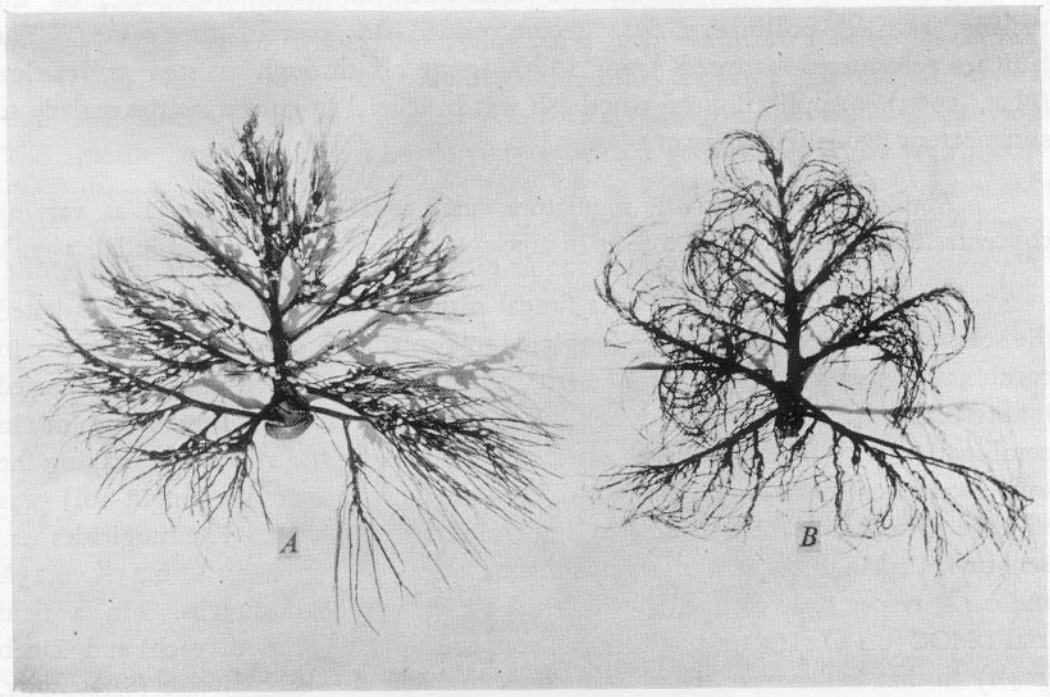

Fig. 7.6 Inflorescence die-back. *A. Healthy inflorescence. B. Inflorescence showing die-back with shrivelled female flowers.*

ii. *Etiology*

Nutritional and physiological factors are reported to be the possible causes of drying of arecanut inflorescence and shedding of buttons. Raghavan and Baruah (1956) were of the view that the shedding of female flowers is due to lack of pollination and fertilisation.

The fungus *Gloeosporium* sp. was associated with the fallen nuts and infected inflorescence (Anonymous, 1938b). The toxin produced by the fungus was considered responsible for the pathological conditions (Menon, 1961b). Saraswathy et al., (1977) could invariably isolate the fungus *Colletotrichum gloeosporioides* Penz. the conidial state of *Glomerella cingulata* (Ston.) Spauld and Shrenk, both from the

fallen buttons and infected inflorescence. The primary pathogenic nature of the fungus was confirmed through inoculation experiments by the production of characteristic symptoms of the disease causing heavy shedding of female flowers.

iii. *Control*

Since lack of pollination was considered as one of the causes of the malady, assisted pollination was recommended to correct the same. The fruit set percentage increased from 12.0% to 26.4% through assisted pollination (Bhat, 1963). Application of wood ash was beneficial in correcting the malady to some extent (Saidalikutty, 1951).

Application of growth regulators such as GA and 2, 4–D at varying concentrations increases the fruit set in arecanut (Yadava, Murthy and Pillai, 1974).

The constant association of a fungal pathogen with the malady necessitated the selection of the most effective fungicide to control the disease. Shell copper in combination with Endrex gave high retention of tender nuts in sprayed inflorescences (Anonymous, 1960a), while 1% Bordeaux mixture alone or in combination with Endrex gave the same result (Anonymous, 1963a). Spraying the inflorescence with Dithane Z–78 and Heptane antibiotic (aureofungin–sol) was effective in reducing the button shedding (Anonymous, 1971). The fungicides *viz.*, Benomyl, Captan, Thiram and phenyl mercury urea *in vitro* at 0.1%, 0.25%, 0.25% and 0.1%, respectively completely inhibited the growth of the fungus. In a field trial DMOC (0.1%), Heptane antibiotic + copper sulphate (50 ppm each) and Zineb (0.4%) in that order were effective in controlling the die–back disease (Saraswathy, Reddy and Nair, 1975).

For controlling this disease, one spraying at the time of opening of female flowers and the second after an interval of 20–25 days are recommended. Removal and burning of infected inflorescences reduce the inoculum and consequently reduce further incidence of the disease.

5. Bud rot

During the course of investigations on *mahali,* Coleman (1910) observed that the same pathogen also affects the spindle of the areca palm causing rotting of the growing bud, which eventually kills the palm. The disease was recorded in a severe form in the heavy rainfall tracts of Karnataka by Nambiar (1949). Though the disease generally occurs in monsoon season, the fresh infection during November onwards becomes severe during succeeding months (Marudarajan, 1950b).

An annual crop loss of 1 per sent or more was recorded by Coleman (1910). However heavy crop loss due to the bud rot disease in endemic areas was also reported (Dorasami, 1956).

i. *Symptoms*

The first symptom of the disease is the discolouration of spindle from the natural light green colour to yellow and then brown. Infection spreads to young leaves which rot rapidly. As the infection spreads inside the bud, the growing point of the stem also rots resulting in the death of the palm. The spindle slumps and can be drawn out with a gentle pull. The outer leaves then become yellow, droop and drop off one by one leaving a bare stem (Fig. 7.7). Secondary organisms colonise the rotting bud and convert it into a slimy mass which would emit a foetid odour (Coleman, 1910).

Fig. 7.7 Bud rot affected crown

ii. *Etiology*

The fungus *Phytophthora arecae* causing the *mahali* disease may pass on to the growing bud from the infected bunches and cause the bud rot. Independent infection of leaf sheaths surrounding the growing point by the pathogen has also been suggested (Coleman, 1910). Rao (1962b) observed rotting of areca spindles from tip downwards caused by *Gloeosporium* sp. A crown rot disease has been observed in a serious form in Assam areas where constant association of fungus *Thielaviopsis paradoxa* was observed (Anonymous, 1970; Sarma and Murthy, 1971).

Naidu and Kumar (1964) recorded *Nigrospora sphaerica* causing severe rotting of young leaves which allows entry of other bud rotting fungi resulting in the death of the palms.

Occurrence of a bacterial rot on spindles of young areca palms was noticed by Naidu (1960) in many areas of Karnataka, characterised by discolouration and drying of heart–leaf from tip downwards. Lightning injury also causes rotting of the bud in areca palms.

iii. *Control*

Infected tissues of bud is to be scooped off and treated with 10% Bordeaux paste. Destruction and removal of dead palms and also bunches affected by *mahali* and drenching crowns of surrounding healthy palms with 1% Bordeaux mixture would help in minimising the incidence of bud rot disease (Nambiar, 1956; Anonymous, 1960a). Drenching the crown with mercuric compounds like 0.2% Ceresan wet or Leytosol helps to bring down the bacterial infection of the crown (Naidu, 1960). Since high humidity and over–crowding predispose the palms to infection, close planting in low lying areas and dense intercropping should be avoided.

6. Bacterial leaf stripe disease

The first report of a bacterial disease on arecanut is that of Orian (1939) who observed natural infection of arecanut by *Xanthomonas vasculorum* (Cobb) Dowson, the incitant of gumming disease in sugarcane. Rao and Mohan (1970) reported the occurrence of the bacterial leaf stripe on arecanut in Tumkur areas of Karnataka state in an endemic form.

i. *Symptoms*

The initial characteristic symptoms of the disease are 1–4 mm wide, dark green water soaked, translucent, linear lesions or stripes alongside and parallel to

the midrib of the leaflet or its other main veins. The lesions may develop at any point on the lamina, but more commonly from the base or towards the tip of the leaflet. The margin of the lesions are usually straight and well–defined, but occasionally it may appear wavy. The lesions are covered with abundant bacterial exudates on the lower surface. The exudate is creamy white and slimy. On drying, it forms a waxy film or creamy white to yellowish flakes or fine granules or irregular yellowish masses. In the advanced stages, the lesions may measure 1 cm or more wide and several centimeters long involving the midrib also. The affected midrib and veins of the leaflet get discoloured and turn black. All the leaflets of a leaf may be affected resulting in complete or partial blighting of the leaf and in severe cases the entire crown may be killed particularly in seedlings (Rao and Mohan, 1970; Kumar, 1981).

ii. *Etiology*

Microscopic examination of the affected leaf tissue shows profuse bacterial streaming throughout the surface of the cut end indicating the parenchymatous nature of the disease (Rao and Mohan, 1970). The organism was isolated in pure culture. On the basis of its cultural and morphological characters, the pathogen was identified as *Xanthomonas arecae* (Rao and Mohan, 1976). The pathogen produced typical symptoms on artificial inoculation on arecanut (Rao and Mohan, 1970; Anonymous 1976). The bacterium caused the development of dark green, water soaked elongated lesions on coconut and other ornamental palms.

The causal bacterium produces large quantities of extra–cellular toxic polysaccharides. The ability to form polysaccharides in phytopathogenic bacteria has been linked to the virulence of the pathogen. In highly susceptible arecanut cultivars, the proliferation of the pathogen results in copious amounts of gum production, chlorosis, localised water soaking and invasion of host cell. The purified extra–cellular polysaccharide produced characteristic symptoms in detached arecanut leaves. The toxin is a heteropolymer of glucose, galactose, mannose and small amount of glucuronic acid (Kumar, 1981).

Most of the phenolic acids were common to both diseased and healthy leaf tissues of arecanut. The diseased leaf tissues contained an extra phenolic acid formed as a result of host pathogen interaction (Kumar, 1981).

iii. *Epidemiology*

The disease remains aggressive during and after the rainy season and it is of little significance during the hot dry summer months. Kumar (1981) obtained

a strong correlation between mean temperature and disease incidence and also observed that the incidence was high during the months of July to October when the average monthly rainfall is 130 mm or more with more than 10 rainy days per month. The organism does not survive in soil for long, indicating that soil may not be a primary source of inoculation.

Three to five year–old palms are highly susceptible to the disease than older palms (Kumar, 1981).

iv. *Control*

Antibiotics like tetracycline and its formulations are effective as prophylactic and curative treatments at 500 ppm concentration. Stem injection of antibiotics has longer residual effect than foliar spray (Kumar, 1981).

7. The *band* or *hidimundige*

The name *band* is given to peculiar diseased condition of arecanut palm reported as a major problem along the Konkan coasts in Maharashtra. The term *band* means barren in Marathi language, as the diseased palm ceases to produce fruits. In Karnataka, the disease is known as *hidimundige*. A similar disease was reported from Sri Lanka known as 'pencil point' and from Australia as 'rosette' disease. According to Joshi and Joshi (1952), the malady was prevalent in Bombay areas even before the year 1889. The disease causes gradual but considerable monetary loss to the growers. Coleman and Rao (1918) attributed the disease to adverse environmental factors.

i. *Crop loss*

Crop loss due to this disease varies from 5 to 25% or more according to locality and time of observation (Joshi and Joshi, 1952). About 50% of the palms in Kolaba and Ratnagiri districts of the Maharashtra are affected by this disease. In general, the plantations on the plains are affected more than those on hill tops (Kibe, Gokhale and Narayana, 1956).

ii. *Symptoms*

The production of smaller leaves is the first symptom. The leaves become dark green in colour, smaller in size and ultimately the crown forms a rosette shape. The leaflets are brittle with wavy margins. The leaves which instead of opening out, in due course tend to remain closely packed round the stem tightly binding the top portion of the palm (Fig. 7.8). This prevents the normal development of the growing point. Sometimes small multiple shoots emerge due to the arrest of normal growth of bud due to the presence of persistent leaf bases of lower leaves.

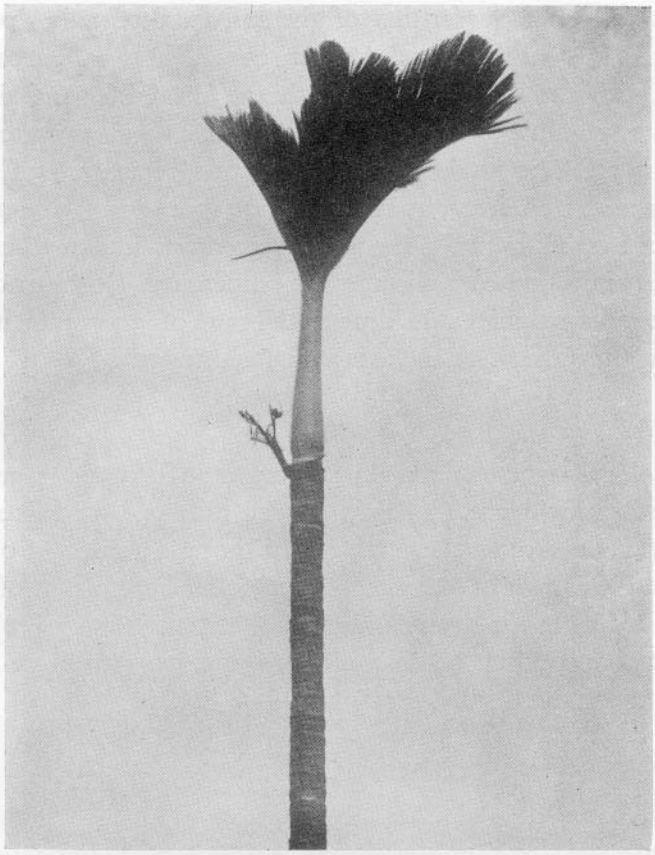

Fig. 7.8 Crown of *band* affected palm

The reduction in internodal length and tapering of the stem towards apex have also been recorded (Patel and Rao, 1958). The affected palms remain mostly unproductive and continue to live for years. The nuts if at all produced are small and malformed. Roots are poorly developed, short, brittle and crinkled.

iii. *Etiology*

The cause of the disease however, is not yet known. No biotic agent is found to be associated with the disease and it does not seem to be contageous. The attribution of this disease to the nematode *(Aphelenchus coccophilus)* infestation (Thirumalachar, 1946) was refuted by Venkatarayan (1946). It has not been possible to transmit the disease from one palm to another (Nayar, 1976).

Poor drainage and low fertility of the soil are reported as possible causes of the disease (Gokhale, Kasaragode and Ajrekar, 1916). Lateritic subsoil pan or hard clayey pan are found to be associated with the disease. Imbalanced nutrition of the palms as a possible cause of the disease has been reported, (Daji, 1948; Joshi and Joshi 1952; Kibe et al., 1956). Comparative analysis of soil samples of healthy and disease affected arecanut areas did not show any significant difference in the contents of major nutrients. The zinc content was low in diseased soils while boron and manganese contents showed no difference (Joshi and Joshi, 1952). Application of copper sulphate and lime at the rate of 30 gm each per palm was effective in controlling the disease (Joshi and Joshi, 1952).

iv. *Control*

Better soil management and improvement of the drainage were found to minimise the disease incidence. Palms in well managed gardens responded more to manurial treatments against the disease than those in neglected ones (Kibe et al., 1956). Removing hard pan of the subsoil and foliar application of micronutrients were effective in reducing the disease intensity (Patel and Rao, 1958). Correction of soil acidity and incorporation of a mixture of copper sulphate and lime to the basal soil could check the disease effectively (Rao, 1960).

8. Sun scorch (Stem breaking)

In many parts of Dakshina Kannada district, the areca growers were reporting breakage of stems in the upper half during heavy winds. A small research unit was established at Vittal, Dakshina Kannada in 1952 with the objective of investigating the factors leading to the incidence of stem breaking disease and to evolve suitable control measures. Detailed observation of the disease in relation to nature of soil, manurial practices, exposure to sun, drainage etc. were made and various prophylactic measures were suggested (Anonymous, 1956a) based on the work carried out under the scheme.

i. *Symptoms*

The initial symptom appears as golden yellow patches on the stem against green background of young stem facing the south west sun. These patches later turn dark brown followed by development of longitudinal cracks of 1–3 cm deep all along the length of stem. The palms standing in the southern and northern borders are severely affected on account of direct exposure to the sun. Saprophytes which harbour the scorched tissue accelerate damage to the stem and the monsoon winds finally break such affected stem (Fig. 7.9).

Fig. 7.9 Stem breaking

ii. *Etiology*

Coleman and Rao (1918) felt that the cracking of arecanut stem was due to exposure to the sun. A number of fungi *viz., Ceratostomella paradoxa, Lenzitus* sp., *Acrothecium* sp., *Polyporus* sp., *Ganoderma lucidum, Polystietus* sp., *Nigrospora* sp., *Pestalotia palmarum* and *Fusarium* sp. were isolated from the affected tissues. Of these, *C. paradoxa, Lenzitus* sp., *G. lucidum* and *Polyporus* sp. could cause infection of stems of young palms when inoculated through wounds caused by sun scorch (Coleman and Rao, 1918; Patel and Rao, 1958). Continued scorching followed by fungal infection weaken the stem and cause its breaking during strong winds (Seshadri and Rawther, 1968). The side of the stem subjected to wide fluctuations in temperature is more prone to sun scorch.

iii. *Control*

Stem breaking can be reduced by trailing pepper vines along the stem, raising rapidly growing trees on the southern and western sides of the gardens (Kurup, 1955) and by protecting the trunk with a cover of dry leaves (Anonymous, 1956a). Reinforcing the cracked portions with split areca stem renders mechanical support to the weak trunk (Anonymous, 1956a). To minimise the incidence of this malady a suitable alignment of the planting was suggested by Bhat (1965).

9. Stem bleeding

Nambiar (1949, 1951) reported the occurrence of stem bleeding disease in isolated pockets of Mettupalayam areas in Tamil Nadu. The disease is of rare occurrence in South India and closely resembles the stem bleeding disease of coconut (Anonymous, 1953b; Sundararaman, Nair and Ramakrishnan, 1928). Though both young and older palms are affected, the young palms are more susceptible (Patel and Rao, 1958).

i. *Symptoms*

Symptoms appear on the basal portions of the stem as small discoloured depressions during initial stages. Later these spots coalesce and cracks develop on the stem. With the progress of the disease, the fibrous layer disintegrates which eventually hollows upto varying depths along the infected portion. Crowns of affected adult palms get reduced in size followed by reduction in yield. Finally a dark brown liquid oozes out from the cracks (Sundararaman et al., 1928).

ii. *Etiology*

The fungus *Thielaviopsis paradoxa* is associated with the disease (Sundararaman et al., 1928).

iii. *Control*

Since the disease is serious in areas with poor drainage (Varadarajan, 1958), improving the drainage may help in minimising the disease incidence. Application of hot 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.

10. Nut splitting

This is considered to be a physiological disorder rather than a pathological disease. Nambiar (1949) reported nut splitting characterised by the

cracking of fruits known as *anduadakke roga* in Karnataka. The disease is known as *achikeeral* in Ponnani in Kerala. This abnormality is seen in patches in individual gardens and are common in young palms.

i. *Symptoms*

Symptoms appear as premature yellowing of the nuts where they are half to three fourth mature. This is followed by cracks at the tips which extend longitudinally towards the calyx exposing the kernel (Fig. 7.10). Kernel also exhibits cracking and as a result becomes malformed. Splitting may begin at the calyx end also proceeding half–way to the apex or the cracking may be restricted to only one end of the nut (Bhat, 1961). All the nuts in a bunch may succumb to the disease. It is also not uncommon to see tender nuts with informal splitting in the husk without showing any sign of external fissures, resulting in nut fall. The disease may occur in the same palm year after year. Palms at the age group of 12–25 years are more susceptible.

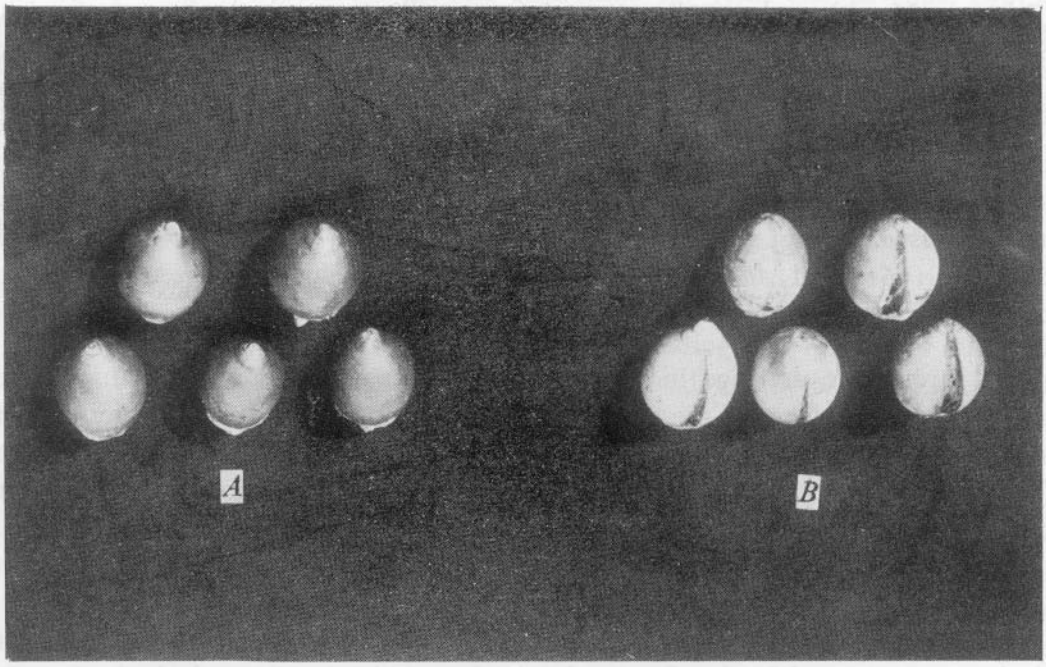

Fig. 7.10 Nuts splitting. *A. Healthy nuts. B. Damaged nuts.*

ii. *Etiology*

The possible causes suggested are hyper nutrition or sudden flush of water after a period of drought or insufficient moisture which upsets the rhythm of development of pericarp and the tissue inside. According to Bhat (1961) application of potash could reduce nut splitting indicating potassium deficiency.

iii. *Control*

The splitting can be reduced by making longitudinal side slits at the base of inflorescence (Bavappa and Sahadevan, 1952; Patel and Rao, 1958). Improving the drainage in gardens having water stagnations or ill-drained conditions may help to minimise the disease incidence. Spraying borax @ 2g per litre of water on the bunches during early stage of disease incidence and application of potash at the base reduce splitting.

II. Diseases affecting the seedlings

1. Yellow leaf spot

Seedlings (1–2½ year old) exposed to the sun are susceptible to this disease (Anonymous, 1961; Rao, 1962a). The disease is severe during summer months (February–March) and continues to infect seedlings until the onset of rains. Symptoms appear on the lamina of the leaf as yellow specks measuring 3–10 mm in diameter. These spots coalesce to form larger lesions surrounded by yellow haloes. Minute brown pin head like structures appear at the centre of the lesions. Infection leads to stunted growth and in severe cases death of the seedlings (Anonymous, 1953a). Menon (1962) isolated *Curvularia* sp. from the affected tissue. Fungi such as *Colletotrichum, Phyllosticta, Helminthosporium* (Rao and Bavappa, 1961) and *Alternaria tenuis* (Agnihotri, 1963) have also been reported to cause leaf spot resulting in stunted growth of seedlings.

Improving drainage in the nursery and main field and providing shade minimise the incidence of the disease. Application of heavy dose of manures and spraying with Dithane Z–78 or 1% Bordeaux mixture reduce the disease incidence (Menon, 1962; Rao, 1962a). Fungicides like Ziram, mercurised copper oxychlorides were also found to be effective in checking the spread of the disease.

2. Leaf blight

The disease is characterised by reddish brown discoloured spots that blight the lamina. Poor soil fertility favours the incidence. Menon, Nair and Abraham, 1962 suggested application of nitrogen and potash to the plants followed by spraying

with Dithane Z–78 to check the disease. The fungus *Pestalotia palmarum* Cooke has been associated with the blight. Roy (1965) reported *Phomopsis palmicola* (Wint) Sacc. *arecae* as the causal organism of leaf blight in seedlings at the transplanting stage. This also leads to stunted growth.

Menon (1959b) reported a seedling blight caused by a pycnidial fungus and suggested shading and spraying with copper fungicides.

3. Red rust

This disease is caused by an algal parasite, *Cephaleuros* sp. which infects the stem and foliage. Circular spots with sunken centres and yellow haloes appear on the foliage. Lesions are irregular on the stem. Infection destroys the epidermis (Paily and Menon, 1960). This alga can be controlled by providing good shade and spraying with Bordeaux mixture (Westcott, 1966).

4. Root/collar rot

This is usually seen in nurseries with poor drainage. The rotting is caused by fungi like *Fusarium* sp. and *Rhizoctonia* sp. (Rao and Bavappa, 1961). The fungi infect roots and cause wilting of seedlings. Sometimes bacteria enter the stem through collar region and cause rotting of bud also. The severity of the disease can be minimised by providing good drainage in the nursery and drenching the soil with Ceresan wet or Bordeaux mixture or cheshunt compound.

III. Post harvest deterioration of arecanut

1. Nature of damage

Lack of proper drying yard, improper spreading and turning of nuts and exposure to unexpected rains during drying period lead to microbial infestation of husk as well as kernel. The infection affects the quality of nuts, renders it unsuitable for consumption and lowers their market value.

The invading fungi first attack the embryo and then spread to the central white core (Jaleel and Govindarajan, 1969; Nambiar, Edison and Nair, 1971). In advanced stages of infection the kernel will present a hollow cavity due to complete disintegration of the tissue by the invading fungi (Fig. 7.11). The affected nuts when cut open, show discoloured tissues of the white core, the colour being dependent on the fungi involved (Nambiar et al., 1971).

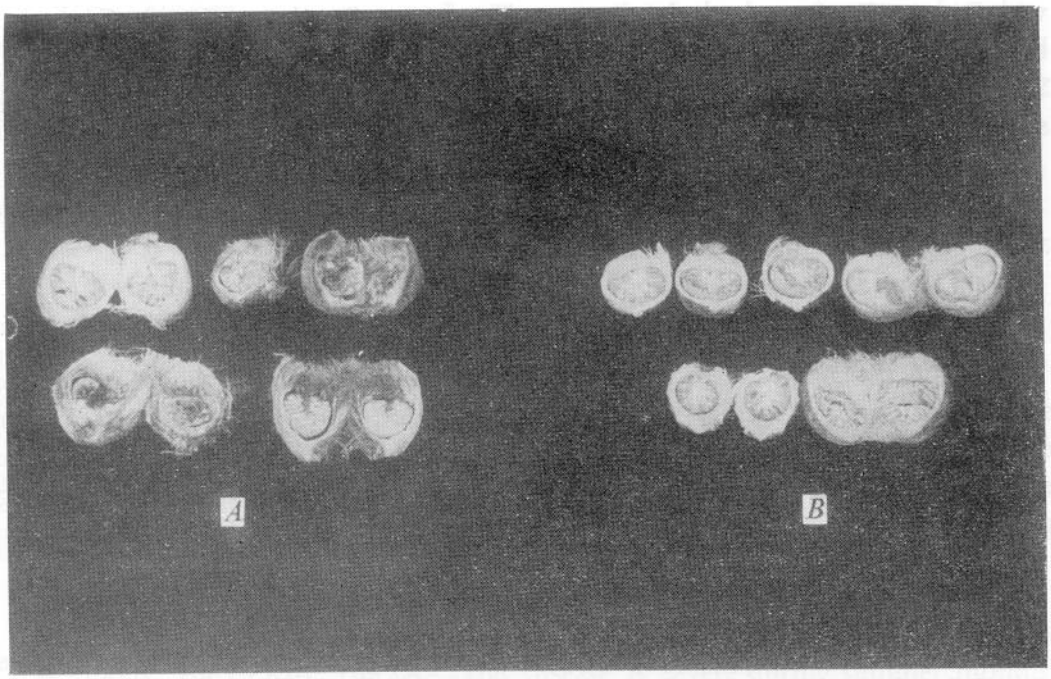

Fig. 7.11 Fungal infection of stored nuts. *A. Infected nuts. B. Healthy nuts.*

2. Loss

The extent of damage due to fungi and other biological agents aiding deterioration depends upon the nature and season of drying. When nuts are stored for one year, the infection increased from 33.7% to 60.7% (Anonymous, 1971). The infection of the kernel will be either mild, moderate or severe according to the duration of storage and fungi involved. Nambiar et al., (1971) found that the percentage of fungal infection was the highest in nuts dried in October (62%) and the lowest in February (21%).

3. Factors associated with deterioration

The increased fungal infection during October is attributed to the prevailing low temperature coupled with rains (157 mm) and consequent high relative humidity (upto 92%) which are congenial for the growth of fungi. The nuts also do not dry quickly under such conditions. The low percentage of infection in February is due to higher temperature and low relative humidity (Table 7.4; Nambiar et al., 1971).

Table 7.4. *Mean percentage of fungal infection in processed arecanuts during different months*

Months	Infection (%)	Range of temperature		Relative humidity (%)	Total rainfall (mm)
		Maximum	Minimum		
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	46.9–87.7	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

Studies on the extent of infection during different stages of drying showed that majority of infection in nuts occurred during the first 5–10 days, presumably from husk. The slow drying of endosperm coupled with its high nutrient content encourage the fungi to penetrate inside and attack the kernel (Nambiar et al., 1971).

4. Fungi associated with deterioration

A normal and unblemished areca fruit is generally considered to be free from any incipient fungal infection. Moreover, when the nuts were harvested by eliminating soil contact and dried in hot air oven at 65°C for 63 hr the kernel was found free of fungal infection. On the other hand, when the harvested bunches and nuts were collected from the ground and the nuts dried in the sun, the infection was about 60 per cent indicating that fungal infection is from the soil either during harvesting or during drying (Nambiar and Nair, 1970).

Usually the microflora associated with the husk and kernel are *Aspergillus* sp., *Diplodia* sp., *Fusarium* sp., *Mucor* sp., *Penicillium* sp., *Thielaviopsis* sp. and certain aerobic bacteria (Anonymous, 1961, 1962), *Cladosporium* sp. (Anonymous, 1970), *Phomopsis heteronema* (Butler and Bisby, 1931) and *Colletotrichum gloeosporioides* (Saraswathy et al., 1977). The fungi found in stored arecanuts are *A. niger arecae* (Lal and Chandra, 1953), *Subramanella arecae* (Srivastava, Banu and Govindarajan, 1962) and *A. chevalieri* (Anonymous, 1971). Fungi associated with spoilage of dried arecanuts with their relative intensities are given in Table 7.5 (Nambiar et al., 1971).

Fungal infection of stored nut was maximum in 'Sweet areca' among different cultivars probably due to the larger endosperm which is a good substratum to the fungi (Anonymous, 1971).

Table 7.5. *Fungi involved in the spoilage of stored arecanuts*

Fungi	Colour of infected kernel	Infection percentage
Aspergillus niger	Black	6.4
A. chevalieri	Yellow	
A. flavus	Yellowish green	
A. fumigatus	Velvetty green	
Penicillium sp.	Felty 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
Total		29.7

5. Control

Elimination of soil contact by the harvested nuts is beneficial in reducing nut infection since it is the prime source of infection. Harvested nuts treated with Blitox showed less infection (Anonymous, 1962). Steeping the nuts in Bordeaux mixture followed by drying in cement floor reduced the percentage of infection significantly. When nuts were harvested without soil contact and dried in hot air oven at 65.0°C for 63 hr, there was no infection (Nambiar et al., 1971). Nuts harvested by the conventional method and dried in mechanical drier (Nambudiri, Govindarajan and Subramanian, 1963) at 62°C for 72 hr contracted 3.6 per cent infection (Nambiar et al., 1971). Nuts dried on cement floor had only 5 per cent fungal infection and the time required for drying was also less in this case (Anonymous, 1972). Polythene lined gunny bags can be used with advantage over plain gunny bags for storing nuts (Nambiar et al., 1971). Arecanuts stored in air-tight bins had only 17.7 per cent infection as against 32.3 per cent infection in nuts stored in gunny bags. Storing nuts in polythene lined gunny bags also reduces fungal infection (Nambiar et al., 1971)

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