Fig. 41: Blight affected *B. nutans* clump in a village grove at Cuttack, Orissa, India

Fig. 42: A severely affected clump of *B. nutans;* note the epicormic shoot from the nodes of partially dead culm

Various fungal organisms have been found associated with blighted culms. However, pathogenic connection between a fungus, or a group of fungi, and the blight disease has not been adequately demonstrated (Boa 1987b).

However, ***Sarocladium oryzae*** is considered to be the principal fungus associated with bamboo blight. The fungus perennates in the affected culms, bamboo debris or in paddy which act as the source of inoculum. The fungus gets inside the rhizome system before blight is established (Boa and Rahman 1987). The spores of ***S. oryzae*** germinate easily in water within a few hours and infect new culms or culm sheaths (Jamaluddin et al. 1992). Water accumulation in debris, weeds or shrubs around the culms enhances the susceptibility of the culms to infection. In India, development of the disease is related to climatic conditions of the area. High humidity owing to canal irrigation and high temperatures during monsoon favour infection. Poor stand management, conducive climatic and soil factors, and insect attack are responsible for the development of the disease (Boa and Rahman 1987). Mining insects are suspected to help in spreading the disease. The spread of infection from one area to another is rather slow.

CONTROL

Silvicultural measures recommended for controlling the disease include: cutting and removing blighted bamboo culms, burning the debris of clumps *in situ*

and addition of new soil to clumps. Light surface fire (controlled burning) before the onset of monsoon is suggested for reducing the inoculum potential of the pathogen in the debris or in the top few centimetres of soil. Weeds and bushes around the clump should be removed as these act as retainers of moisture, which is considered favourable for causing infection. Offsets for planting out should not be taken from blighted clumps. Soil or debris from infected clumps or nearby areas should not be transferred to healthy clumps. Application of Carbendazim combined with Mancozeb (Carbendazim 0.15% a.i. + Mancozeb 0.3% a.i.) or with Fytolan (Carbendazim 0.25% a.i. + Fytolan 0.3% a.i.) is recommended. Drenching the soil around the bamboo clumps with copper oxychloride and Mancozeb is also desirable to check the disease (Rahman 1988).

Top Blight

The disease, also called 'withered tip disease' and 'die-back', has been reported in *Phyllostachys pubescen/ heterocycla* including *edulis* in China (Shi et al. 1979; Deng and Yu 1980; Zhang 1982; Liu and Pan 1983; Yu 1981, 1986; Lin 1988; Xu et al. 1989; Lin and Qiu 1993; Zhang and Ou 1993; Ou and Zhang 1993a,b). The disease is widespread in Jiangsu, Zhejiang, Anhui, Jiangxi, Fujian and Shanghai Provinces, causing heavy damages to the bamboo groves. Peak incidence of disease occurs in July-September. Bamboo groves located in drought affected areas, at forest borders, on hill tops and on poor soils with poor growth have been seriously damaged. The disease has been studied in detail and a disease prediction model has been generated for forecasting the infection (Ou and Zhang 1993a). The disease distribution pattern over time is that of a polyetic epidemic disease

(monocyclic disease) and the disease index increases more often 2.5 to 3.4 times in accordance with the logistic model; the spatial distribution pattern of the causal agent obeys the law of aggregation distribution, with an obvious infection centre and the disease spreading repeatedly from that centre to a distance of about 5-10 m (Ou and Zhang 1993b).

SYMPTOMS

The disease affects mainly new culms of the current season. Infection causes browning and necrosis of the culm and branch internodes, and subsequent withering of branches, minor branches and whole culms. Accordingly, depending on the plant parts affected and the degree of damage caused, the symptoms manifest as branch withering, minor branch withering and plant withering (Fig. 43). Lightly infected groves belong to branch-withered type, while heavily infected groves suffer from all three infection types. The causal fungus produces its fructifications on the necrotic tissues. Severe infection occurs during August-September and subsides after October. Die-back of the affected culms occurs from infection in successive years.

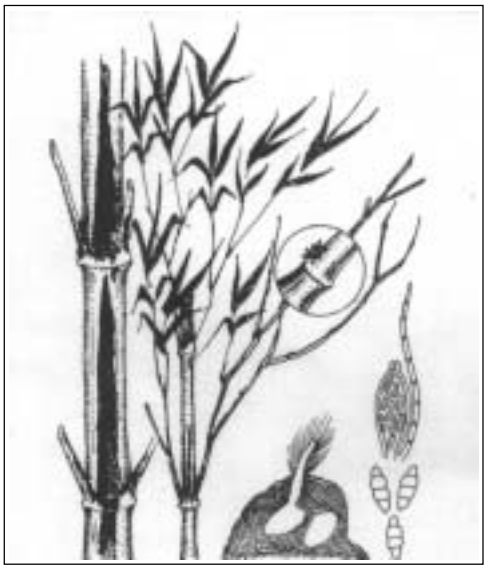

Fig. 43: Top blight of *Phyllostachys* sp. caused by ***Ceratosphaeria phyllostachydis*** (source: Anonymous 1982)

CAUSAL ORGANISM

Ceratosphaeria phyllostachydis Zhang (Zhang 1982; Xu et al. 1989).

ETIOLOGY

Fungal spores, extruded from the perithecia of diseased bamboo tissues, are disseminated by wind or rain splashes. The spores germinate on the host surface and invade the tissues during May through natural

openings or wounds at the branch fork of new shoots. The latent period of infection is about 30-90 days. High atmospheric humidity and temperatures in the range of 22-25°C are favourable for the fast spread of the infection. The infection spreads and causes necrosis, followed by the withering of affected plant parts. The hyphal structures of the fungus overwinter in the infected tissues and can be alive for 3-5 years. The fructifications of the pathogen are produced in the infected tissues during April. Factors responsible for the high incidence and severity of the disease include fast spread of the pathogen, poor resistance of the bamboos against the disease and conducive environment for the development of the disease.

CONTROL

Silvicultural measures suggested for controlling the disease are cutting and removal of infected branches and culms in order to reduce the inoculum, clearing miscellaneous trees nearby and tending young bamboos to eradicate alternate hosts of the pathogen. Adoption of cultural measures in bamboo groves reduced the disease incidence to 20-70% (Xu et al. 1989). Quarantine measures suggested include preventing the transport of diseased bamboos out of the infected areas. Application of fungicides — Carbendazim (0.05% a.i.), Thiophanate methyl (0.02% a.i.) or Bordeaux mixture (1:100) — during May-June is also desirable. The fungicidal spray should be given 2-3 times successively once every 10 days.

Branch Die-back

Branch die-back has been reported in bamboo natural stands and plantations in Kerala, India (Mohanan 1994a,b). The disease was widespread

in *Bambusa bambos, B. vulgaris* and *Dendrocalamus strictus* plantations and *B. bambos* natural stands; percent disease incidence was higher in plantations (14-67%) than in natural stands (2-37%). In plantations, culms produced by 2 to 3-year-old clumps were severely affected. The disease was recorded during September-October; severe infection occurred during December-January, causing die-back of branches and culm tips, and premature defoliation. A similar disease in *Phyllostachys* sp., causing foliage blight and branch die-back, has been reported also from Yongtai County, Fujian Province, China (Kuai 1987).

SYMPTOMS

Infection occurs on branches and top three to five internodes of young culm in the form of small, greyish magenta, linear lesions which later develop into necrotic streaks (Fig. 44). Foliage infection appears as pale yellowish, linear lesions. They later spread to the entire leaf lamina, resulting in leaf necrosis, withering and premature defoliation. The necrotic streaks on branches and culm internodes coalesce to form large streaks; the affected branches and often the entire length of apical three to five culm internodes become discoloured. As the leaves of infected branches wither and defoliate prematurely, discolouration and necrosis on the branches and culm tip become very prominent. Infection spreads from branches to culm nodes, and from there downwards to internodes. Under high humidity, the causal fungus

Fig. 44: Branch die-back in *B. bambos* caused by ***F. pallidoroseum***

Fig. 45: Branch necrosis and premature defoliation in *B. bambos*

sporulates on the infected necrotic areas of culm internodes and branches. Infection causes premature defoliation and die-back of branches and culm tip (Fig. 45).

CAUSAL ORGANISMS

Fusarium pallidoroseum (Cooke) Sacc. (Mohanan 1994a,b) and ***Fusarium*** sp. (Kuai 1987).

ETIOLOGY

Fusarium pallidoroseum is an air-borne pathogen, and the conidia dispersed by wind serve as the primary source of inoculum. The fungus invades the host tissue through natural openings like stomata and lenticels, or through wounds; the fungus proliferates in the leaf and stem tissues and causes necrosis of the invaded tissues. Under warm-humid conditions, the fungus sporulates on the affected tissues and these conidia serve as the source of secondary infection. In young plantations, infection in successive years may affect culm vigour and production.

CONTROL

Aerial application of the fungicide Mancozeb (0.2% a.i.) is suggested for controlling the disease in young bamboo stands.

Witches'-broom

Witches'-broom disease has been reported on different species of bamboos in China, India, Indonesia, Japan, Taiwan-China and Vietnam. The disease has been recorded on a cultivar of *Phyllostachys viridis*, *P. glauca*

McCl. and other *Phyllostachys* species in China (Lin and Wu 1987; Zhu and Huang 1988, 1992; Zhu 1989a,b). Recently, the disease has been reported on *Phyllostachys pubescens*/*heterocycla*/*edulis, P. praecox* Chu et Chao, *P. nuda* McCl., *P. aurea* Carr. ex A. & C. Riv., *P. arcana* McCl., *P. nidularia* Munro and *Bambusa multiplex* (Lour.) Raeusch. ex Schultes in Nanjing and Yixing in China (Mohanan 1995, unpublished observation). The disease is widespread in Hunan Province, causing 95-100% infection (Lin and Wu 1987).

In India, witches'-broom disease has been recorded on *Ochlandra travancorica, O. travancorica* var. *hirsuta, O. scriptoria* and *O. ebracteata* Raizada et Chatterji in the southern states (Mohanan 1990, 1994a,b). A large-scale malformation in *D. strictus* has also been reported from Ranchi Forest Division, Bihar State. The deformed culms developed witches'-broom on the nodes. Out of the 1 498 culms in 124 clumps observed, 655 (43.7%) were affected with witches'-broom. Two types of the disease were recorded. Deformity in a large number of the affected culms was due to certain physiogenic factors that kill the tip of the shoots without causing major die-back. Only 37 culms (2.4%) showed the brooming symptoms characteristic of that caused by fungi, insects, viruses, mites, etc. However, no causal agents could be detected from the affected tissues (Bakshi et al. 1972). In southern India, witches'-broom is widespread in reed bamboo areas, and about 6-15% disease incidence was recorded during 1988-92 (Mohanan 1994b). The infection was also observed on a grass, *Pennisetum polystachyon* (L.) Schultes, growing in the vicinity of the affected bamboos.

The disease has been reported on *Gigantochloa apus* Kurz, *G. atter* (Hassk.) Kurz and *G. robusta* Kurz (=*G.*

verticillata Munro) from Indonesia. In Japan, the disease has been recorded on *P. bambusoides* Sieb. et Zucc., *P. nigra* var. *henonis* Stapf. ex Rendle, *Sasa borealis* var. *purpurascens, S. kurilensis* (Rupr.) Makino et Shibata, *S. paniculata* Makino et Shibata, *S. tectoria* Makino ex Koidz and *S. veitchii* var. *tyugokensis* (Makino) Muroi (Shinohara 1965; Nozu and Yamamoto 1972; Nonaka 1989; Zhu 1989).

Witches'-broom has been recorded in *P. aurea, P. bambusoides, P. lithophila* Hayata*, P. makinoi* Hayata*, P. nigra* var. *henonis, P. pubescens, P. lithocarpa, P. nuda B. multiplex, B. oldhamii* and *B. dolichoclada* in Taiwan-China (Chen 1970, 1971; Kao and Leu 1976; Lin et al. 1981). In Vietnam, the disease has been observed in *B. nutans* and *Dendrocalamus membranaceus* (Mao 1993).

Fig. 46: Witches'-broom in *P. praecox* caused by ***Balansia take***

SYMPTOMS

Numerous highly shortened shoots develop at the nodes of mature bamboo culms. These abnormal shoots do not develop into normal branches and produce only highly reduced shoots successively from their nodes. The culm sheaths which cover the internodes also become shortened in size and become boat-shaped, often with a prominent ligule. The internodes show purplish pink discolouration with reduced pale green foliage. Successive development of a large number of thin shoots in tuft from the nodes of the infected culms give rise to the characteristic appearance of witches'-broom (Fig. 46).

Fig. 47: Witches'-broom in *O. travancorica* caused by ***B. linearis***

Fig. 48: Diseased *O. travancorica* clump; note the diseased wiry shoots produced from the rhizome

New shoots emerging from the rhizome during the growing season also show pronounced brooming symptoms. From an infected rhizome, a large number (often ranging from 30 to 800+) of abnormal, greatly shortened shoots are developed. The shoots grow only up to 10-50 cm in height, showing typical symptoms of the disease (Figs. 47-49).

Shining black fungal fructifications develop on the affected shoots after 5-6 months of infection (Figs. 50-52). Often, one or two normal culms also develop from the infected rhizome and give rise to apparently healthy branches and foliage. Possibly owing to these apparently healthy culms, the diseased clump is not killed outright.

CAUSAL ORGANISMS

Fungi associated with the witches'-broom include: ***Balansia take*** (Miyake) Hara (Zhu 1989a,b); ***B. linearis*** (Rehm.) Diehl (Mohanan 1994a,b); ***B. take*** and a bacteria-like organism (Lin and Wu 1987); ***Aciculosporium take*** Miyake (= conidial state of ***B. take***); ***Loculistroma bambusae*** (Chen 1971; Shinohara 1965; Nozu and Yamamoto 1972; Kao and Leu 1976); ***Epichloe bambusae*** Pat.;

Fig. 49: *P. viridis* affected by witches'-broom; note the wiry shoots produced from the rhizome

Fig. 50: Ascocarps of ***B. linearis*** developed on the affected shoots of *O. travancorica*

E. sasae Hara (Zhu 1989). A phytoplasma was also detected on the diseased tissues (Zhu and Huang 1992).

ETIOLOGY

Macroconidia and ascospores produced in large quantity in the affected host tissues are suspected to be sources of infection in natural stands; rhizomorphs, which overwinters on the affected bamboo shoots, also serve as an infection source. The spores germinate on culm internodes and penetrate the host tissue. A shining white fungal mycelial weft appears on the infected shoot, culm sheaths and foliage. The distal end of the abnormal shoot, as well as the shoot developed immediately from each node of the abnormal shoot, become modified into structures bearing fungal fructification. White, powdery fungal stroma develop at the base of the nodes and spread to the proximal end, later developing into greyish white to pale yellow, uniformly raised ascomata. The ascomatal stroma extend from the base of the nodes to the distal end, except for 1-2 cm at the terminal portion.

As the development of the fructification progresses, the whole structure turns shining brown or greyish brown, with a white basal portion. At this stage, the shoot portion bearing the developing fungal fructification becomes free from the culm sheath and forms an

Fig. 51: Ascocarps of ***B. take*** on *P. viridis*

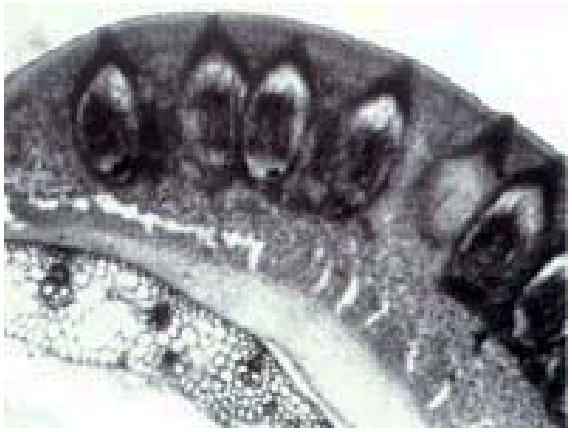

Fig. 52: Cross section of ***B. linearis*** fructification showing asci

inwards curved, sickle-shaped structure. The fructification further matures and turns shining black in colour. Development of the fructification starts during September-October and usually matures during January-February. After the discharge of the ascospores, the fructification degenerates during May-June. As new shoots are produced successively from the infected abnormal shoots, the disease spreads to the new shoots. The fungus also produces long, hair-like, black rhizomorphs on the affected withered shoots, foliage and culm sheaths during the dry period. Infection now spreads to the rhizomes and infected rhizomes give rise to a large number of slender wiry shoots. The pathogen overwinters in infected shoots.

CONTROL

Silvicultural measures, such as pruning and removal of infected minor branches, have been suggested by Zhu and Huang (1988). Fungicides, and antibiotics such as tetracycline and oxytetracycline, have also been screened against ***Balansia take*** in China. However, no chemical treatment is suggested against the witches'-broom of bamboos. Improving the ventilation in the stands, cutting old and weak culms, and clearing diseased clumps and burning them outside the forest have been suggested for disease management in *Phyllostachys* in Japan and in reed bamboos in India (Nonaka 1989; Mohanan 1994a,b).

Little Leaf Disease

Little leaf disease of bamboo has been reported from India (Mohanan 1990, 1994a,b,c, 1995b,c) on *Dendrocalamus strictus* clumps in natural stands in Kerala, Karnataka, Tamil Nadu and Andhra Pradesh. In Kerala, very high incidence (90%) of the disease was recorded in dry tracts and sandal reserves (Mohanan 1994a,b). *D. strictus* had earlier been reported as a collateral host of sandal spike (Nayar and Ananthapadmanabha 1977). Clump-to-clump spread of infection was found to be slow and an increase of 2-13% disease incidence over a period of four years was observed in different areas in the State. Recently, a similar disease has been recorded in *Bambusa multiplex* stands in Kanchanburi, Thailand (Mohanan 1995, unpublished observation).

SYMPTOMS

The disease is characterized by the development of numerous, highly reduced, abnormal bushy shoots from the nodes of newly emerged culms and culm branches. Foliage develops from these shoots, showing prominent size reduction and needle-like appearance. Profuse development of such abnormal shoots from each node of the developing culm and their subsequent growth give rise to a massive bushy structure around each node. The disease also affects culm elongation; infected culms show stunted growth, and curve inwards mainly from the weight of the abnormal bushy shoots at the nodes. Healthy looking, straight-growing culms are also produced from low to

Fig. 53: *D. strictus* shoots affected with little leaf disease

Fig. 54: *B. multiplex* culms affected with little leaf disease

Fig. 55: Development of abnormal bushy shoots in *D. strictus*

moderately infected clumps. In this case, development of abnormal shoots occurs from the culm branch nodes (Figs. 53, 54).

Even though culm growth is completed within six months of emergence, the abnormal shoots continue to develop from culm nodes and branch nodes year after year, and form a massive structure of highly reduced and branched nodal shoots (Fig. 55).

In severely affected clumps, all the culms produced from the rhizome in a growing season become infected. The whole clump becomes bushy with a only few diseased and highly deformed culms.

CAUSAL ORGANISM

Association of a mycoplasma-like organism (MLO) with the diseased tissue was proved by Dienes' staining (Fig. 56), fluorescence microscopy using aniline blue staining (Fig. 57), electron microscopy (Fig. 58) and tetracycline hydrochloride therapy (Mohanan 1994a,b). Attempts to culture MLOs associated with bamboo little leaf were unsuccessful (Mohanan 1994c).

ETIOLOGY

Etiology of little leaf disease of bamboo caused by MLO is not known. However, etiology of MLOs causing

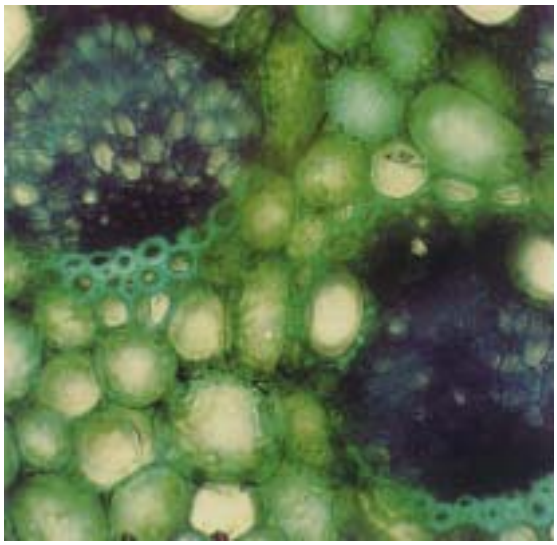

Fig. 56: Diene's staining reaction of diseased culm internodal tissues (870x); note the deep blue staining in phloem tissue

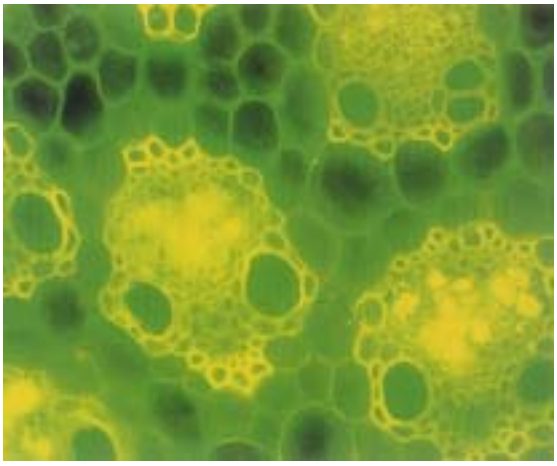

Fig. 57: Aniline blue staining reaction of diseased internodal tissues; note the yellow green fluorescent spots in the phloem tissue

similar diseases in other hosts is available. MLOs are transmitted through insect vectors — aphids, leaf hoppers, etc. — during the process of sap sucking. Incubation period varies depending on the MLOs associated and the vectors.

CONTROL

Antibiotic treatment to control plant diseases associated with MLO has been attempted after the discovery of the suppressive effect of tetracycline antibiotics on symptom development. Since, in bamboos, the process of culm production, elongation and development are completed within six months, and after that only a biological consolidation takes place, it is not worthwhile to control the disease of emerged culms by chemicals or antibiotics.

Bamboo Mosaic

Mosaic disease affecting foliage and young developing culms of bamboo has been reported from Taiwan-China. The disease affects two major cultivated bamboos — *Dendrocalamus latiflorus* Munro and *Bambusa oldhamii* Munro — and about 70-80% disease incidence has been reported (Lin et al. 1977, 1979, 1993; Chen 1985; Lin and Chen 1991).

SYMPTOMS

Infection causes mosaic on leaves and brown

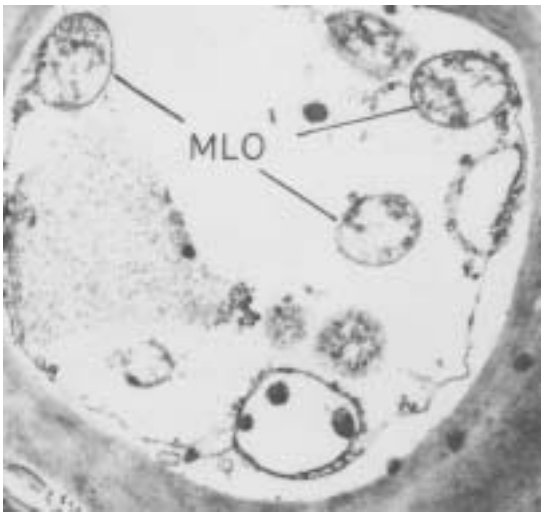

Fig. 58: Transmission electron micrograph of diseased tissue showing MLOs in the phloem sieve cells (19 200x)

internal streaking in emerging and growing shoots. Infected culms exhibit poor growth and development and their internodes become shortened in length. The emerging shoots show a hard texture and are of low quality for eating and canning.

CAUSAL ORGANISM

Bamboo mosaic virus (BoMV) belongs to the potex virus group (Lin et al. 1993). BoMV is a flexuous, rod-shaped virus about 480-500 nm in length.

ETIOLOGY

BoMv has no insect vectors and is likely to be transmitted mechanically, as well as through vegetative planting materials. The infection process and development are the same as in the case of other virus transmitted diseases. The virus proliferates in the affected culm, leaf and rhizome tissues. The affected culms become hard in texture, and internal tissue gets discoloured and quality deteriorates. Immunological detection of BoMV antigen by tissue blotting (Lin et al. 1993) demonstrated that most cultivated bamboo species in Taiwan-China with pachymorph rhizomes, including *Bambusa* and *Dendrocalamus*, are susceptible to bamboo mosaic. Infection of *B. oldhamii* results in the formation of a large number of unusual electron-dense bodies in the infected cells.

CONTROL

The large-scale use of vegetative propagules of bamboos has caused the disease to spread throughout Taiwan-

China. Infection studies showed that *Phyllostachys edulis, P. nigra* and *Bambusa vulgaris* var. *striata* (Lodd.) Gamble are susceptible to the disease, while *P. makinoi, P. pubescens, Dendrocalamus giganteus* and *D. strictus* are resistant. Cultivation of bamboos resistant to mosaic disease should be promoted. For controlling as well as checking the spread of the disease, preparation and use of vegetative propagules from diseased clumps should be avoided. At the same time, strict quarantine measures against the movement of infected planting materials within the growing areas also should be followed.

Thread Blight

Thread blight affecting culms, branches and foliage of different bamboo species has been reported from India. In Kerala States, the disease affects *Bambusa balcooa, B. bambos, B. multiplex* (Lour.) Raeusch. ex J.A. & J.H. Schultes, *B. polymorpha, B. tulda, B. tuldoides* Munro, *B. vulgaris, Dendrocalamus brandisii, D. longispathus, D. strictus* and *Thyrsostachys siamensis* in plantations, and *B. bambos, D. strictus, Ochlandra travancorica, O. travancorica* var. *hirsuta, O. scriptoria* and *O. ebracteata* in natural stands (Mohanan 1990, 1994a,b). In *B. vulgaris* plantations, 95% disease incidence was observed during 1991 (Mohanan 1994b). Thread blight affecting *Dendrocalamus* sp. has also been reported from Karnataka State (Rogers 1943). Disease appears during monsoons, subsides, and almost disappears during the dry period. Usually, infection occurs after the onset of south-west monsoon during June, and continues till the end of North-East monsoon during September-October. In high altitude areas, the disease affects reed bamboos during June-July and continues till December-January, often affecting the entire shoots of the clump, depending