KFRI Research Report 36

DISEASE SURVEY IN NURSERIES AND PLANTATIONS OF FOREST TREE SPECIES GROWN IN KERALA

J.K.Sharma
C.Mohanan
E.J.Maria Florence

KERALA FOREST RESEARCH INSTITUTE
PEECHI, THRISSUR

December 1985 Pages: 268

Index is Linked in this Report

CONTENTS

ABSTRACT

During the disease survey in ***Tectona grandis, Bombax ceiba, Ailanthus triphysa, Gmelina arborea. Dalbergia latifolia, Ochroma pyramidale*** and ***Eucalyptus*** spp. a total of 65 pathogenic and 13 other diseases (unknown etiology, non-infectious and phanerogamic parasite) were recorded. With these diseases altogether 88 pathogens were associated, of which 64 are new host record, including seven new species viz. ***Pseudoepicoccum tectonae*** and ***Phoinopsis variosporum*** on ***T, grandis, Meliola ailanthii*** on ***A. triphysa, Griphospharria gmelinae*** on ***G. arborca, Physalospora dalbergiae*** on ***D. latifolia*** and ***Cytospora eucalypti*** and ***Valsa eucalypticola*** on ***Eucalyptus*** spp., while 29 are first record from India,

T. grandis had fifteen diseases, two in nursery and fourteen in plantations; one being common to both. Ten organisms were associated with these diseases; mostly causing foliar damage; six pathogens are new host record and four first record from India. None of the two diseases in nurseries were serious whereas in plantations die-back caused by insect-fungus complex and a phanerogamic parasite, ***Dendrophthoe falcata*** were serious diseases capable of causing large-scale destruction. Three diseases viz. pink disease ***(Corticium salmonicolor),*** Phomopsis leaf spot and a disease of unknown etiology appeared to have potential to become serious.

In ***B. ceiba*** there were eight diseases, four in nursery and six in plantations; two were common to both. Four pathogens are new host record. Collar rot and seedling blight were the severe diseases causing appreciable loss to stocking. None of the plantation diseases were found to be serious, except Myrothecium leaf spot which in certain humid areas could pose some problem due to extensive premature defoliation.

A. triphysa had a total of nine diseases of which eight were in nursery and four in plantations; three were common to both. All the eight pathogens are new host record while two were first record from India. Among relatively large number of seedling diseases, two viz. collar rot and seedling blight were very widespread and damaging as they resulted in large-scale mortality. In plantations though none of the diseases were very serious, three viz. Botryodiplodia stem canker, pink disease and shot-hole were potentially serious, especially the former two, which killed the affected trees in certain localities.

There were ten diseases in ***G. arborea,*** three in nursery and eight in plantations; one was common to both. All the ten pathogens associated with these diseases are new

host record while three are recorded for the first time from India. In nurseries onlv seedling blight was of serious consequences though stem infection ***(Phoma nebulosa)*** also appeared to be potentially serious in certain localities. In plantations a die-back disease caused by ***Griphosphaeria gmelinue*** was the major disease as it resulted in heavy mortality.

In ***D. latifolia*** none of the four diseases of foliage recorded were of serious nature. Leaf spots caused by ***Physalospora*** and ***Colletotrichum*** are new diseases while for leaf rust and Phyllachora leaf spot ***D. latifolia*** is a new host.

Only two diseases were recorded in plantations of O. ***pyrumidale*** and none in the nursery. One of the diseases, die-back, resulting in large-scale mortality, was caused by two pathogens, ***Calonectria rigidiuscula*** and ***Fusarium moniliforme,*** the former being the first record from India.

Eucalyptus spp, recorded the highest number of 30 diseases, 13 in nursery and 21 in plantations; four were common to both. Of the 46 pathogens associated with these diseases, 30 are new host record and 18 first record for India. In nurseries, damping-off, seedling blight and leaf and shoot blights were serious seedling diseases affecting the nursery stock considerably, especially in high rainfall areas. In plantations, pink disease and leaf and shoot blights ***(Cylindrocladium*** spp.) were the major limiting factors during the first one to three years of establishment. A number of provenances of various species of ***Eucalyptus*** screened against pink disease following toxin bio-assay revealed variation in susceptibility between species and within provenances of a species. Other potentially serious diseases were web blight ***(Rhizoctonia solani)*** in nursery and stem cankers caused by ***Cryphonectria cubensis*** and ***Cytospora eucalypticola*** (both recorded for the first time from India), which can result in heavy mortality.

Control measures for 18 seedling diseases of various tree species were worked out and field tested for the efficacy of the fungicide and its dosage.

INTRODUCTION

Kerala, situated between 8°15' and 12°50' North and 74°45' and 77°30' East has typical tropical warm-humid climate with average annual rainfall of 3,020 mm and mean monthly temperature ranging from 17.5 to 29.5°C; the mean monthly relative humidity ranges from 75 to 92 per cent. About 24 per cent of the total geographical area of the State is under forest cover, including plantations. Large-scale afforestation programme was initiated in Kerala during 1960s to meet the growing industrial demand of wood. Moist-deciduous, semi-evergreen to evergreen forests, situated at high and low elevations, were clear-felled and 153189.64ha planted with various plantation species (Anon., 1985). Some of the important tree species raised in Kerala. are ***Tectona grandis*** L.f. (teak), ***Eucalyptus*** spp., ***Gmelina arborea*** **L.,** ***Ailanthus triphysa*** (Dennst.) Alston, ***Bombax ceiha*** **L.,** ***Dalbergia latifolia*** **Roxb.** (rosewood) and ***Ochroma pyramidale*** (Cav. ex. Lam.) Urb. (Balsa). The total area under some of these species is given in Table 1.

Table 1. Total area under various plantation species in Kerala[a]

Tree species	Total area (ha)	Per cent of the total
1. Teak	78452.2	51.21
2. Eucalypts	34330.4[b]	22.41
3. Soft wood plantations[c]	22796.4	14.88
4. *Ailanthus triphysa*	322.9	0.21
5, Rosewood	171.2	0.11
6. Balsa	117.5	0.08

[a]Anon.(1985)

[b]This does not include area planted by Kerala Forest Development Corporation.

[c]Mixedplantation of teak either with ***Bombax ceiba*** or ***Ailanthus triphysa.***

With the increasing demand for wood, production forestry has gained importance in recent years. While more area may not be available for further planting programmes in the State, need for increasing the productivity of the existing plantations by intensive management is being felt. Availability of healthy stock of seedlings for planting, and their disease-free condition subsequently in the field are the important aspects of the management, and if this is overlooked it may affect the yield considerably. To meet this requirement the first step is to minimise the disease hazards by proper nursery or cultural practices and secondly, if diseases do occur, to

control them by means of chemicals. The latter i. e., the control of disease, depends on the recognition of the causal organism of disease through symptoms. The incidence of a disease needs to be monitored for certain period of time to understand its level of severity, so that the chemical control can be justified economically. These facts establish a factual basis for methods of controlling a particular disease. If such basic information is not available attempts to control specific diseases will not be successful. In this way, it becomes necessary to have a detailed knowledge of various diseases affecting different forest plantation species grown in the State.

High rainfall combined with tropical warm-humid climate provides a conducive environment for the development and spread of several diseases, especially when the host is also susceptible. Indigenous species raised in monoculture plantations are seldom affected seriously with indigenous pathogens. However, if they do, they suffer severely. A good example of this is that of rubber in Brazil, where due to the native leaf blight fungus, ***Dothidiella ulei*** P. Henn., rubber plantations have never succeeded. On the other hand, exotic tree species are exposed to two types of disease hazards. Firstly, they may be attacked by a pathogen inadvertently introduced from the host's natural range of distribution where it is a minor pathogen. If the local climatic conditions are congenial this pathogen may become a serious problem causing epidemic. Examples may be cited those of twig blight of Mexican pines by ***Diplodia pinea*** (Desm.) Kickx, and ***Dothistroma*** and ***Cercospora*** blights of ***Pinus radiata*** D. Don. In another situation an exotic may be threatened by an indigenous parasite to which it possesses no resistance. In Kerala, pink disease of eucalypts comes under this category.

Forest disease surveys are usually aimed at periodical or continuous surveillance of forests or plantations with the objective to detect or even predict outbreaks of disease and damage, and diagnose the cause with a view to suggest control measures. The outcome of such surveys form the basis for assigning priorities for intensive research on specific disease problems. Bakshi initiated a systematic survey of forest diseases in India in 1967. But it could not be done as intensively as in the USA (Brown and Davidson, 1968) because of some practical problems (Bakshi *et al.*, 1972). His survey was mainly problem oriented in which intensive research was undertaken on some forest diseases of economic importance.

Concern about forest diseases in Kerala began during the early 1970s with the outbreak of diseases in industrial eucalypt plantations, which drastically affected the productivity. Infection by ***Cylindrocladium quinqueseptatum*** Boedijn & Reitsma was a serious problem in raising healthy nurseries. This pathogen, which was already well established on other crops such as cashew, clove, etc., adopted susceptible ***Eucalyptus*** quickly and caused heavy mortality of seedlings and young coppice shoots. The

problem was more alarming in high rainfall areas where 60 - 100 per cent eucalypt seedlings (in seedbeds and in containers) died due to ***Cylindrocladium*** infection within a short period. Soon an epidemic of pink disease of ***E. tereticornis*** caused by ***Corticium salmonicolor*** also came to forefront. Losses due to this disease were estimated at 55 - 95 per cent on account of severe infection in 5- to 11-year-old plantations at low and medium altitudes (Gibson and Armitage, 1979). Besides fire and establishment failures, these two diseases had so much adverse impact on the productivity of plantations that the availability of estimated 265000 t of eucalypt wood to various industries in Kerala has become doubtful (Karunakaran, 1982). In Kerala, though some studies on ***Cylindrocladium*** leaf blight and pink disease of eucalypts (Bakshi, 1972; Sehgal *et al.*, 1978; Seth *et al.*, 1978) and water blister in teak (Bakshi and Boyce, 1959) have been carried out, no attempt has been made to undertake a systematic forest disease survey. Considering the impact of diseases on the productivity of plantations it is essential to have a detailed knowledge of overall disease pressure on a particular host. The objective of this project was to prepare a checklist of all the diseases occurring on important plantation tree species ***(T. grandis, Eucalyptus*** spp., ***G. arborea, A. triphysa, B. ceiba, D. latifolia*** and ***O. pyramidale)*** grown in Kerala and assess the incidence of major diseases, suggest control measures wherever possible and identify major disease problems on which detailed investigation is warranted.

MATERIALS AND METHODS

Diseases in nurseries

For raising plantations, seedlings are grown by the Kerala Forest Department in nursery beds (12 x 1.2 m) each year during the dry season (December/January), usually near a permanent water source. For the first 45 to 60 days, a shade panda1 of coconut thatch is provided to protect seedlings from sun scorch. The seedbeds are watered at regular intervals with a prescribed quantity of water. When seedlings attain a height of 10 - 15 cm, they are pricked out into polythene containers (18 x 12 cm) (filled with sieved soil from nearby natural forest) during February/ March. For the first two to three weeks the container plants are also kept under shade. The container plants are watered regularly until they are outplanted in the field during June/July, after the onset of monsoon showers. In this way maintenance of the nursery seedlings of most of the tree species is required for atleast six months. However, this period is longer for teak where 12- to 18-month-old seedlings areutilized for preparing stumps. which are outplanted directly after the pre-monsoon showers.

During the survey information on location of nursery and host species was gathered from various Forest Divisions in the month of November/December. As far as possible most of the nurseries were visited frequently between January and May/June, when the seedlings were at different stages of growth. Occurrence of disease (s), if any, their symptoms and nature of damage caused to seedlings were recorded. Other relevant information pertaining to nursery practices, such as total number of seedbeds/container beds, sowing date, quantity of seeds per standard bed, soil characteristics, watering schedule, type of shade, besides the date of appearance of disease, were collected from the field staff. The incidence of a disease was estimated either by counting the number of disease patches and the approximate area covered by them or per cent seedlings affected for a given density of seedlings in a seedbed (Table 2). Appropriate specimens of diseased seedlings and soil samples were collected for isolation of the causal organism.

Table 2- Disease scoring scale for assessing the severity of seedling di seases in nursery

Disease severity	Disease scoring scale		
	Per cent seedlings affected per seedbed	No. of patches of diseased seedlings per seedbed	Per cent of seedbed area affected
Low (L)	1 – 25	1 – 25	1 – 10
Medium (M)	26 – 50; 10-15% seedlings dead	26 – 50	11 – 25
Severe (S)	50-75 or more; 25% seedlings dead	>50	>25

Diseases in plantations

Before selecting the representative plantations, a reconnaissance was undertaken to assess the disease situation in plantations of ***Tectona grandis, Bombax ceiba, Ailanthus triphgsa, Gmelina arborea, Dalbergia latifolia, Eucalyptus*** spp. and ***Ochroma pyramidale*** Representative plantations with some disease potential, easy accessibility and workable terrain were selected in various regions of Kerala. Selection of plantations was done in such a way that each species had young and mature plantations, if possible, in low and high elevation areas. The list of representative plantations is given under each tree species.

In each plantation, three to five plots of 20 x 20 trees were selected at random and alternate trees in alternate rows paint-marked for recording observations. Thus, in each plot 100 trees were observed. For recording intensive observations on certain diseases of ***Eucalyptus*** such as Botryodiplodia stem canker, pink disease and ***Cryphonectria*** canker, all the trees in a plot of either 10 x 10 trees (Botryodiplodia stem canker), 20 x 20 trees (pink disease) or 25 x 25 trees ***(Cryphonectria*** canker) were scored. In the case of ***D. lafifolia*** (rosewood) since no plantations were available with good stocking, observations were confined to 100 plants in a regeneration area at Kudhirakkode (Begur, Wynad Div.) and isolated trees in other plantations and natural forests.

As far as possible, the plantations were visited during the dry period (December - April) and wet period (June - September) and observations recorded on disease data sheets (Fig. 1). At each observation, several disease specimens and fructifications of stem decay fungi were collected for isolation and identification of causal organisms.

Apart from the representative plantations and nurseries, other areas from where some disease problems were referred to the Institute by the Kerala Forest Department and Kerala Forest Development Corporation, were visited and supplementary information gathered.

Severity and incidence of serious diseases: Severity (low, medium, severe) of serious diseases was rated on a numerical scale (1 - 3) of disease rating index as given in Table 3. The average severity index of a disease (DSI) in a plantation was calculated from the sum of total number of trees of each disease severity rating (DSR) in all the plots multiplied separately by the disease index (1 - 3) and dividing it by the total number of trees assessed (N) as given in the following formula.

$$\text{Disease severity index (DSI)} = \frac{n\,L \times I + n\,M \times 2 + n\,S \times 3}{N}$$

DISEASE SURVEY DATA SHEET

Host species: *Eucalyptus grandis* Year of planting: *1977* Date of observation: *21-F 1980.*

Locality: *T. Ramsbillerry (Wynaad)* Plot No: *3.* Row No: *6*

Severity of disease and name of the pathogen

Tree Number	LEAF: Leaf Spot Pathogen	LEAF: Leaf Spot Intensity	LEAF: Blight Pathogen	LEAF: Blight Intensity	LEAF: Rust Pathogen	LEAF: Rust Intensity	LEAF: Powdery mildew Pathogen	LEAF: Powdery mildew Intensity	LEAF: Virus Symptoms	LEAF: Virus Intensity	LEAF: Myco Plasma Symptoms	LEAF: Myco Plasma Intensity	STEM: Shoot blight Pathogen	STEM: Shoot blight Intensity	STEM: Die-back Pathogen	STEM: Die-back Intensity	STEM: Pink disease Pathogen	STEM: Pink disease Intensity	STEM: Canker other than Pink disease Pathogen	STEM: Canker other than Pink disease Intensity	STEM: Heart rot Pathogen	STEM: Heart rot Indicators	STEM: Gummosis Parasite	STEM: Gummosis Intensity	STEM: Angio-spermic parasite Parasite	STEM: Angio-spermic parasite No. of Clumps	ROOT: Wilt Pathogen	ROOT: Wilt Intensity	ROOT: Rot Pathogen	ROOT: Rot Intensity	ROOT: But rot Pathogen	ROOT: But rot Intensity	Remarks (Defoliation, injury, mortality, etc)
1	a	L																															
2	a c	S L	a	S													q	L															Defoliation Cattle damage
3	a c	L M																	i	L													
4	b	M															q	L															Branch infection Cat-Head stage
5	a b	L L																	i	L													
6	—	—																															Dead Cattle damage
7	c d	L L															q	L															
8	a c	L L																															
9	a c	M L																	j	L													Basal canker
10	a d	L L																	q	L													Basal & trunk canker

Pathogen/Parasite
a, *Cylindrocladium quinqueseptatum*
b, *Cercella fragariae*
c, *Phaeoseptoria eucalypti*
d, *Pestalotia sp*
e,
f, *Corynebacterium Subindis*
g, *Cortician salmonicolor*
h,
i, *Valsa eucalypti*
j,

Disease intensity: L - Low; M - Medium; S - Severe

Fig. 1. A ıple data sheet to illustrate the categories of diseases surveyed and method of recording observations

Table 3. **Disease index to assess the severity of foliage infection, stem canker and root diseases (wilt, root rot) in plantations**

Disease severity	Symptoms					Disease severity index (1 - 3)
	Foliage infection	Pink disease and others		*Cryphonectria* canker	Root disease	
		main stem canker	branch canker			
Nil	Nil	Nil	Nil	Nil	Nil	0
Low (L)	Upto 25% of the foliage infected	1 canker, no apparent harm to tree	Upto 25% of the shoots of trees affected	1 canker, no apparent harm to tree	Die-back of branches (> 25%) in the crown	1 (0.1-1)
Medium(M)	25 - 50% of the foliage infected; > 10% defoliated prematurely	1 - 2 cankers, epicormic shoots present	> 25 - 50% of the shoots of trees affected	1 - 2 cankers, more than a meter long, gummosis present	Die-back of branches (> 50%), thinning of crown	2 (1.1-2)
Severe (S)	50 - 75% or more foliage infected; > 25% defoliated prematurely	1 - 2 or more cankers, epicormic shoots present, apical shoot dead due to girdling	> 50 - 75% of the shoots of trees affected	1 - 2 or more cankers, several meters long, gummosis present, tree dead due to girdling	Foliage pale yellow accompanied by premature defoliation, extensive die-back, death of tree	3 (2.1-3)

Where nL, nM, nS represent total number of plants with Low, Medium and Severe disease severity; 1, 2, 3 Disease Severity Index (DSI) for Low, Medium and Severe respectively and N the total number of trees assessed in all the observation plots.

The per cent incidence of a disease in plantation was calculated from the total number of plants affected (nd) and total number of plants observed in all the plots (N):

$$\text{Per cent incidence} = \frac{nd}{N} \times 100$$

Isolation and identification of causal organism

Disease specimens and soil samples collected from the field were transported in separate clean polythene bags to the laboratory. To avoid any saprophytic growth on the specimens, isolations were made within a week of collection. Only under unavoidable circumstances the specimens were stored in a refrigerator.

Generally, the media used for isolation of the organisms were potato dextrose agar (PDA) for fungi and nutrient agar for bacteria. After isolating the causal organisms in pure culture, identification was attempted at least upto generic level, based on cultural and morphological characteristics. For specific identification or confirmation, the cultures, herbarium specimens bearing fructifications and fructifications of basidiomycetous fungi were referred to Commonwealth Mycological Institute, Kew, U. K. or Royal Botanical Garden, Kew, U. K. The identified cultures were subcultured and stored in a cold room at $25 \pm 2^{o}C$. The cultures were subcultured at three to four month's interval, except in the case of bacteria where frequent subculturing was required.

Pathogenicity tests

The pathogenicity of the isolates was confirmed in artificial inoculation trials. For leaf and seedling diseases the tests were conducted in the laboratory, and for stem diseases in the field. During the experiment, the temperature and relative humidity were recorded.

As most pathogens usually require high humidity (>95% r. h.) for infection and expression of disease symptoms, the laboratory experiments were carried out in a specially designed humidity chamber (Fig. 2), fabricated locally with perspex sheet and light aluminium angles.

For inoculation of leaves, wherever possible, detached leaf culture technique was employed. The detached leaves or leaflets were floated on a solution of appropriate growth hormone, standardized to prevent senescence (Goldthwaite and Laetsch, 1968). Benzimidazole and gibberellic acid, used separately either at 5 or

10 ppm, were found to prolong greenness of leaves for about four to six weeks depending upon the tree species used.

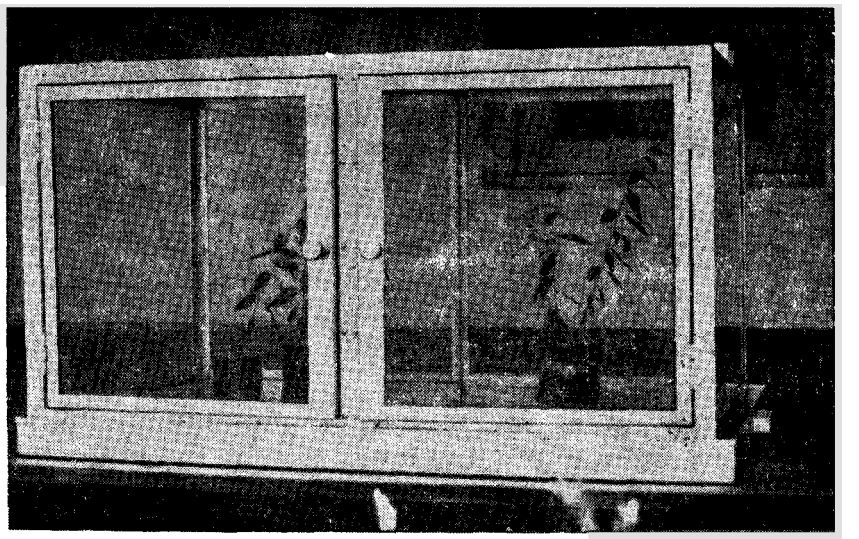


Fig.2 A view of the specially designed humidity chamber used in pathogenicity and other epidemiological studies.

In the case of root: stem and shoot diseases of seedlings, the pathogenicity was tested in aluminium trays (30 x 30 x 5 cm). Initially, the seedlings were raised in sterile soil and then transplanted in the trays containing 2 kg of fine sieved sterile soil. The seedlings were allowed to establish for a few days in the humidity chamber and then appropriately inoculated. For soil-borne diseases, the soil was infested with appropriate quantity of inoculum of the test fungus, usually raised on corn meal sand medium, dried and powdered. The trays were maintained in the humidity chamber throughout to observe the development of disease.

Transmission studies: In the case of diseases caused by bacteria (especially ***Agrobacterium),*** virus or mycoplasma like organisms (MLO) transmission studies were carried out following standard sap and graft procedures.

Specific details of pathogenicity tests or transmission studies are described separately under each disease.

Photography

To study the symptomatology of the disease, photographs (colour and B/W) were taken as far as possible of fresh diseased specimens.

Microtomy, light microscopy and photomicrography

To study the detailed structure of fructifications of various pathogenic fungi (pycnidia, perithecia, rust sori, etc.) appropriate specimens were selected and their sections (2 - 6 pm) cut using Minotome cryomicrotome (IEC, USA). The sections were either double stained with safranine and fast green and mounted in DPX or stained and mounted in lactophenol cotton blue and the edge of the cover glasses sealed with DPX mountant to make it permanent.

Sections of fructifications of various pathogens were observed under Leitz Dialux - 20 microscope and photomicrographs taken using automatic Vario Orthomat camera.

Scanning electron microscopy (SEM)

Details of the morphology of fructifications of various pathogens were studied under Hitachi S - 540 scanning electron microscope. Appropriate specimens were prepared for SEM after freeze drying and coating them with gold under vacuum.

Meteorological data

Data on rainfall and temperature gathered for different locations in the State are presented in Figs. 3a, b.

Evaluation of fungicides for disease control

Various fungicides (Appendix - I.) were evaluated for their efficacy against different pathogens following poison-bait technique and / or soil method as described below.

Poison-bait technique: To obtain a desired concentration, an appropriate quantity of the test fungicide was mixed thoroughly with the sterilized PDA medium before it solidified. Each concentration of a fungicide was replicated in three to five petri dishes which were inoculated in the centre with a mycelial disc of 3 or 5 mm diam., taken from the margin of an actively growing colony of the test fungus. The inoculated petri dishes were incubated at 25 $\pm$ 2°C and three to four observations of radial or diameter growth of the colony recorded.

Soil method: The soil-fungicide screening method described by Zentmeyer (1955) and Cordon and Young (1962) was modified and used to evaluate the efficacy of fungicides against soil-borne fungi, especially those producing sclerotia or micro-sclerotia. The procedure is detailed below.

Air dried soil was sieved through a sieve (3 mesh cm-2) and autoclaved for 45 minutes at 1 kg cm-2 pressure. The soil was allowed to cool and then 10 g of this soil was placed in a sterile glass vial, 30 mm diam. and 80 mm long. An 8 mm disc

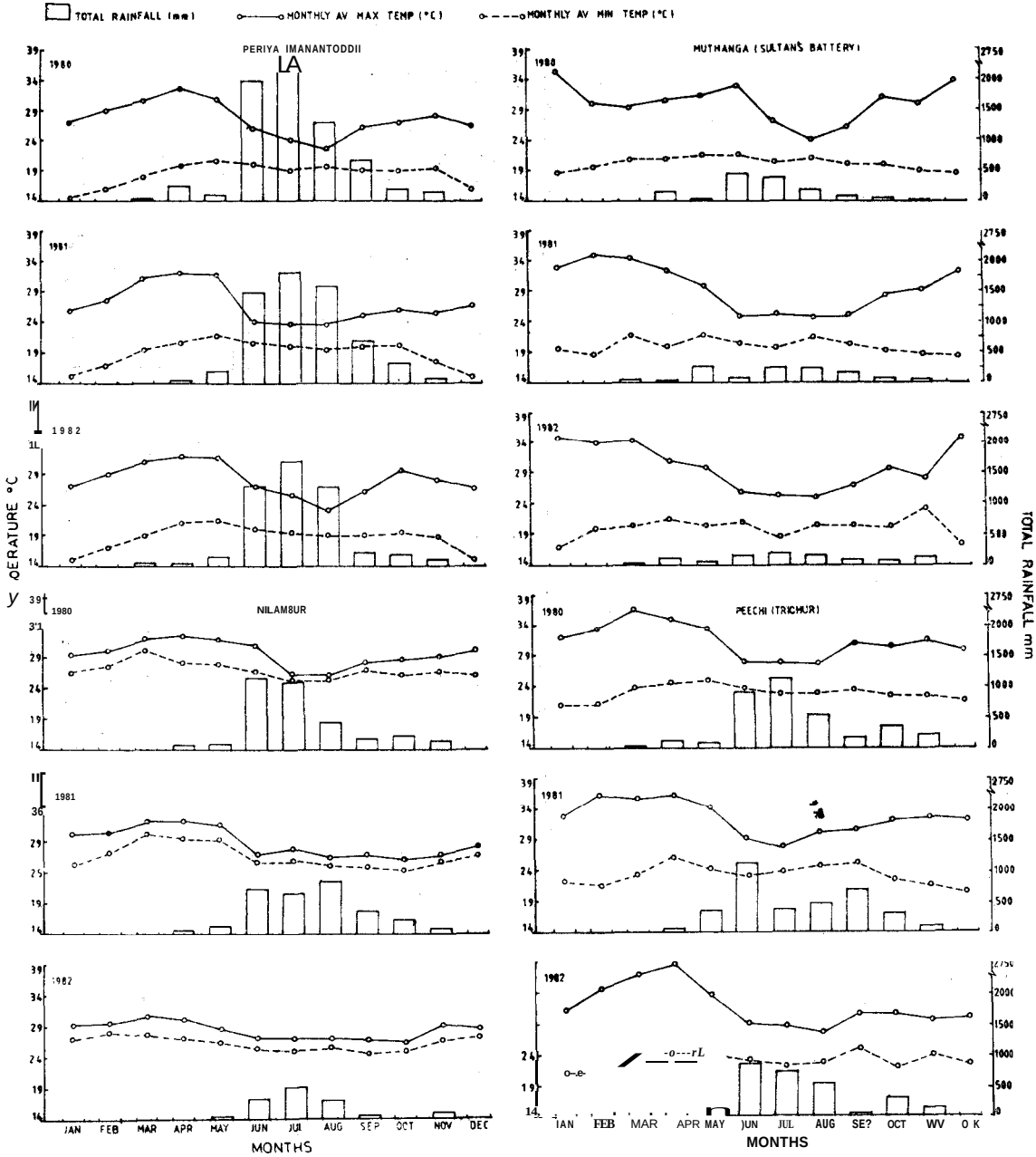


Fig. 3a. Rainfall and temperature for Periya (Manantoddy), Muthanga *(S*. Battrey), Nilambur and Peechi (Trichur) during 1980-1982.

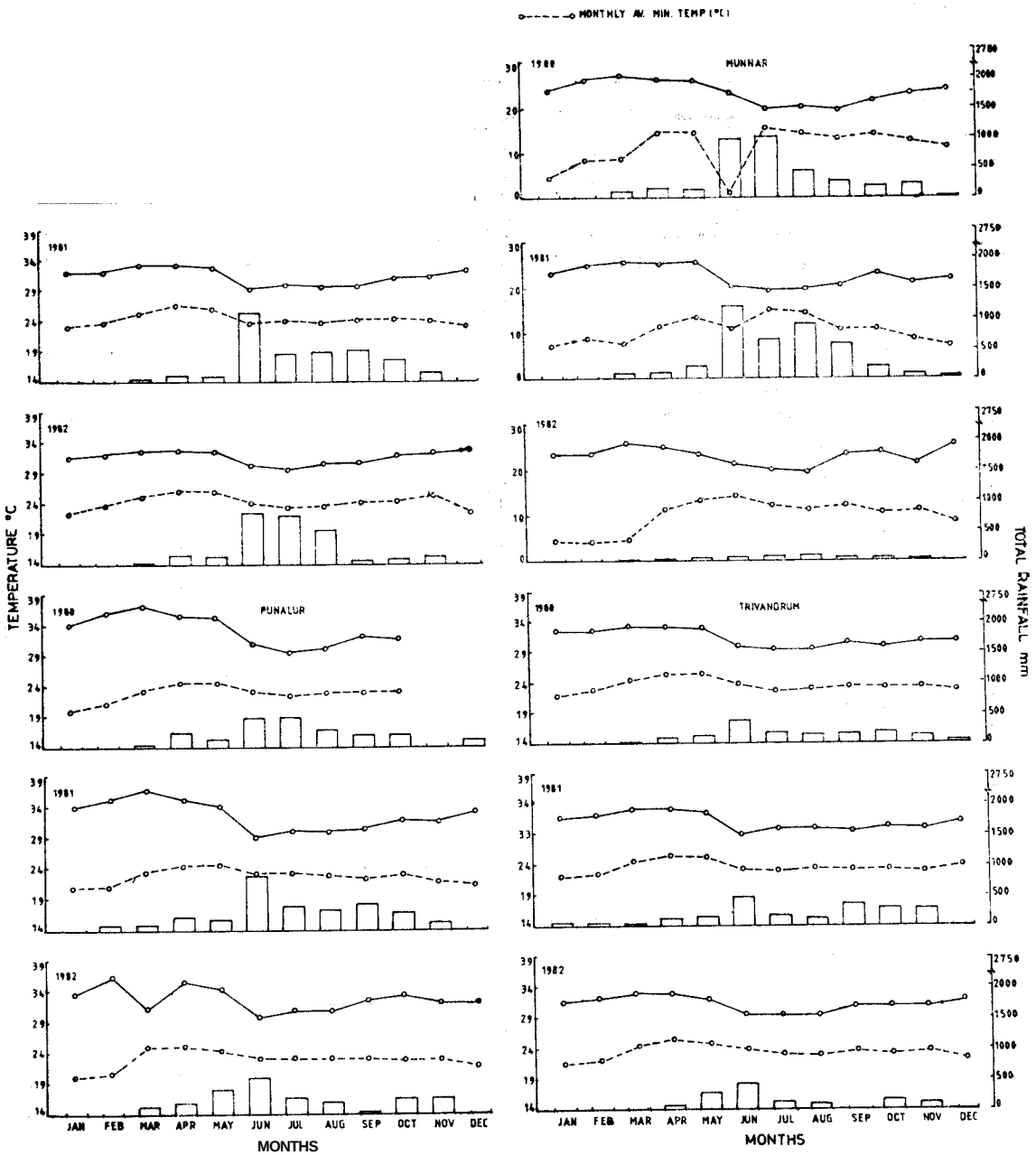


Fig. 3b. Rainfall and temperature for Cochin, Munnar, Punalur and Trivandrum during 1980-1982.

punched from an actively growing, 7- to 10-day-old, colony of the test fungus on PDA was transferred over the soil. Another 10 g of sterile soil was placed over the disc. Fungicide solution (7 - 9 ml) of a desired concentration, prepared in sterile water, was gently poured over the soil surface using a sterile pipette. The mouth of the vial was covered with aluminium foil. At least three replications of each concentration were kept. These vials were incubated for 24 h at 25 + 2°C. After incubation, the soil from the vials was emptied gently and the agar disc removed with sterile forceps. The disc was washed in three changes of sterile water to remove the adhering soil particles and transferred to a petri dish containing PDA. Observations on the diameter growth of the colony, if any, were recorded after seven days.

RESULTS

DISEASES OF VARIOUS TREE SPECIES

Most of the works in forest pathology including survey conducted by Bakshi *et. al*. (1972) have been organized by the cause of the disease rather than the tree species attacked. Keeping in view that anyone with a problem in diagnosis is presented first with a diseased plant and then only comes the question of knowing the symptoms of the disease and the pathogen associated with it, for convenience all the diseases occurring on a particular tree species are described together. This way it will also help to project the overall impact of diseases on a given species. The diseases listed under each host are divided into nursery and plantation diseases. Some diseases occur exclusively either in nurseries or in plantations while others are found in both. In the latter case the diseases are described in detail under plantation and brief reference made under nursery. Wherever possible, control measures of serious diseases were worked out and recommendation made. For others, information, if available, was collected from the literature and presented at appropriate place. Each disease is discussed separately and a general discussion on significant diseases is provided at the end. Cultural and morphological characters of fungal and bacterial pathogens are provided in Appendix 11.

TECTONA GRANDIS

Tectona grandis L., a large deciduous tree, is indigenous to India, Burma, Thailand, Vietnam, Indonesia and Java. Now it is also grown as one of the major plantation species in a number of countries of Africa, South America and in Sri Lanka and West Indies. In Kerala, trial planting of teak was initiated in 1842 by Conolly and large-scale planting started from 1844 onwards. Since then 73,929 ha of teak plantations have been raised in the State (Anon., 1983). The major teak growing areas are located in Wynad, Nilambur and Konni Forest Divisions.

To maximise the yield in teak platitations at various stages of growth, six thinnings (mechanical and silvicultural) are carried out at 4, 8, 13, 20, 30 and 44 years. Depending upon the growth and yield, in teak three site quality classes have been recognised. Initially the rotation age was fixed at 80 years but due to increase in demand subsequently it has been reduced to 50 to 70 years in various Forest Divisions.

A list of eight plantations surveyed for diseases is given in Table 4.

Table 4. List of representative plantations of *Tectona grandis* surveyed for disease occurrence during 1980 - 1982

Sl. No.	Locality (Forest Range)	Altitude (m above msl)	Forest Divn.	Area of plantation (ha)	No. of plots surveyed	Year of planting	Age of trees at survey in 1980 (years)
1.	Kudhirakkode (Begur)	350	Wynad	62.50	5	1975	5
2.	Rampur (Sultan's Battery)	850	Kozhikode	56.52	5	1975	5
3.	Ezhuthukallu	50	Nilambur	5	5	1974	6
4.	Mundakkadavu	150	Nilambur	—	5	1920	60
5.	Thundathil*	25	Malayatoor	64.48	5	1975	5
6.	Naduvathumoozhi (Perunthomoozhi serie:	80	Konni	10.20	3	1963	23
7.	Mampazhathara (Pathanapuram)	150	Punalur	49 85	3	1974	6
8.	Choodal* (Thenmala)	200	Thenmala	—	5	1977	3

* Mixed 'softwood' plantation with *Bombax ceiba*

NURSERY DISEASES

No typical nursery diseases such as damping-off or seedling blight were found in teak. The commonest disease was rust followed by a bacterial collar rot of seedlings. Teak rust also prevalent in the nurseries is dealt with under plantation diseases.

1. BACTERIAL COLLAR ROT

Occurrence

Bacterial collar rot disease was recorded in 5-month-old teak seedlings at Kayad, Moovattupuzha (Malayattoor Div.) and Chethaleth, S. Battery (Kozhikode Div.). About 5 - 10 per cent of seedlings in all the nursery beds were found to be affected.

Symptoms

In the affected seedlings, initially the collar area just above the ground showed a slight shrinking. At this stage the top leaves became flaccid and drooped (Fig. 4a). When the affected area turned blackish-brown and got further constricted, the seedlings showed scorching of leaves with pronounced symptoms of wilting (Fig. 4b). The wilted seedlings died soon.

Other diagnostic characteristic of the disease: When the freshly cut stem above the affected area was immersed in a clean glass of water, streaks of off-white bacterial ooze from the vascular elements confirmed the systemic bacterial infection.

Etiology

From all the above-ground parts of the affected seedlings consistently a ***Pseudomonas*** sp. (possibly P. ***solanacearum*** (E. F. Smith) E. F. Smith) was isolated on nutrient agar. The bacterium was gram negative, rod-shaped and motile.

Pathogenicity tests

Six-month-old teak seedlings, collected from a healthy nursery, were transplanted separately in plastic containers filled with pasturised soil. The seedlings were allowed to establish for about a month in shade. Before inoculation, seedlings, grouped in seven sets of five seedlings each, were washed with sterile distilled water. Four sets of seedlings, allotted for inoculation with or without injury had either inoculation done only on the stem or on the stem as well as the container soil drenched with 50 ml of bacterial suspension (1×10^{10} per ml), prepared from 3-day-old actively growing culture. In the fifth set only the container soil was drenched with the bacterial suspension. The last two sets were uninoculated controls. For injury, about 20 pricks were made in an area, about 2 cm in length, near the collar

region using a sterile needle. The injured and uninjured areas of the collar were sprayed with the bacterial suspension till run-off employing a fine atomizer. All the seedliflgs were transferred to a humidity chamber, maintained at > 95% r. h. The containers were watered with measured quantity of sterile distilled water whenever required. During the experiment the temperature ranged between 25 to 31°C. Observations on the appearance of disease symptoms were recorded daily.

Disease appeared first in the injured seedlings on the fifth day of inoculation. The apical leaves became flaccid and drooped down. Four of the five seedlings died. In treatment without injury, only one seedlings died. None of the seedlings, where the soil was drenched with bacterial suspension, developed any disease even after a month of inoculation.

Control measures

The disease was controlled by an application of Plantamycin 0. 0 l % (a.i.) as a soil drench at Kayad.

The following measures are recommended for avoiding the development and spread of Bacterial collar rot of teak seedlings:

i. Since the disease is manifested mainly under water logged or high soil moisture regimes, seedbeds should be properly levelled, raised and made up of loose well drained soil; clayey soil should be avoided.

ii. Weeding operations should be carried out carefully so as not to cause any injury to seedlings.

iii. After the appearance of the disease affected seedlings should be uprooted and burnt and watering quantity and frequency minimised.

Discussion

This is the first report of bacterial collar rot disease of teak. It is not considered to be a serious problem as it was observed only in a few nurseries. Whether clayey soil of the nursery raised in the paddy fields, coupled with high soil moisture and high temperature were responsible for the disease needs further study. Though pathogenicity trials clearly indicate that the disease is manifested through wounds, detailed investigation? are required on the factors responsible for the development of the disease.

PLANTATION DISEASES

A total of 14 diseases were recorded in plantations. The most prevalent was teak rust followed by Phomopsis leaf spot and Pseudoepicoccum leaf spot. Severe infection of these three diseases usually resulted in premature defoliation. There

Fig. 4. Bacterial diseases and powdery mildew of teak. a, A wilted seedling in a nursery bed affected with bacterial collar rot. Other diseased seedlings have been uprooted from the empty patch for examination; b, A seedling affected with collar rot. Note darkening of the tissues at the collar region (marked with an arrow) and unaffected root; c, A one-year-old sapling affected with bacterial wilt at Kariampanny, Kannoth, where paddy was grown as taungya; d, A leaf affected with powdery mildew, *Uncinula tectonae*.

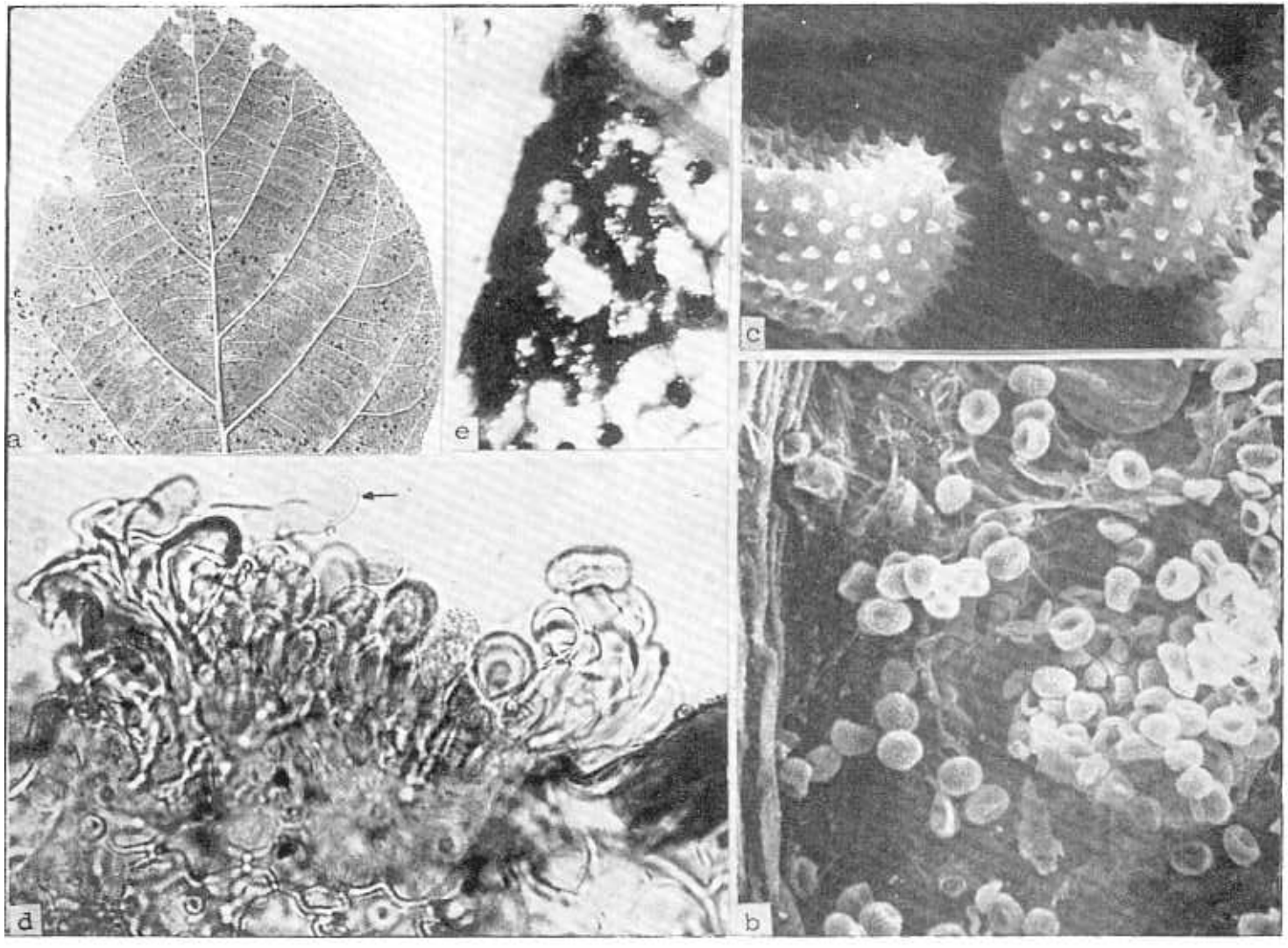


Fig. 5. Leaf rust *(Olivea tectonae)* of teak. a, Abaxial surface showing minute necrotic spots due to severe rust infection; b, SEM of a uredinium on the abaxial surface (222 X); c, A magnified view of urediniospores in SEM (1440 X). Note the spiral arrangement of echinulations; d, A vertical section of leaf through uredinium. Note abundant marginal paraphyses with curved distal ends; e, A rust pustule surrounded with a necrotic spot.

were three diseases of unknown etiology namely i. mosaic, ii. vein clearing and yellowing of interveinal areas, and iii. stunting and shoot die-back.

1. BACTERIAL WILT

Occurrence

Bacterial wilt of teak was recorded in young plantations, varying from 6-month-old to 2-year-old, at Thundathil (Malayattoor Div.), Kariampanny, Kannoth (Wynad Div.), Mullachal (Trivandrum Div.) and Gurunathanmannu, Angamoozhi (Ranni Div.). The incidence of the diseases varied from <1 per cent to ca. 20 per cent in a given area of the plantation, maximum being at Thundathil.

Symptoms

The disease manifests during warm and wet period, especially just after the onset of monsoon showers. The infection usually occurred through injury and occasionally through lenticels. The symptoms, characteristic of vascular wilt disease, were expressed in the following dry period. The bacterium caused systemic infection of vessels, which showed necrosis and discolouration.

Initially the bottom leaves of the affected plants turned yellow and showed scorching and browning of leaf tissues in between the veins. The upper leaves, near the apex became flaccid and drooped. Such plants wilted and failed to survive (Fig. 4c). The roots of the affected plants got discoloured and decayed.

Other diagnostic characters of the disease: The vascular tissue of the wilted plants showed characteristic browning, which could be easily seen in the stem or at the leaf scar (point of attachment of petiole to the stem). When the cut edge of the affected tissue was immersed in a clean glass of water an off-white streak of ooze, emerging within a few seconds, confirmed systemic bacterial infection.

Etiology

From all the affected plant parts a ***Pseudomonas*** sp. (possibly ***P. solanacearum*** (E. F. Smith) E. F. Smith) was isolated. The bacterial colony was very slow growing on PDA and has to be maintained and frequently subcultured on nutrient agar with precipitated $CaCO_3$. The bacterium was gram negative, motile, and rod-shaped with round ends.

Control measures

Once a plant is wilted it cannot be cured. However, with proper sanitation methods and precautions suggested below the deyelopment and spread of the disease can be checked to a considerable extent (Bakshi, 1975).

i. Planting in water-logged areas should be avoided.

ii. As the disease is easily manifested through an injury to the root system, utmost care should be taken during weeding and soil working operations.

iii. The affected plants should be uprooted carefully and burnt.

iv. For casualty replacement, the planting should be done in a separate pit dug away from the site of the infected plant.

v. If possible, the soil at the site of the infected plant should be drenched with Plantamycin 0.01% (a. i.).

Discussion

A taungya crop of tapioca ***(Manihot esculanta*** crantz) or ginger ***(Zingiber officinaIe*** Rose.) was grown at all places where this disease was observed, except at Kariampanny where it was paddy. It is most likely that some injuries may have been caused to the root system during weeding and soil working operations for the taungya crop, which acted as the entry point for the soil-borne bacteria. If skilled workers are engaged and precautions are taken, such a disease hazard can be minimised. In general the disease does not appear to be a serious problem.

This is the first report of the occurrence of bacterial wilt disease of teak from India. Earlier, it has been recorded in Sumatra (Anon., 1937), Philippines (Rolden and Andres, 1953), Malaysia (Mitchell, 1962) and Burma (Doo, 1968).

2. POWDERY MILDEW

Occurrence

Powdery mildew of teak was recorded from Rampur, Walayar (Palghat Div.), Peechi (Trichur Div.), Thundathil, Choodal, Achenkoil (Teak Plantation Div.). Though trees of all the age groups were found to be affected with powdery mildew, generally it was observed in old plantations (>15-year-old). Except at Walayar, where it was of medium severity, the incidence of the disease was generally very low.

Symptoms

Irregular white patches, consisting of mycelium and asexual conidia, developed on the upper leaf surface towards November/December just before the senescence (Fig. 4d). These patches coalesced and covered the entire surface of the leaf giving greyish-white powdery appearance. Severely infected leaves were defoliated prematurely. Black fructifications, cleistothecia, were observed only on the fallen leaves at Walayar.

Etiology

Uncinula tectonae Salm.

Control measures

Sulphur dust was found to be the most effective fungicide in controlling ***Uncinula tectonae*** on 2-year-old seedlings followed by Baycor (triadimenor), Morestan (quinomethionate) and Calixin (tridemorph) (Kulkarni *et al.,* 1979).

Discussion

U. tectonae appears only towards the end of the growing season, when favourable high temperature and humidity are encountered. Except at Walayar, where it caused severe premature defoliation, this disease was not of major concern. Due to severe infection the whole leaf surface is covered with mycelial growth which substantially reduces the photosynthetic area, consequently causing premature leaf fall. The disease does not appear to be of serious consequence as it occurs only when the senescence is approaching.

Powdery mildew of teak has been reported earlier from coastal areas of Bombay, Madhya Pradesh, Coorg (Karnataka), and foot hills of Himalayas (Bagchee, 1952) and Gujarat (Bakshi *et. al.,* 1972). This is the first report of powdery. mildew of teak from Kerala.

3. LEAF RUST

Occurrence and severity

Teak leaf rust, one of the important leaf diseases, was widespread in nurseries as well as in plantations, especially in dry areas. Though the rust occurred almost round the year, it was most prevalent during August to January/February. The rust was seen appearing in February/March on the older leaves of the new flushes on which it continued even during and after the rainy season. On coppice shoots, the rust was recorded almost throughout the year.

The severity of rust varied from locality to locality and year to year from low to medium depending upon the climatic conditions (Table 5). Medium infection was recorded at Rampur during 1980, at Mampazhathara in 1981 and at Thundathil and Choodal in 1982. At Nilambur (Karulai and Ezhuthukallu) and Naduvathumoozhi no rust infection was observed. Medium severity of rust was always accompanied with high incidence; however, this was not so with low severity. The severity of rust was relatively higher in nurseries and younger plantations (<10-year-old) than in older ones and in areas with low rainfall (Figs. 3a, b). Severe rust infection of seedlings at Ariankavu (Thenmala Div.) and Kariampanny, Kannoth (Wynad Div.) caused extensive premature defoliation,

Table 5. Severity of rust caused by *Olivea tectonae* in teak plantations at different localities in Kerala during 1980 - 1982

Sl. No.	Locality	1980 % incidence	1980 DSI	1980 DSR	1981 DSIa	1981 DSRb	1982 % incidence	1982 DSI	1982 DSR
1.	Rampur		1.81	M	59.95	0.6	L	45.96	0.57	L
2.	Kudhirakode		0.03	L	5.05	0.05	L	3.56	0.09	L
3.	Mundakkadavu					0	Nil	--	—	—
4.	Ezhuthukallu		0	Nil	0	0	Nil	—	—	—
5.	Thundathil	44.0	0.46	L	37.25		L		1.5	M
6.	Naduvathumoozhi	0				0	Nil	—	—	—
7.	Mampazhathara		0.23	L	68.22	1.33	M	—	—	—
8.	Choodal		0.44	L	71.50	0.9	L	96 02	1.2	M

[a] DSI, Disease Severity Index
[b] DSR, Disease Severity Rating
-, Observations not recorded

Symptoms

The rust infection appeared first on mature leaves during August/September and as the younger leaves matured it proceeded upwards reaching upto the top leaves towards the end of the growing season (December/January). The upper surface of affected leaves showed scattered dull green flecks corresponding to the orange yellow uredinia on the lower surface (Figs. 5a,b). These flecks turned necrotic in due course and appeared as small brown spots. Severe rust infection caused premature defoliation in nurseries and young plantations, which possibly affected the growth of plants, especially in the former.

Etiology

OIivea tectonae (T. S. & K. Ramakr.) Mulder (IMI 273439) is a micro-cyclic rust known only in telial and uredinial stages on teak. In Kerala only uredinial stage was observed (Figs. 5b-d).

Possibly a different strain of the rust occurred during January/February to April/May. First it appeared on the mature leaves of the new flushes or young leaves of seedlings in nursery and later spread to mature leaves. The rust was

characterised by a necrotic reaction on the upper surface of leaves (Fig, 5a). The uredinia which produced fewer spores than the normal rust, became dull yellowish-brown and lost their fecundity after the necrotic spot increased in size (Fig. 5e). Urediniospores were deep yellowish orange, slightly smaller, 15.0-21.0 x 15.5-24.5 um, than the normal rust.

Rust hyperparasites

During the course of the survey two fungal hyperparasites, namely ***Acremonium recifei*** (Leao & Lobo) W. Gams (IMI 284045) and ***Cladosporium oxysporum*** Berk. & M. A. Curtis (IMI 284044) were recorded on teak rust. Both the fungi were isolatetd on potato dextrose agar (PDA) medium.

A. recifei, the main hyperparasite, parasitises the uredinia during the comparatively dry period of the year from October to February/March. Often the uredinia are completely covered with the parasitic growth and appear white (Fig. 6a). The parasitic fungus sporulates profusely on the uredinia (Figs. 6b,c).

C. oxyspurum has mainly been observed just after the rainy season during September to November. It appears in the form of olivaceous green to black growth (Fig. 7a) with abundant sporulation over the uredinia (Figs. 7b, c): often covering a large part of the leaf.

Control measures

Some control of rust has been obtained in young plantations by opening up the crop by thinning or pruning the branches. In nurseries the rust was controlled by foliar sprays of sulphur based fungicides (Khan, 1951). Application of Plantvax (0.0l% a. i.), a systemic fungicide effective against rusts, may also be found effective in controlling teak rust.

Discussion

Teak rust, **O.** tectonae, widely distributed in the South Asian region, has been reported from India, Sri Lanka (Bagchee, 1952: Singh and Bakshi, 1964), Bangla Desh (Khan, 195l), Indonesia (Kelshoven, 1928) and Thailand (Chandrasrikal, 1962). Earlier, since only the uredinial stage was known teak rust was placed under ***Uredo tectonae*** by Raciborski (1900). Later, when telial stage was discovered it was transferred to ***Chaconia*** by Ramakrishnan and Ramakrishnan (1949). Thirumalachar (1950) placed it in Olivea because of characteristic telia developing on thick cellular base.

As the alternate host of teak rust is not known and in Kerala only uredinial stage occurs, the survival of ***O. tectonae*** is possibly only through urediniospores. Our

field observations confirm this view since uredinia can be observed almost throughout the year on seedlings in nurseries and on coppice shoots or naturally regenerated seedlings in plantations.

In Kerala, build up of severe rust infection in plantations was usually noticed only towards the beginning of autumn when the senescence of leaves sets in. Therefore, the impact of rust on the growth of trees may not be very significant. Gibson (1975) has reported some serious loss in increment due to teak rust. However, in nurseries severe rust infection, which causes extensive necrosis and premature defoliation, definitely will have detrimental effect on the growth of seedlings.

Depending upon the climatic conditions the intensity of rust varied every year during the past five years of observation. Good rains followed by dry period appeared to be conducive for the build up of severe rust infection. Usually two peaks of high rust incidence were observed, one in October/November and another during January/February. In November/December because of the North-East monsoon showers the rust infection apparently subsides. During the past five years of observation severe rust infection was noticed only during 1984 after the monsoon. But hyperparasitism of uredinia by ***C. oxysporum*** and ***A. recifei*** checked the upward trend of the rust infection. Infection does not build up to an epidemic scale in Kerala possibly due to these two hyperparasites of the rust. To understand the host parasite relationship of ***O. tectonae*** and the role of the two hyperparasites in biological control detailed investigations are necessary.

4. PHOMOPSIS LEAF SPOT

Occurrence and severity

This is one of the most common leaf diseases of teak in Kerala. Leaf spots appeared during August/September and infection continued till November/December.

The severity of Phomopsis leaf spot varied from low to severe, the latter being recorded at Mundakkadavu during 1980 (Table 6); no infection was recorded at Naduvathumoozhi (1980-1982) and Choodal during 1982. Though trees of any age group were susceptible, high incidence accompanied with medium/severe infection was often observed only in young plantations (4- to 10-year-old), especially those situated in humid tracts.

Symptoms

Initially the spots appeared as minute dark brown dots, 2-3 mm across, during late August. Soon these spots enlarged to 5 to 8 mm in diameter and became light pale brown in colour with a dark brown outline. The latter grew outwards and

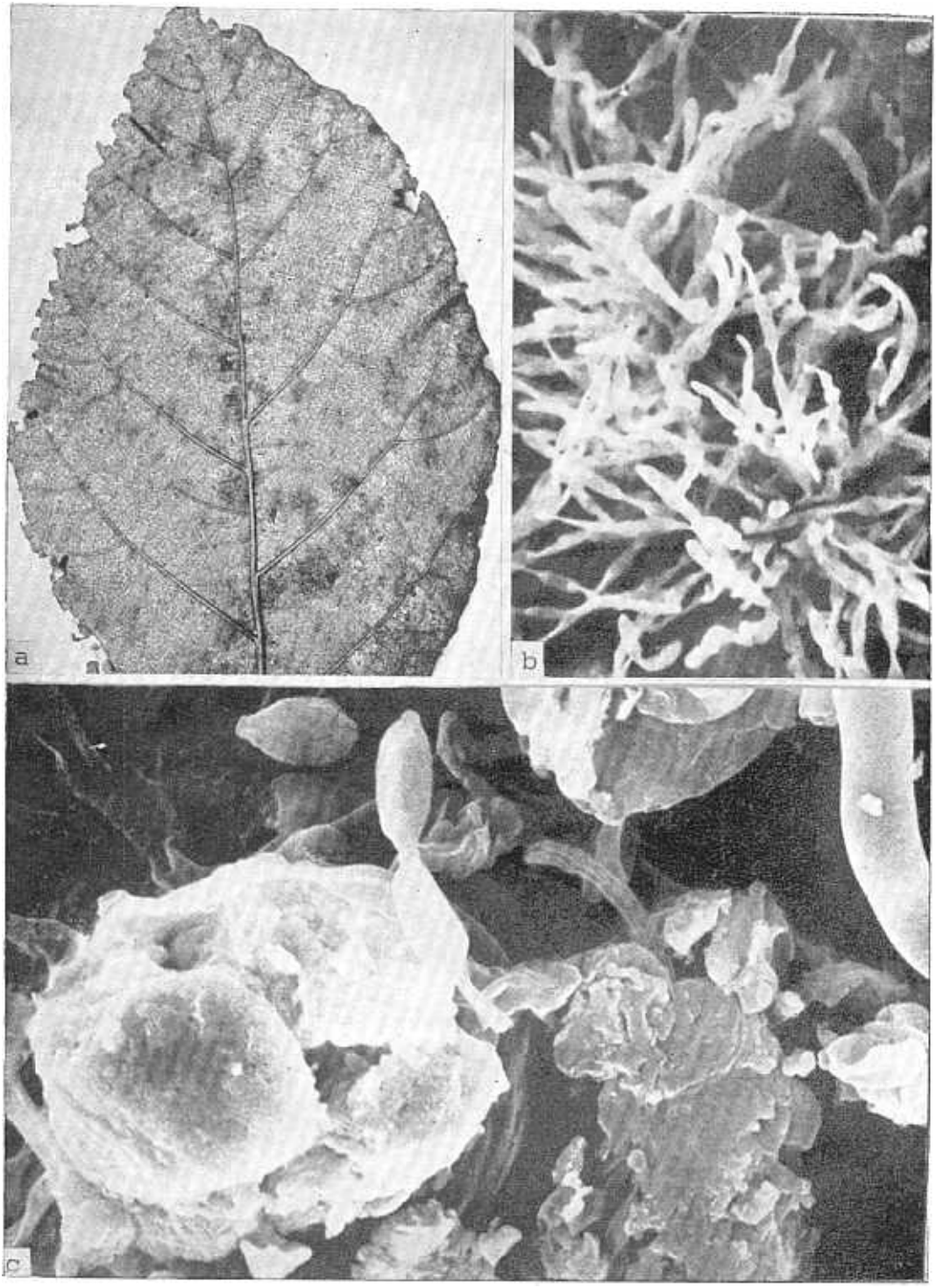


Fig. 7. Hyperparasitism of teak rust by *Cladosporium oxysporum*. a, A leaf showing profuse dark growth over the rust pustules; b, SEM of a sporulating colony of *C. oxysporum* (380 X); c, Urediniospores parasitized by *C. oxysporum* (2050 X).

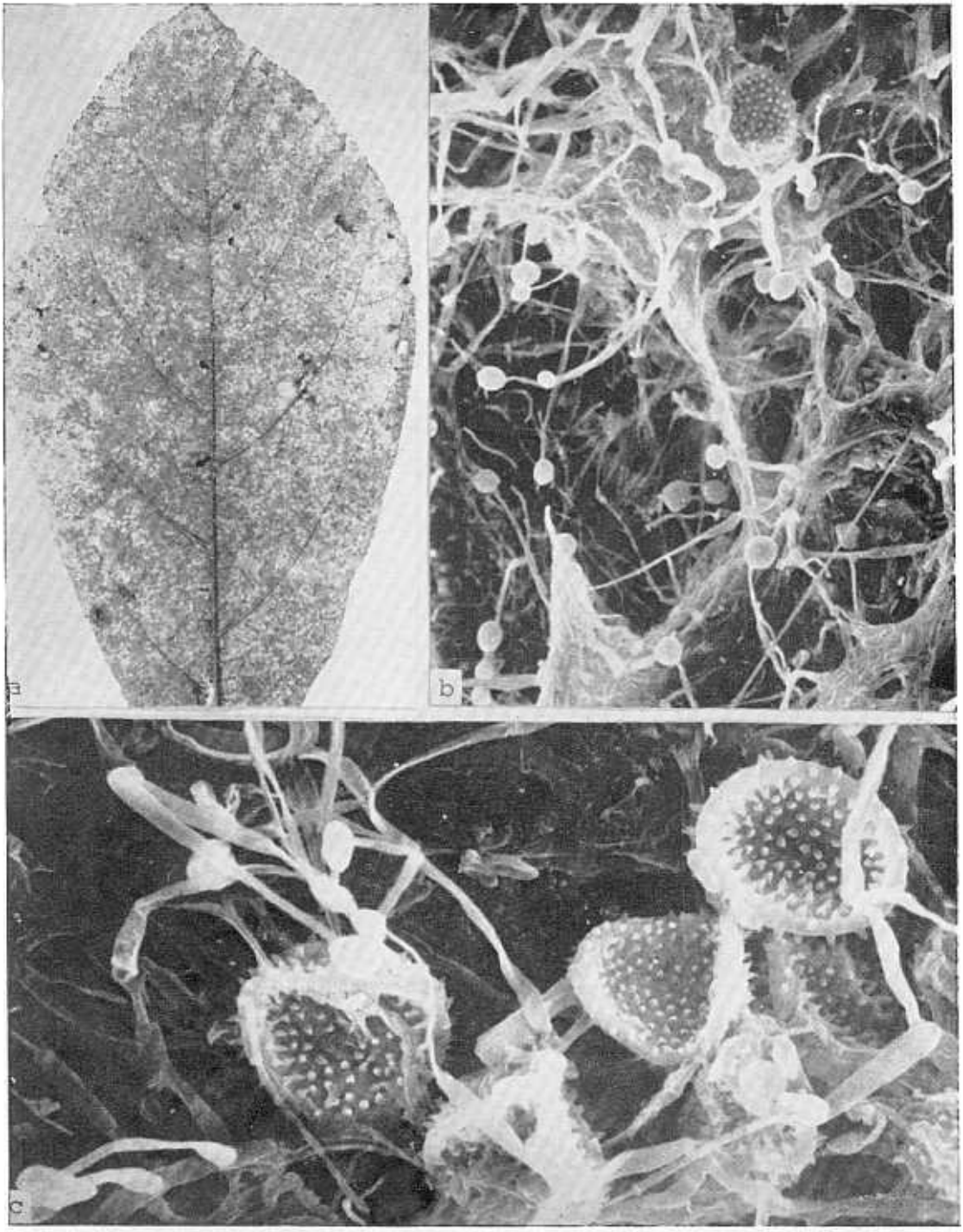


Fig. 6. Hyperparasitism of teak rust by *Acremonium recifei*. a, A leaf showing profuse growth of *A. recifei* over the rust pustules; b and c, SEM showing colonization and sporulation by *A. recifei* on urediniospores of *O. tectonae* (194 X, 1020 X).

formed one to three dark brown concentric rings around the light coloured central spot (Figs. 8a, b).

Table 6. **Severity of leaf spot caused by *Phomopsis Variosporum* in teak plantations at different localities in Kerala during 1980 - 1982**

Sl. No.	Locality	1980			1981			1982		
		% incidence	DSI[a]	DSR[b]	% incidence	DSI	DSR	% incidence	DSI	DSR
1.	Rampur	0.61	1 006	L	52.08	0.20	L	34.67	0.53	L
2.	Kudhirakkode	70.55	D.98	L	56.00	0.81	L	94.97	1.50	M
3·	Mundakkadavu	90.00	2.06	S	93.55	1.95	M	—	—	—
4·	Ezhuthukallu	49.84	0.78	L	43.25	0.83	L	—	—	—
5.	Thundathil	100.00	1.11			1.30	M	97.60	1.83	M
6.	Naduvathumoozhi	0	0			0	Nil	—	—	—
7.	Mampazhathara	99.71	1.42	M	0	0	Nil	—	—	—
8*	Choodal	42.41	0.4	L	99.00	1.48	M	0	0	Nil

aDSI, Disease Severity Index
bDSR, Disease Severity Rating
—, Observations not recorded

Etiology

The pathogen was identified as ***Phomopsis*** sp. (IMI269014). As this isolate differed in morphological characters from the other known species from teak, P. ***tectonae*** (Tiwari *et al.*, 1981), it was designated as a new species of ***Phomopsis*** namely ***Phomopsis variosporum*** sp. nov.

Pathogenicity

Pathogenicity of the isolate was canfirmed on detached leaves, floated on 5 ppm solution of gibberellic acid (GA) in sterile aluminium trays and on twigs, the cut end of which was immersed in GA solution. The conidia were harvested from 10-day-old culture of ***P. variosporum*** and a suspension prepared in sterile distilled water, containing 10 - 15 conidia per drop of Pasteur pipette. Both the surfaces of leaves were washed thoroughly with sterile water and allowed to dry. Small drops of the conidial suspension were placed at five marked spots on the adaxial and abaxial

surfaces of the lamina of the detached leaf as well as leaves attached to twig. The inoculated leaves were transferred to a humidity chamber, maintained at > 95% r. h. The temperature during the incubation period varied between 25 and 28°C.

Minute brown spots appeared on detached as well as attached leaves on the fifth day of inoculation. Characteristic leaf spots with concentric rings developed in 15 to 16 days of incubation. The pathogen was reisolated from the spots in pure culture.

Discussion

Phomopsis leaf spot is the first disease to appear during the late monsoon. Fresh infection persists till the leaves are shed in December/January. The leaf spots cause considerable damage to the photosynthetic area. Where more than 50 per cent of the area is covered with necrotic lesions, leaves are defoliated prematurely. In young plantations, especially where the canopy is almost closed, before the first thinning, Phomopsis leaf spot emerges as the major disease. Usually either in very young (1- to 2-year-old) or old (> 20-year-old) plantations the incidence of the disease was not found to be high.

Phomopsis variosporum sp. nov., causing leaf spot is a new pathogen recorded for teak.

5. PSEUDOEPICOCCUM ZONATE LEAF SPOT

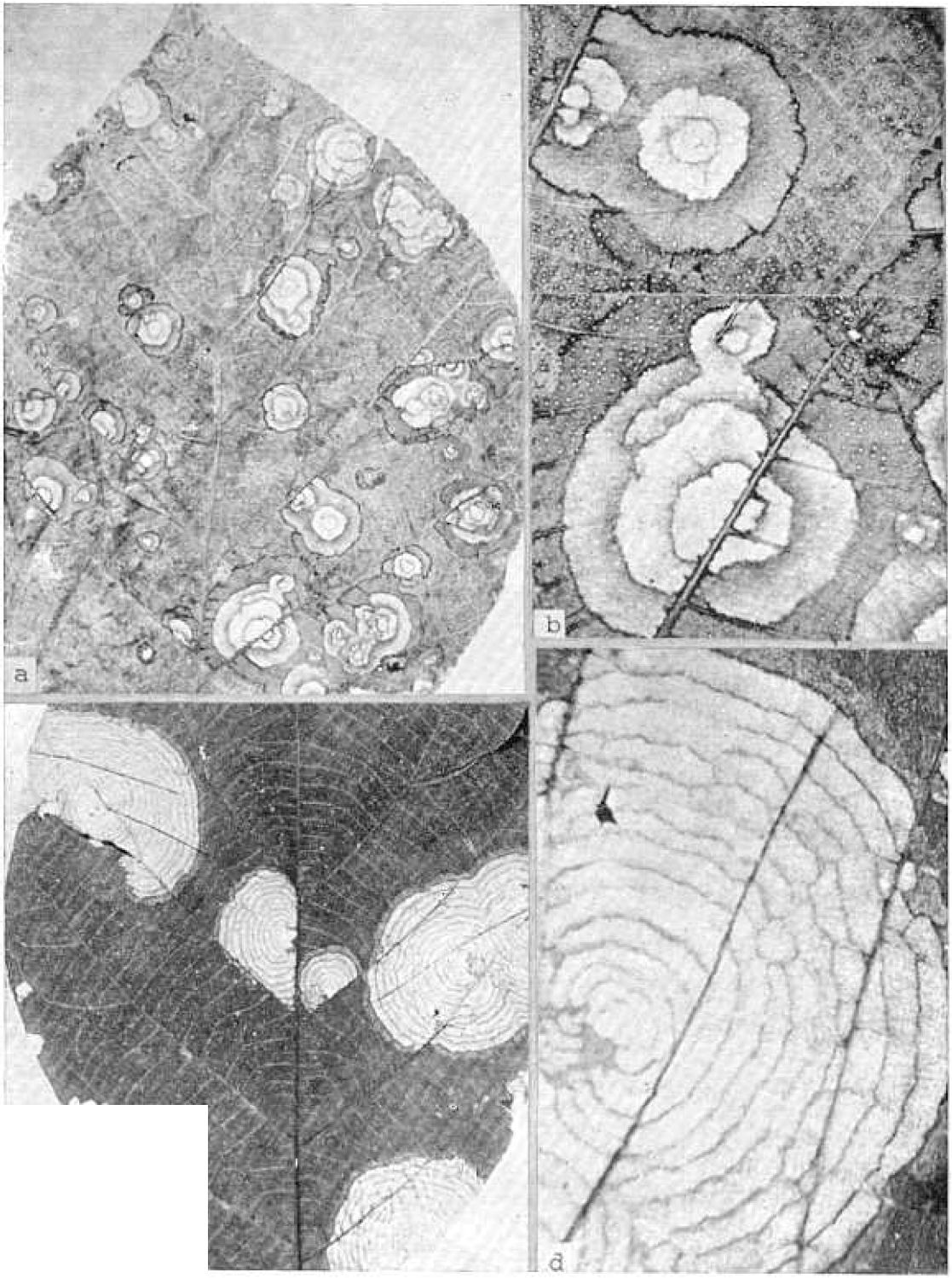
Occurrence and severity

The disease appeared during September/October and continued to affect the leaves till November/December. The disease incidence was usually high in plantations located in humid tracts. Though trees of any age group were attacked youn plants (1- to 4-year-old) were more susceptible than older ones.

In the plantations surveyed high incidence with medium infection, was only recorded at Naduvathumoozhi (Table 7). However, at Kudhirakkode and Ezhuthukallu though the severity was low the incidence of the disease was moderately high; in other plantations both were low. At Thundathil, Mampazhathara and Choodal no infection was recorded. In a given plantation the severity of the disease was not uniform as it was localized in patches, especially where there was a thick weed growth.

Symptoms

The leaf spot was characterised by several well demarcated dark brown concentric rings. Initially minute dark brown spots, 2-3 mm across, developed on the leaves which under high humidity enlarged by adding brown rings, IS–2 mm wide. Subsequently when more rings were added to the spot it gave an appearance


a
b
a
d

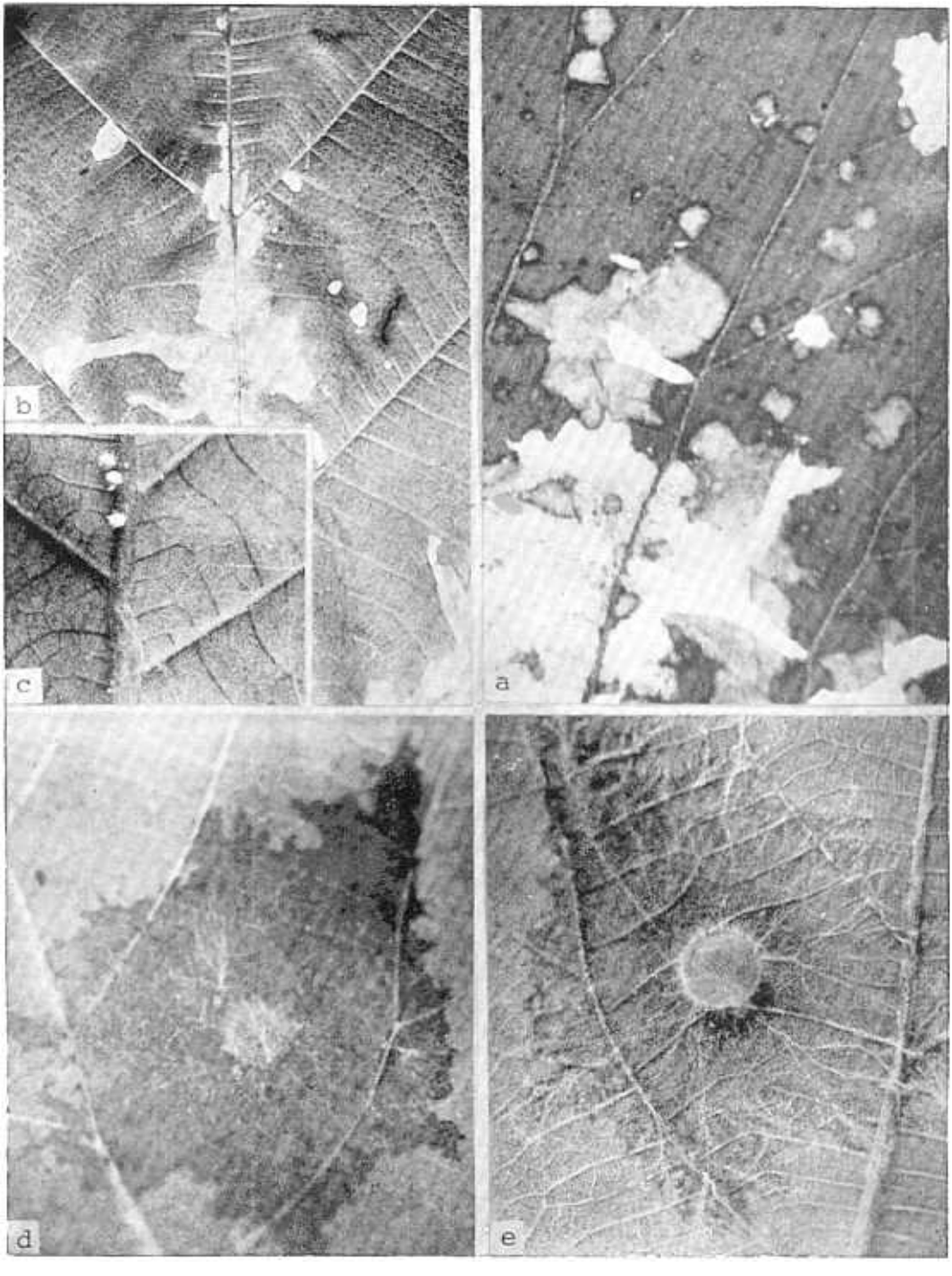


Fig. 9. Leaf spot diseases of teak. a, A part of the leaf infected with *Colletotrichum* state of *Glomerella cingulata*; b, c, d and e, *Sclerotium* leaf spot; b, Typical field symptoms; c, Sclerotia over the infected leaf; d and e, A large spot produced in artificial inoculation test on adaxial (d) and abaxial (e) surfaces.

of the target, hence the name target leaf spot (Figs. 8c, d). Nearby spots coalesced to form large necrotic area on the leaf. Occasionally a single spot covered about half of the leaf lamina. Severely infected leaves were prematurely defoliated, During monsoon, light greyish brown mycelial growth was frequently observed at the margin of the spot on the abaxial side from which the pathogen could be isolated easily.

Table 7. **Severity of leaf spot caused by *pseudoepicoccum tectonae* in teak plantations at different localities in Kerala during 1980-1982**

Sl. No.	Locality	1980			1981			1982		
		% incidence	DSI[a]	DSR[b]	% incidence	DSI	DSR	% incidence	DSI	DSR
1.	Rampur	9.16	0.09	L	1.70	0.01	L	16.63	0.20	L
2.	Kudhirakkode	19.22	0.27	L	56.84	0.87	L	25.35	0.27	L
3.	Mundakkadavu	23.33	0.28	L	20.00	0.62	L	—	—	—
4.	Ezhuthukallu	49.84	0.85	L	30.15	0.78	L	—	—	—
5.	Thundathil	0	0	Nil	0	0	Nil	0	0	Nil
6.	Naduvathumoozhi	98.88	1.53	M	87.37	1.24	M	—	—	—
7.	Mampazhathara	0	0	Nil	0	0	Nil	—	—	—
8.	Choodal	0	0	Nil	0	0	Nil	0	0	Nil

[a] Disease Severity Index
[b] DSR, Disease Severity Rating
—, Observations not recorded

Etiology

Pseudoepicoccum (M. B. Ellis) (IMI 286905). Since this isolate differs morphologically from all the known species of ***Pseudoepicoccum,*** it is described as a new species, ***Pseudoepicoccum tectonae*** sp. nov.

Pathogenicity

Pathogenicity of the isolate was confirmed on detached leaves, floated on GA solution (***5*** ppm), as detailed under Phomopsis leaf spot disease. Characteristic spots with concentric zonations developed after seven days of incubation, The pathogen was reisolated from these spots.

Discussion

Pseudoepicoccum leaf spot, though not widespread, may become a serious problem in young teak plantations with closed canopy, especially those situated in humid tracts.

Pseudoepicoccum tectonae is a new pathogen recorded on teak.

4. COLLETOTRICHUM LEAF SPOT

Occurrence and severity

The disease was of common occurrence in plantations. It usually appeared on mature leaves during the monsoon (July/August) (Figs. 3a, b).

Plantations of all the age groups were equally susceptible to this disease. Medium infection was recorded only at Ezhuthukallu and Choodal in 1980 and Rampur during 1981 (Table 8). In most of the other plantations even though the severity was low, the incidence of the disease was moderate to high. In certain plantations (Rampur and Mampazhathara 1980; Ezhuthukallu 1981) no infection was observed in a given year.

Table 8. **Severity of leaf spot caused by *Colletotrichum* state of *Glomerella cingulata* in teak plantations at different localities in Kerala during 1980-1982**

Sl. No.	Locality	1980			1981			1982		
		% incidence	DSI[a]	DSR[b]	% incidence	DSI	DSR	% incidence	DSI	DSR
1.	Rampur	0	0	Nil	85.66	1.12	M	32.26	0.37	L
2.	Kudhirakkode	84.66	0.27	L	48.63	0.85	L	4.02	0.04	L
3.	Mundakkadavu	65.83	0.97	L	51.25	0.56	L	—	—	—
4.	Ezhuthukallu	72.64	1.19	M	0	0	Nil	—	—	—
5.	Thundathil	21.09	0.2	L	62.09	0.83	L	69.55	0.96	L
6.	Naduvathumoozhi	0	0	Nil	0	0	Nil	—	—	—
7.	Mampazhathara	0	0	Nil	55.76	0.69	L	—	—	—
8.	Choodal	65.52	1.44	M	18.00	0.03	L	0	0	Nil

aDSI, Disease Severity Index
bDSR, Disease Severity Rating
–, Observations not recorded

Symptoms

The leaf spots, irregular, light to dark brown in colour, with a pale margin were usually found on mature leaves. The individual spots coalesced to form large irregular spots (Fig. 9a), which caused drying up of leaves and consequently premature defoliation.

Etiology

Colletotrichum state of ***Glomerella cingulata*** (Stonem.) Spauld. & Schrenk **(IMI 246479, 246480).**

Pathogenicity

The pathogenicity of the isolate was confirmed on detached leaves of teak floated on GA solution as described earlier.

Discussion

Colletotrichum state of ***Glomerella cingulata*** is a common pathogen having a wide host range. Teak is a new host for ***Colletotrichum.***

7. SCLEROTIUM LEAF SPOT

Occurrence

Sclerotium leaf spot of teak, affecting one-year-old saplings in the campus of Kerala Forest Research Institute, was observed during the 1982 monsoon. Lesions were noted on leaves ca. 1 m above the ground. The disease spread very rapidly after appearance and attacked nearby healthy plants. The affected leaves wilted and shed prematurely.

Symptoms

The disease was characterised by the initial dull-brown coloured small leaf spots, 2-3 mm across, which under prolonged wet periods increased rapidly in size irregularly (Fig. 9b), often covering more than one-fourth of the lamina. Frequently when the midrib or petiole was attacked, leaves wilted and dried up; rarely, tender parts of the stem were also found to be infected and growing shoots were killed. A profuse mycelial growth was generally observed on the lower surface of the necrotic area of leaf. Abundant white coloured sclerotia were produced on the mycelial strands(Fig. 9c), becoming dark when mature and dispersed by rain drops and wind to lower leaves, where they caused fresh infections.

Etiology

Sclerotium rolfsii Sacc. (IMI 271722).

Pathogenicity

Pathogenicity of the isolate was tested on leavesof 6-month-old plants in a humidity chamber (>95% r.h.). The upper and lower surface of young and mature leaves were inoculated separately with 5mm mycelial discs taken from the margin of an actively growing culture and sclerotia. The disc was placed with the mycelial side in contact with the leaf surface and the sclerotium in a small drop of sterile water.

Irrespective of maturity of leaf or the surface of inoculation, within 24 h brown circular spots developed around the inoculum with fan-like mycelial strands growing out from the necrotic to healthy tissues. By the third day the leaf spots were 10-15 mm diam. (Fig. 9d), with profuse mycelial growth on the lower surface (Fig. 9e) on which sclerotia of *S. **rolfsii*** developed by the end of the week.

Discussion

Diseases caused by ***S. rolfsii*** occur mainly in tropical and subtropical areas, with high moisture and temperature. Although ***S. rolfsii*** is best known as a parasite of stems, it is apparently able to infect any part of a susceptible plant under favourable environmental conditions (Aycock, 1966). This is the first report of the pathogen on teak, and the first record on leaves in India, although foliage infection has been reported from the USA on soybean (Lehman *et al* , 1951) and ***Alleurites fordii*** (West, 1936), from Indonesia on ***Hibiscus sabdariffa*** (Reitsma and Sloof, 1950), and from Ivory Coast on ***Dioscorea*** spp. (Baudin, 1956).

8. PINK DISEASE

Occurrence and severity

Pink disease of teak was usually observed in young plantations (1- to 5-year-old). Besides the plantations surveyed it was also recorded at Kodanad (Malayattoor Div.), Nilambur (KFRI Sub Centre), Neriamangalam (Kothamangalam Div.) and Kissimum (Ranni Div.). Occasionally pink disease infection was also found on branches of older trees at Kudhirakkode, Tholpetty (Wynad Div.) and Neriamangalamm (Kothamangalam Div.). The disease was prevalent during the monsoon period (June-September) (Figs. 3a, b).

Pink disease was recorded in all the plantations surveyed, except in plantations at Mundakkagavu and Naduvathumoozhi (Table 9). Though the per cent incidence and severity of pink disease was low it showed an upward trend in all the plantations with the exception at Ezhuthukallu where it decreased. The highest incidence of the disease was observed at Choodal during 1982 (31.61 per cent) and in a 2-year-old,plantation at Thundathil (> 20.8 per cent).

Table 9. **Severity of pink disease caused by *Corticium salmonicolor* in teak plantations at different localities in Kerala during 1980 - 1982**

Sl. No.	Locality	1980			1981			1982		
		% incidence	DSI[a]	DSR[b]	% incidence	DSI	DSR	% incidence	DSI	DSR
1.	Rampur	4 07	0.07	L	5.42	0.17	L	6.27	0.25	L
	Kudhirakkode	8.08	0.13	L	32.42	0.13	L	13.88	0.16	L
2 3:	Mundakkadavu	0	0	Nil	0	0	Nil	—	—	—
4.	Ezhuthukallu	7.82	0.08	L	1.05	0 . 0 3	L	—	—	—
5.	Thundathil		0.08	L	11.43	0.I2	L	12.82	0.16	L
6	Naduvathumoozhi	0	0	Nil	0	0	Nil	—	—	—
7.	Mampazhathara	7'27 13.12	0.13	L	19.90	0.18	L	—	—	—
8.	Choodal	3.35	0.05	L	17.56	0.09	L	31.61	0.17	L

aDSI,Disease Severity Index
bDSR,Disease Severity Rating
–, Observations not recorded

Symptoms

The disease was characterised by a pink encrustation over a canker, formed at the site of infection on the stem. The infection occurred at any place on the main stem, which resulted in killing of inner bark comprising of phloem and cambial tissue and subsequent development of a canker. When the stem was completely girdled by the invading mycelium, the portion of shoot above the canker was killed outright. The bark at the canker showed longitudinal splitting during the dry weather (Fig. 1Oc). Just below the canker epicormic shoots developed, one of which usually became the leader shoot in the following season.

Etiology

Corticium salmonicolor Berk. & Br.

The first sign of the infection on the stem was the development of cobweb stage during the monsoon. Soon pustules, small pin head size white mycelial bodies, developed over the cobweb. The bark of this area got depressed and showed browning. At this stage leaves of the affected shoots wilted due to killing of the tissues of inner bark. The perfect stage, characterised by pink encrustation, developed

over the dead tissue. Numerous club shaped basidia with four sterigmata produced air–borne basidiospores, a source of inoculum for fresh infection. No necator stage was observed on teak.

Discussion

The pink disease of teak commonly occurs in all the areas of the State which receive high rainfall (ca. 3000 mm p. a.) (Figs. 3a, b). The disease may become serious in 1- to 3-year-old teak plantations where the terminal shoot is killed, consequently affecting apparently the height growth of the infected trees. High incidence of the disease has been observed in teak plantations situated near rubber plantations, which form the permanent source of inoculum.

Pink disease of teak has been recorded from Karnataka, India (Bakshi, 1975) and Indonesia (Schwarz, 1925). This is the first record of the disease from Kerala.

9. **DIE-BACK**

Occurrence

The die-back of teak was noticed in a 40-year-old plantation at Palappilly (Chalakudy Div.). Later it was also found to occur at Vellikulangara (Chalakudy Div.), Peechi (Trichur Div.) and other places. But high incidence of the disease appeared to be localized in Trichur Div. and Chalakudy Div. Apparently, trees more than 10-year-old were only found affected. The severity of the disease varied greatly within a plantation, localised more or less in pockets. The spread of the disease within a tree and from tree to tree was facilitated by an insect vector.

Symptoms

After initial insect attack at the base of the branch near the main stem (Fig. 11a) the lower side branches were killed. Besides decay in the wood, partial girdling of the inner bark due to tunnelling was possibly also responsible for the death. Usually dead branches snapped and fell off from the area of intense insect attack, which could be seen in the form of a sieve of large number of holes, more or less in a circular fashion (Figs. 11 a,d). In due course of time, such areas characterised by a group of holes, were found all over the stem, except at the lower region, one to two meters above ground. Subsequently, a number of epicormic shoots developed. But as the insect attack increased, gradually all the branches showed die-back and later the whole tree succumbed (Figs. 11c,b)

The death of the tree, which possibly may take between 10-15 years depending upon the age at which the infection started and intensity of insect attack, was primarily due to the fungal decay affecting sap as well as heartwood. Infection in

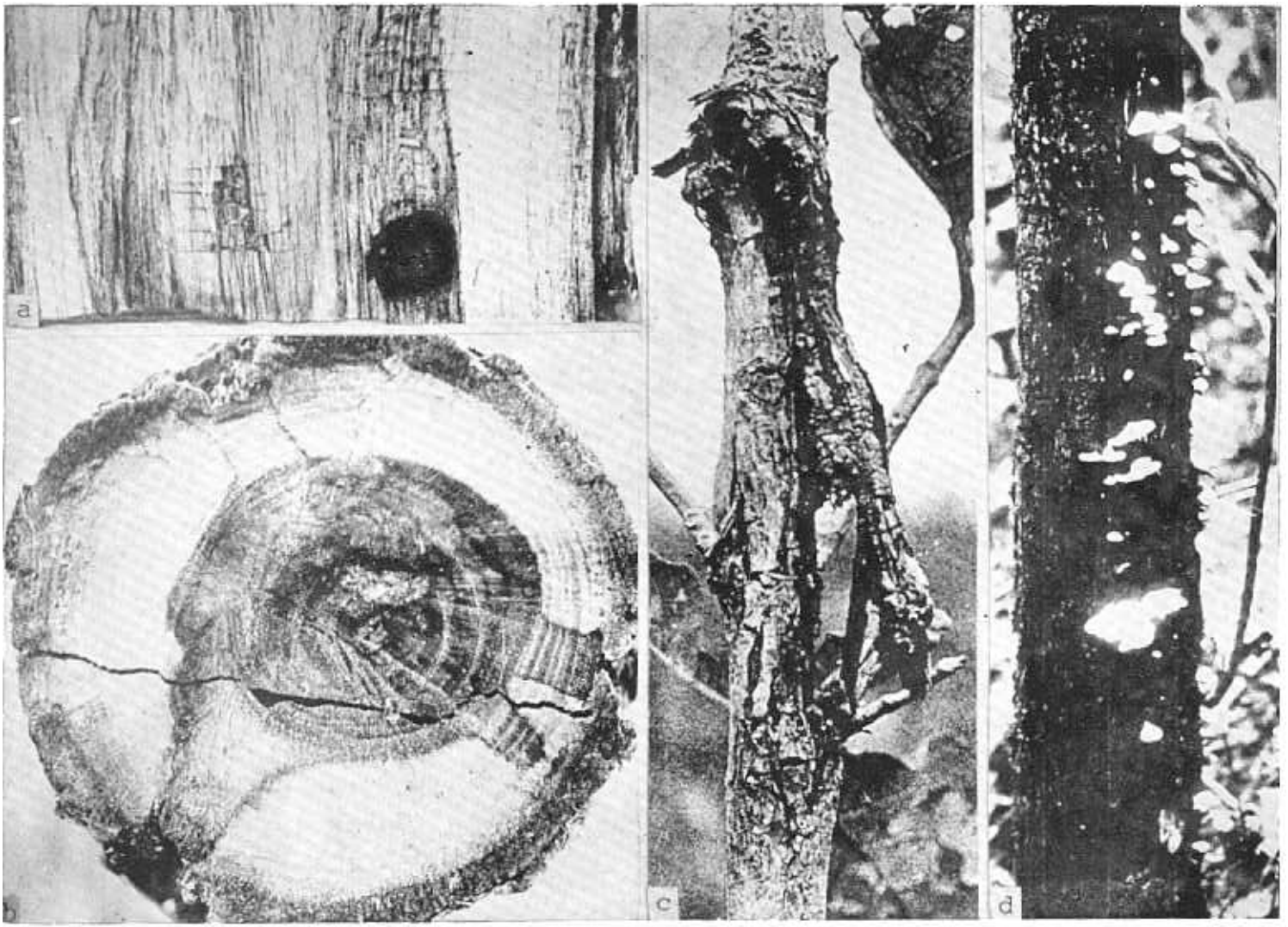


Fig. 10. Stem diseases of teak. a and b, Artificial inoculation test showing pathogenicity of *Phialophora richardsiae* causing die-back — a, longitudinal section; b, transverse section; c, A stem canker caused by *Corticium salmonicolor* in 2-year-old teak; d, Fructifications of *Marasmiellus ignobilis* causing stem rot.

Fig. 11. Die-back of teak caused by *Phialophora richardsiae*. a, A tree partially infested with *Cossus cadambae* showing a dead branch hanging downward, snapped due to infestation; b, The epicormic shoots produced are killed due to stem decay; c, A dead tree severely attacked with *C. cadambae* and wood completely decayed; d, A magnified view of the holes of the tunnels (arranged in the circular fashion) centered around the base of a branch, which gets detached due to intense insect attack; e, The initial insect attack at the base of the branch near the main stem.

the wood was initiated from the larval galleries or tunnels, sides of which appeared dirty dark brown in colour, The browning extended laterally as well as up and downwards in the wood; it even spread in the lower portion of the trunk, where no insect attack was found. As and when the whole. trunk was girdled. due to decay and insect tunnelling, the tree died.

Insect vector

Cossus cadambae Moore. was found to be responsible for the entry of the fungus in the wood. It is a known borer of living teak trees, producing tunnels in the wood. Eggs are laid at snags or other wounded areas of branches. The larvae, which survive for about 10-11 months, are red in colour and measure about 3-4 cm in length when full grown. Larva bores tunnels into the wood of the side branches, especially near the joint of branch with the main stem. From the tunnel it feeds on the wood.

Etiology

Phialophora richardsiae (Nannf.) Conant (IMI 257551). The fungus was also recovered in 40 per cent of the isolations from the larvae of C. ***cadambae.***

Pathogenicity

Pathogenicity of ***P. richardsiae*** was tested on 10 branches, about 8 - 10 cm in diameter, of healthy 12-year-old teak trees at Palappilly. Four holes (2 cm deep of 5 mm diam), at right angles of each other on the branch near the main stem, were drilled using a sterile auger. Before drilling, the bark was cleaned with 95% ethyl alcohol followed by sterile water. Each hole was inoculated with two agar discs coataiding mycelia and abundant conidia of ***P. richardsiae*** and the hole plngged with a moist sterile cotton. The plug was sealed with bees wax. Appropriate controls were also maintained. Observations on the deveIopment of external as welt as internal symptoms of the disease were made regularly.

After two months of inoculation the browning in the wood had spread 7 - 10 cm in both the directions. This increased to 15 - I7 cm in the following month. During this period no external symptoms of the disease were observed. At the end of six months, decay had extended to about 20 - 25 cm on both the sides of the inoculated site and more than three-fourth (Figs. 10 a, b) of the'wood, in cross section was found to be affected. One branch also showed die-back symptoms confirming the role of ***P. richardsiae*** in teak decline. The pathogen was consistently isolated from the affected wood. During the observation period controls remained unaffected and the hole got covered wit4 the callus growth.

Discussion

From the inoculation trials it becomes clear that the die-back disease caused by ***P. richardsiae*** spreads very slowly within the tree and it may be about 10-15 years before the mortality of the trees was caused. This also gets support from personal enquiries from the local people living in the viscinity of the affected plantations. Since the pathogen needs the help of the vector, ***C. cadambae,*** for entry into the host, the latter plays an important role in the spread and for attaining a specific level of severity of the disease in a given locality.

Earlier, Beeson (1941) found that the attack of borers was limited to trees of poor quality, which are unhealthy or badly lopped. However, our observations indicate insect attack even in very healthy robust trees, especially where there was no lopping or no mechanical injury. Nevertheless, lopping of branches which leaves open wounds possibly facilitates increased insect activity.

As far as the cause of death of the tree is concerned, possibly girdling of the tree at different levels due to extensive tunnelling above may not be responsible. It is the infection of ***P. richardsiae,*** which spreads from the tunnels and invade the living sapwood as well as the heartwood, that kills the tree already weakened due to insect attack. In this way insect infestation accentuates the death process. Curiously, it is to be noted that often there is no insect attack on the trunk about one to two meters above the ground but still the whole tree dies even without producing any epicormic shoots from the base of the stem. The reason is the decay of wood which also extends to this lower part of the trunk.

This is the first report indicating the role of ***P. richardsiae*** in causing decay in teak. 'Earlier, Singh and' Tiwari (1970) and Bakshi (1975) have shown that prior colonization of teak by ***Phialophora*** sp,, a non-decay fungus, conditions the wood to make it more susceptible to attack by decay fungi ***(Fomes lividus*** (Kalchbr.) Sacc. and ***Polyporus zonalis*** Berk.).

10. STEM ROT

Occurrence

Stem rot of teak was observed only in one 15-year-old plantation at Thrissillery (Wynad Div.). The disease was found in an area having high soil moisture as it was situated near a perennial stream. The incidence of the disease was <1%. No mortality of trees was recorded.

Symptoms

Generally the infection occurred on one side of the trunk near the ground. The affected area became soft and spongy with profuse mycelial growth, During

the rainy season numerous white to pale cream coloured basidiocarps (fructifications.) developed on the affected area (Fig. 10 d), measuring 30 - 75 om in length.

Etiology

Marasmiellus ignobilis (Berk. & Br,) Pegler.

Pathogenicity

The pathogenicity of the isolate was tested i n ,April 1982 on healthy teak trees at Thrissillery, where the disease was' observed. One-month-old inoculum of ***M. ignobilis,*** raised on saw dust corn meal medium, was utilized for inoculation, with and without wound. For the former, using a sharp chisel two centimeter square area of bark was removed to form a cavity with sapwood exposed. The inoculum was placed in this cavity and the bark piece replaced. The cut areas of the bark were sealed with bees wax. For inoculating trees without wound the inoculum was placed over the bark and covered with cotton swab which was covered with bees wax. Appropriate controls were also maintained. Observatians were recorded after six months following the monsoon.

In wound inoculation the infection had spread to about 25 cm^2 area. The affected area of bark and sapwood became soft and covered with profuse mycelial growth and fructifications also developed, thus confirming the pathogenic nature of ***M. ignobilis*** No infection occurred either in unwounded inoculations or controls.

Discussion

Teak is a new host record for ***M. ignobilis*** from India. The disease appears to be unimportant as it was recorded only at one place and with low incidence.

11. PHANEROGAMIC PARASITE (MISTLETOE)

Occurrence and Severity

Though mistletoe infestation is widespread in northern and central Kerala, during the survey it was recorded only at Mundakkadavu (Nilambur Div.) where about 53 per cent trees were infested; none of the trees died due to mistletoe. In some other plantations at Nilambur even > 80(percent of the trees were found to be attacked by the angiospermic parasite.

Etiology

Dendrophthoe falcata (Linn. f.) Ettingsh var. ***pubescens.***

D. fulcata is a woody, highly branched, evergreen,. semi-parasitic shrub attached to the branches of teak through a swollen base, called holdfast. The parasite usually attacked only the side branches of the' tree and never the main stem. The

mistletoe clump, as the parasite commonly known, usually hangs down from the branches.

Control measures

At present a practice of mechanical removal of the parasite is followed in heavily infested teak plantations in Kerala. But the efficacy and implementation of the operation have not been found to be very satisfactory due to numerous reasons, Ghosh *et. al.* (1984) attempted the chemical contol of ***D. falcata*** through trunk injection and reported Sencor (Metribuzin) (BASF) as a promising herbicide capable of killing the mistletoe clumps selectively.

Discussion

No mistletoe infestation was recorded in most of the plantations surveyed, except at Mundakkadavu in Nilambur Div. The reason for the absence of infestation could be the young age of teak trees. Ghosh *et al.* (1984) have reported the mistletoe infestation in teak plantations above the age of seven years. At Naduvuthumoozhy where no infestation was observed, the age is not the factor but it is possibly due to the distribution pattern of ***D. falcata*** in Kerala. The present observation is in conformity with Ghosh *et al.* (1984) that the parasite infestation is more in the Northern and Central Forest Circles than in Southern Forest Circle and high ranges.

DISEASES OF UNKNOWN ETIOLOGY

1. MOSAIC

Occurrence

A disease, very similar to mosaic virus disease was observed in 6- to 18-month-old nurseries at Angamoozhi (Ranni Div.), Chethaleth (Kozhikode Div,), Kariampanny (Wynad Div.) and in young plantations (1- to 2-year-old) at Rampur (Kozhikode Div.), Cheruvancherry, Kannoth (Wynad Div.).

Symptoms

The affected plants were easily distinguishable in the nurseries or plantations due to their mottled leaves. The affected leaves had a number of small to large irregular creamy white to light pale irregular spots, which gradually coalesced to form large chlorotic areas, characteristic of mosaic disease (Fig. 12 a). Mosaic symptoms were also seen in very young leaves. Often only a few of the leaves of a plant either had only half of the lamina with mosaic symptoms and rest with normal colour or only some of the leaves in a seedling showed mosaic symptoms while the others were normal. No crinkling of leaves and stunting of plants were observed.

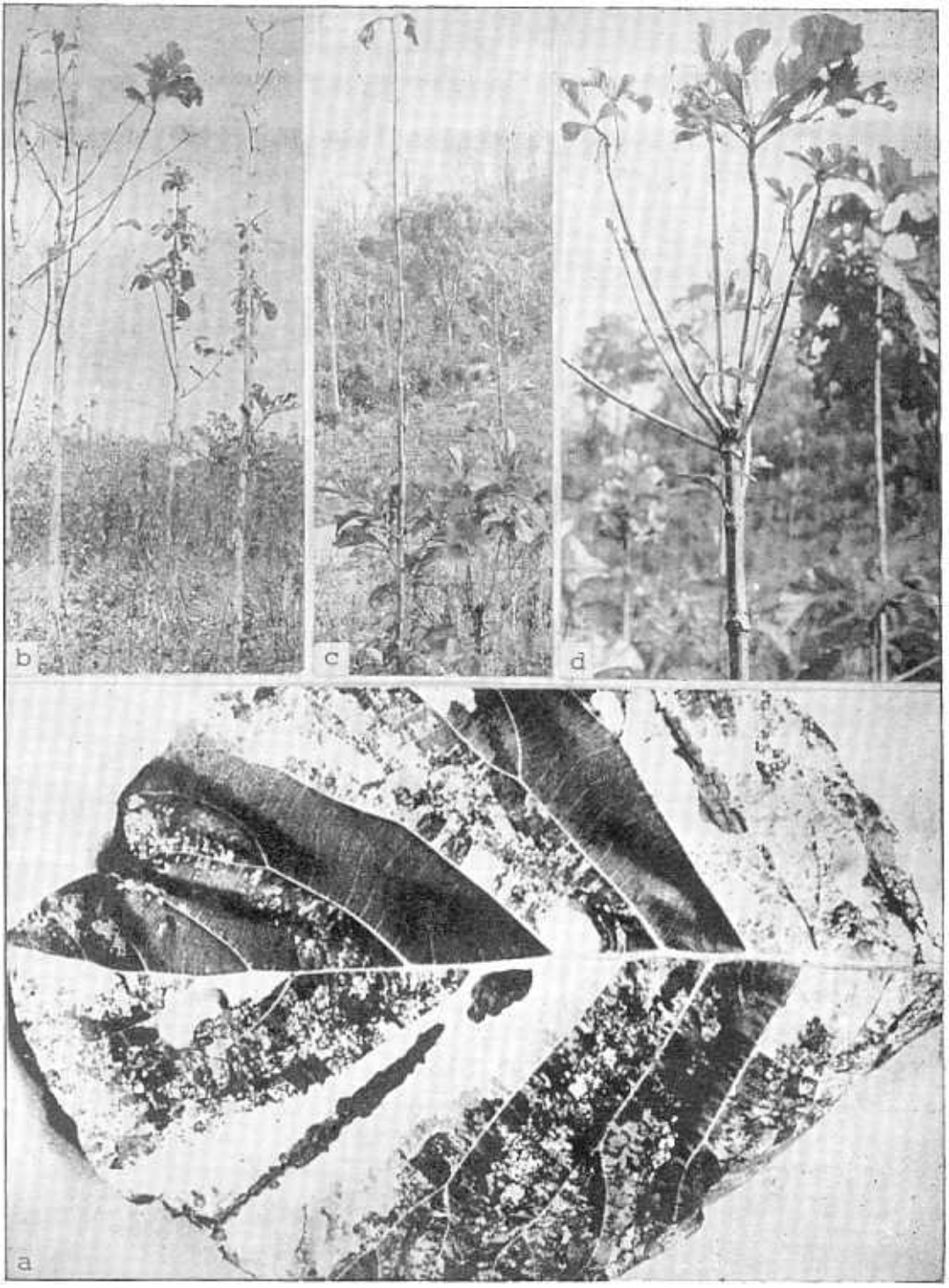


Fig. 12. Diseases of unknown etiology of teak. a, Leaf mosaic; b, c, and d, Different stages of stunting, defoliation and shoot die-back.

Discussion

In the absence of transmission studies and electron microscopy it was not possible to identify the cause of this disease. As the incidence of the disease was below 0.1 per cent it appears to be unimportant. This is the first report of a mosaic disease in teak.

2. VEIN CLEARING AND YELLOWING OF INTERVEINAL AREAS OF LEAVES

Occurrence

This disease has been observed occasionally in patches in young plantations (1- to 5- year-old). The highest incidence was observed at Kissimum (Ranni Div.) with about 10 per cent of the plants affected.

Symptoms

The affected plants either showed vein clearing, especially of major veins or prominent yellowing or both of the intervenial area of the- lamina. No other abnormalities of leaves or stunting of plants were observed. The symptoms first appeared in the older leaves and proceeded towards the apex.

From the symptoms it appeared to be a deficiency disease.

3. STUNTING AND SHOOT DIE-BACK

Occurrence

Stunting of shoots followed by die-back of branches was observed in 2- to 4-year-old plantations at Thundathil (Malayattoor Div.) and 3-year-old plantations at Kudhirakkode. Begur and Rampur (Kozhikode Div.). These symptoms were widespread in the above plantations, being severe at Thundathil.

Symptoms

The affected plants had stunted internodes and smalling of leaves (Fig. 12)b) The leaves on these plants turned yellow and defoliated prematurely (Fig. 12 c). At Thundathil these symptoms were accompanied with profuse branching of shoots (Figs. 12 b, d). The branches which remained stunted later developed die-back symptoms.

Etiology

From the dead shoots though ***Colletotrichum gloeosporioides*** was isolated its pathogenicity in causing die-back could not be confirmed.

Discussion

Most of the affected trees in these plantations showed stunting of shoots. Profuse branching possibly indicates loss of apical dominance due to some

physiological and/or soil factores. Since the disease would have a considerable impact on yield a multidisciplinary study on the cause of this disease is warranted.

GENERAL DISCUSSION

For a long rotation crop like teak, grown extensively in Kerala, during the survey relatively afew diseases and pathogens associated with them were found. From other teak growing countries also so far only a few diseases have been recorded

Table 10- Checklist of diseases of ***Tectona grandis*** recorded in Kerala

Sl. No.	Disease	Pathogen/parasite	New pathogen record for teak	First record of pathogen from India
	A. *NURSERY*			
1.	Bacterial collar rot	***Pseudomonas*** sp. (possibly ***P. solanacearum)***		
2.	Rust	***olivea tectonae*** **(T.S.** & **K.** Ramakr.) Mulder		
	B. PLANTATION			
	(i) ***Parasitic***			
1.	Bacterial wilt	***Pseudomonas*** **sp.** (possibly ***P. solanacearum)***		
2.	Powdery mildew	***Uncinula tectonae*** Salm**.**		
3.	Rust	***Olivea tectonae***	−	−
4.	Phbmopsis leaf Spot	***Phomopsis variosporum*** sp. **nov.**	+	+
5.	Pseudoepicoccum leaf spot	***Pseudoepicoccum tectonae*** sp. nov.	+	+
6.	Colletotrichum leaf spot	***Colletotrichum*** state of ***Glomerella cingulata*** (Stonem.) Spauld. & Schrenk	+	−
7.	Sclerotium leaf spot	***Sc/erotium rolfsii*** Sacc.	+	−
8.	Pink disease	***Corticium salmonicolor*** Berk. & Br.		−
9.	Stemrot	***Marasmiellus ignobil(*** Berk. & Br.) Pegler	+	+
10.	Die-back	***Phialophora richarasiae*** (Nannf.) Conant	+	+
11.	Phanerogamic parasite	***Dendrophthoe falcata*** (L. f.) Ettingsh	−	−
	(ii) ***Unknown etiology***			
12.	Mosaic			
13.	Vein clearing and yellowing of interveinal areas			
14.	Stunting and shoot die-back			

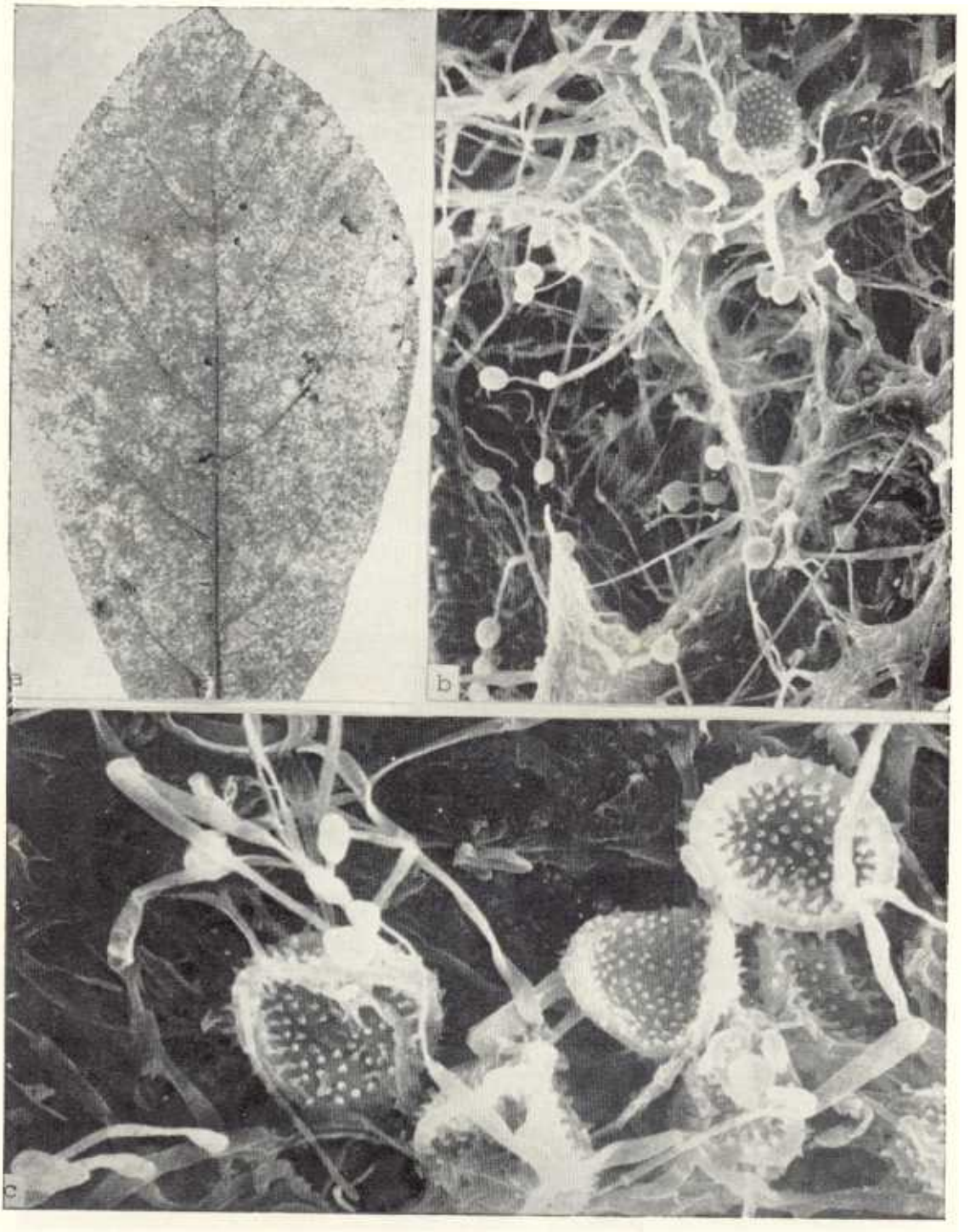


Fig. 6. Hyperparasitism of teak rust by *Acremonium recifei*. a, A leaf showing profuse growth of *A. recifei* over the rust pustules; b and c, SEM showing colonization and sporulation by *A. recifei* on urediniospores of *O. tectonae* (194 X, 1020 X).

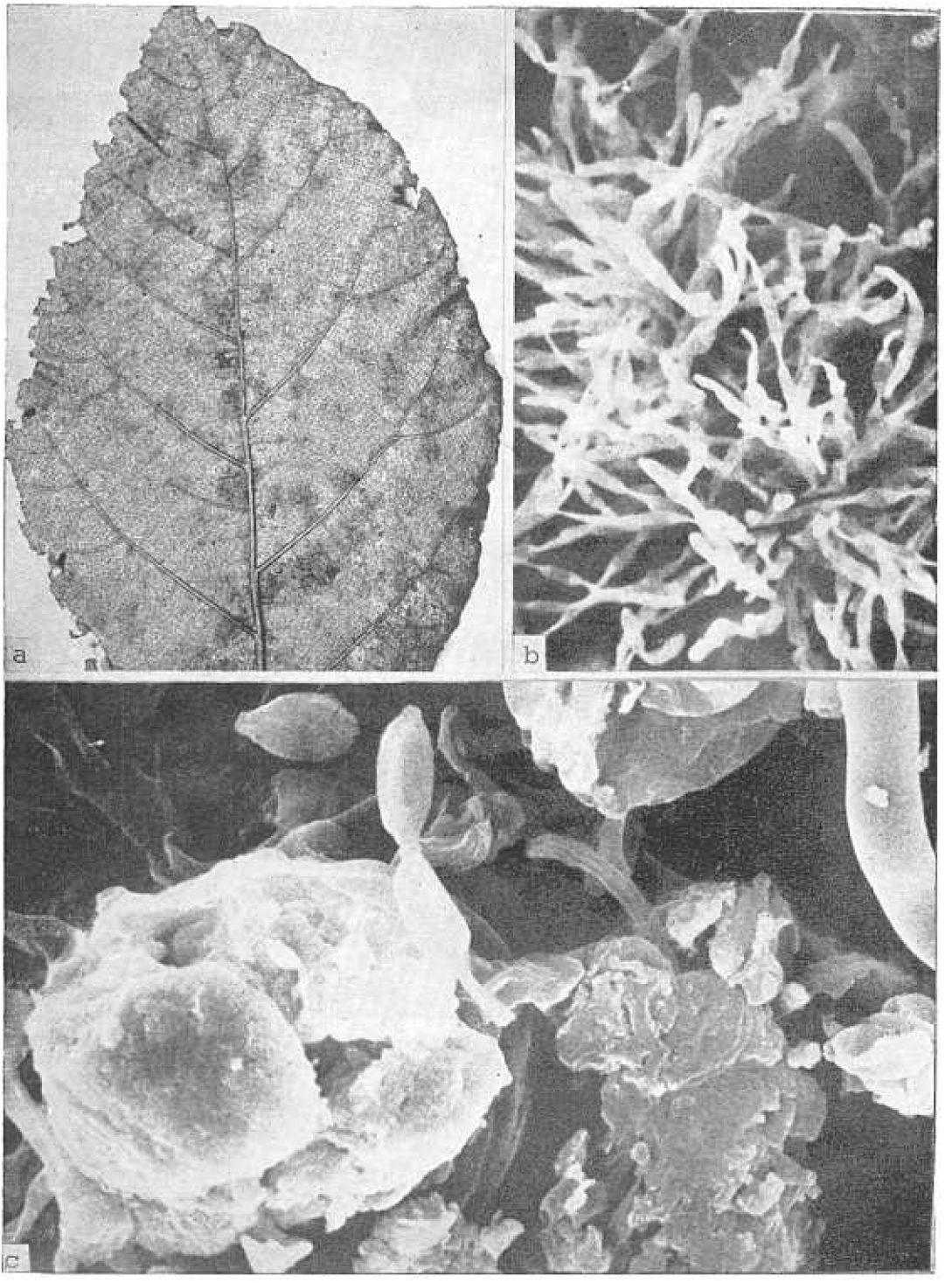


Fig. 7. **Hyperparasitism of teak rust by *Cladosporium oxysporum.* a, A leaf showing profuse dark growth over the rust pustules; b, SEM of a sporulating colony of C. *oxysporum* (380 X); c, Urediniospores parasitized by C. *oxysporum* (2050 X).**

on teak (Gibson, 1975). This possibly reflects the hardy nature of this species. Bakshi (1975) has earlier reported only three diseases i. e., rust, an angiospermic parasite ***(Dendrophthoe falcata*** (L. f.) Ettingsh) and a physiological disorder - water blister disease from Kerala. During this survey a total of 15 diseases were found, of which one (Bacterial wilt) is a new report from India and six others are new pathogenic diseases reoorded for the first time in teak; three diseases of unknown etiology are also new reports (Table 10). Of the total of ten pathogens associated with various diseases, four including one new species each of ***Pseudoepicoccum*** and Phomopsis and ***Marasmiellus*** **ignobilis** and ***Phialophora richardsiae*** are first records from India.

In nurseries none of the diseases were of serious nature, However, leaf rust by causing premature defoliation in young seedlings may pose some problem in dry tracts of the State, In plantations, only a few of the 14 diseases recorded such as die-back caused by insect-fungus complex, pink disease, mistletoe infestation, stunting and shoot die-back (unknown etiology) and Phomopsis leaf spot appear to be of serious consequences.. Though the high severity of most of these diseases was localized in some areas, they are capable of spreading in epidemic form.

Curiously, no heart rot or root diseases, other than Bacterial wilt, which are of common occurrence in other parts of India (Bakshi *et al.,* 1972) and other teak growing countries (Gibson, 1975) and water blister in teak (Bakshi and Boyce, 1959) were recorded during this survey.

Since chemical control of diseases is not economically feasible in forest plantations, incidence of some of the foliar diseases and bacterial wilt could be minimized by proper management practices, such as thinning of branches, opening of canopy; proper weeding and soil operations

BOMBAX CEIBA

Bombax ceiba L., distributed throughout India, occurs specially in moist-deciduous forests. Being an important softwood species it is raised in plantations as well as in homesteads and farmlands. In Kerala, it is usually grown in 'soft wood plantations', mixed either with teak or ***Ailanthus triphysa.***

A list of representative plantations of ***B. ceiba*** surveyed is given in Table 11.

Table 11. List of representative plantations of *Bombax ceiba* surveyed for incidence and severity of diseases during 1980 - 1982

Sl. NO.	Locality	Altitude (m above msl)	Forest Divn.	Area of (ha)	No. of observation plots surveyed	Year of planting	trees at survey (years)
1.	Kannoth*	200	Wynad	51.0	3	1974	6
2.	Thundathil**	15	Malayatoor	64.48	5	1975	5
3.	Kuttampuzha*	80	Malayatoor	—	5	1976	4
4.	Mampazhathara**	150	Konni	49.85	3	1974	6
5.	Choodal**	200	Thenmala	—	5	1977	3

* **Mixed 'soft wood' plantation with *Ailanthus triphysa***
** **Mixed 'soft wood plantation' with *Tectona grandis***

NURSERY DISEASES

Leaf rust caused by *Uredo bombacis* and a leaf spot caused by *Colletotrichum gloeosporioides,* which were common to nurseries and plantations are dealt with under plantation diseases.

1. COLLAR ROT

Occurrence

The disease, resembling damping-off, was recorded in 1- to 2-month-old seedlings of *B. ceiba* at Peechi. The disease, first noticed in isolated patches spread fast in all seedbeds affecting a large number of seedlings. More than 50 per cent of the seedlings died due to this disease within a month.

Symptoms

The initial symptom of the disease was the appearance of water soaked lesions on the hypocotyle. Later these lesions turned brown in colour and the infected area got decayed (Fig., 13'a). Affected seedlings usually collapsed from the decayed portion, fell over the ground and died.

Etiology

***Rhizoctonia* solani** Kuhn state of *Thanarephorus cucumeris* (Frank.) Donk (IMI 280235).

Pathogenicity

Pathogenicity of ***R. solani*** was tested on 6- and 8-week-old seedlings of ***B. ceiba*** grown in sterile soil. The inoculum was ,raised on sand corn meal medium in large culture bottles. Mycelial mats with abundant microsclerotia, harvested from I-month-old,cultures, were air dried for 12 hours and blended in a waring blender. One gram of this powdered inoculum was mixed thoroughly with 4 kg of sterile soil. This soil was transferred to two sterile aluminium trays (30X 30X 5cm) and incubated for one week in the laboratory. Fifteen test seedlings of ***B. ceiba*** were transplanted in each tray. Control seedlings were maintained in sterile uninfested soil. All the trays were transferred to a humidity chamber with > 95% r. h. and temperature 20-31°C. Observations on the appearance of disease symptoms were recorded every day.

Typical symptoms of collar rot developed on the second day of incubation. On the following day majority of 6-week-old seedlings died, while 8-week-old ones took another four days to succumb to infection. The pathogen wasreisolated from the infected seedlings.

Control measures

Emisan-6 was found to be most effective against ***R. solani*** (see collar rot under ***Alianthus*** triphysa). Collar rot of ***B. ceiba***was controlled by drenching.the seedbeds twice with Emisan-6 (0.0025% a. i.) at an interval of ten days, Since the disease is manifested under high soil moisture regimes, the watering frequency. as well as quantity per bed should be reduced after the appearance of the disease to check its further spread.

Discussion

R. solani is a common facultative parasite known to cause ,varied kinds of diseases in a number of hosts. Collar rot of ***Bombax*** is a new disease record and a new host record for the pathogen.

2. LEAF BLIGHT

Occurrence

The disease was first observed during the monsoon of 1983 (June-July) affecting seedlings of ***B. ceiba*** and ***B. insigne*** in a nursery raised at Peechi. Usually severe leaf blight was followed by stem infection which killed the seedlings. In some of the seedbeds as many as 25 per cent of the seedlings died.

Symptoms

The initial symptoms of the disease were the appearance of small circular brownish-yellow spots in concentric rings on the leaflets. These spots increased in size and coalesced to form large necrotic areas (Fig. 13b), which often covered the entire leaf and even the petioles (Fig. 13c) Soon, because of rotting of petioles, the leaves bent downwards and dried up. The infection spread rapidly through contact of diseased leaves with healthy ones (Fig. 13 d) causing extensive premature defoliation. Contact with stem caused stem decay which resulted in death of seedlings. On the affected leaves and stem numerous off-white sclerotia developed (Figs. 13c, d) which fell over the ground after getting detached easily from the mycelium.

Etiology

Corticium rolfsii Curzi (IMI 280236, 280239).

Pathogenicity

Pathogenicity of the isolate was tested on 6-week-old potted seedlings of *B. ceiba.* The leaves were inoculated by placing a sclerotium, obtained from 15-day-old culture, in a drop of sterile water. In a separate experiment ten seedlings were transplanted to an aluminium tray (30 x 20 cm) containing sterile soil. The seedlings were allowed to establish for about a week. Five sclerotia were placed one cm apart, 2-3 mm below the soil surface around the root collar region of each seedling. All the seedlings were kept in a humidity chamber where r. h. was > 95% and temperature ranged between 20 and 30°C.

None of the sclerotia in the soil germinated and caused any infection. However, on leaf the sclerotia germinated and produced profuse white cottony mycelium on the third day of inoculation. On the following day, typical brownish yellow spots appeared and the infection spread from the leaves to the stem. Affected seedlings died within six to seven days of inoculation; abundant sclerotia developed on the dead seedlings.

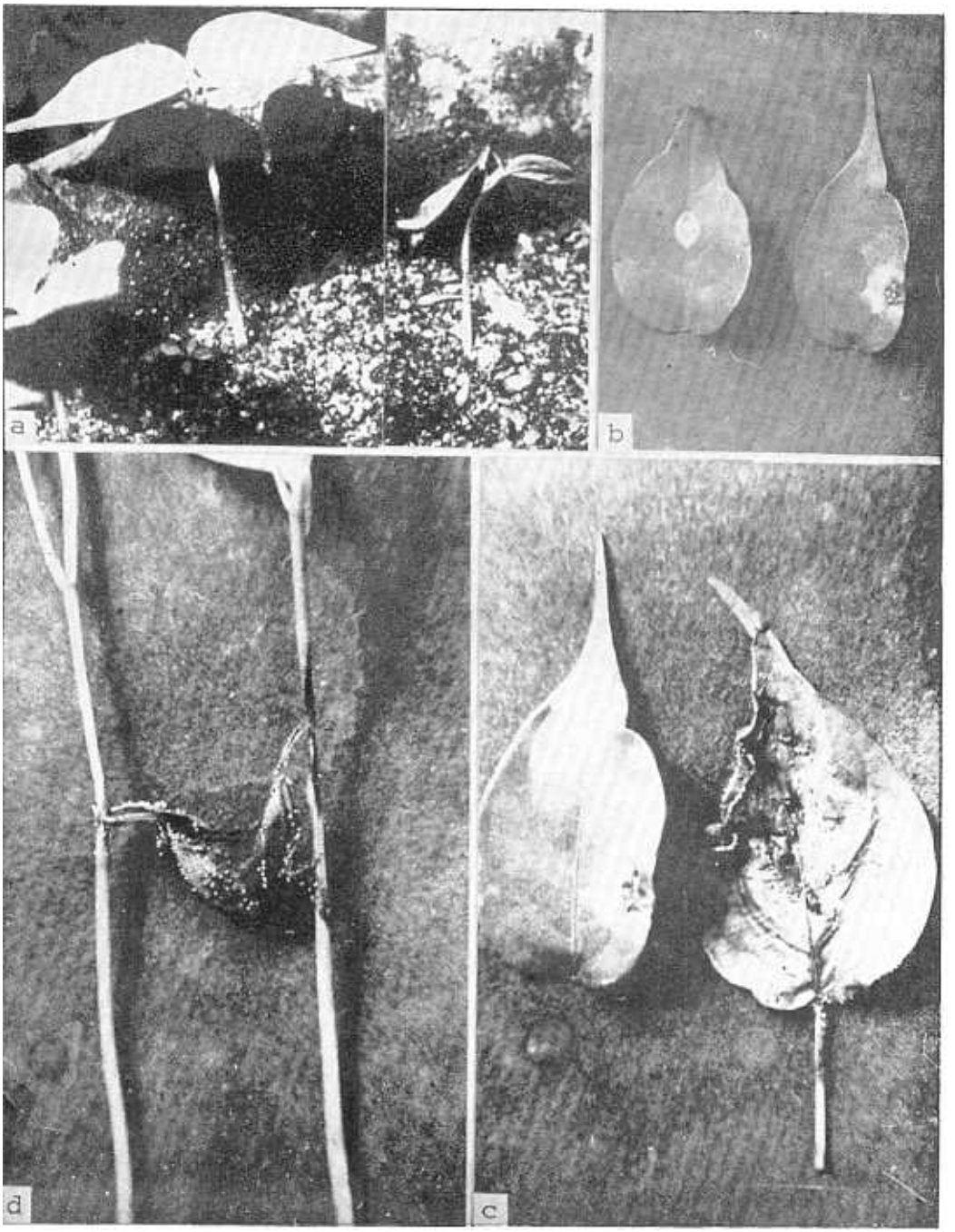


Fig. 13. Seedling diseases of *Bombax ceiba*. a, Collar rot caused by *Rhizoctonia solani* in 1-month-old seedlings; b, c and d, Leaf blight caused by *Corticium rolfsii*—b, c, gradual development of spots into blight; d, Spread of disease through contact.

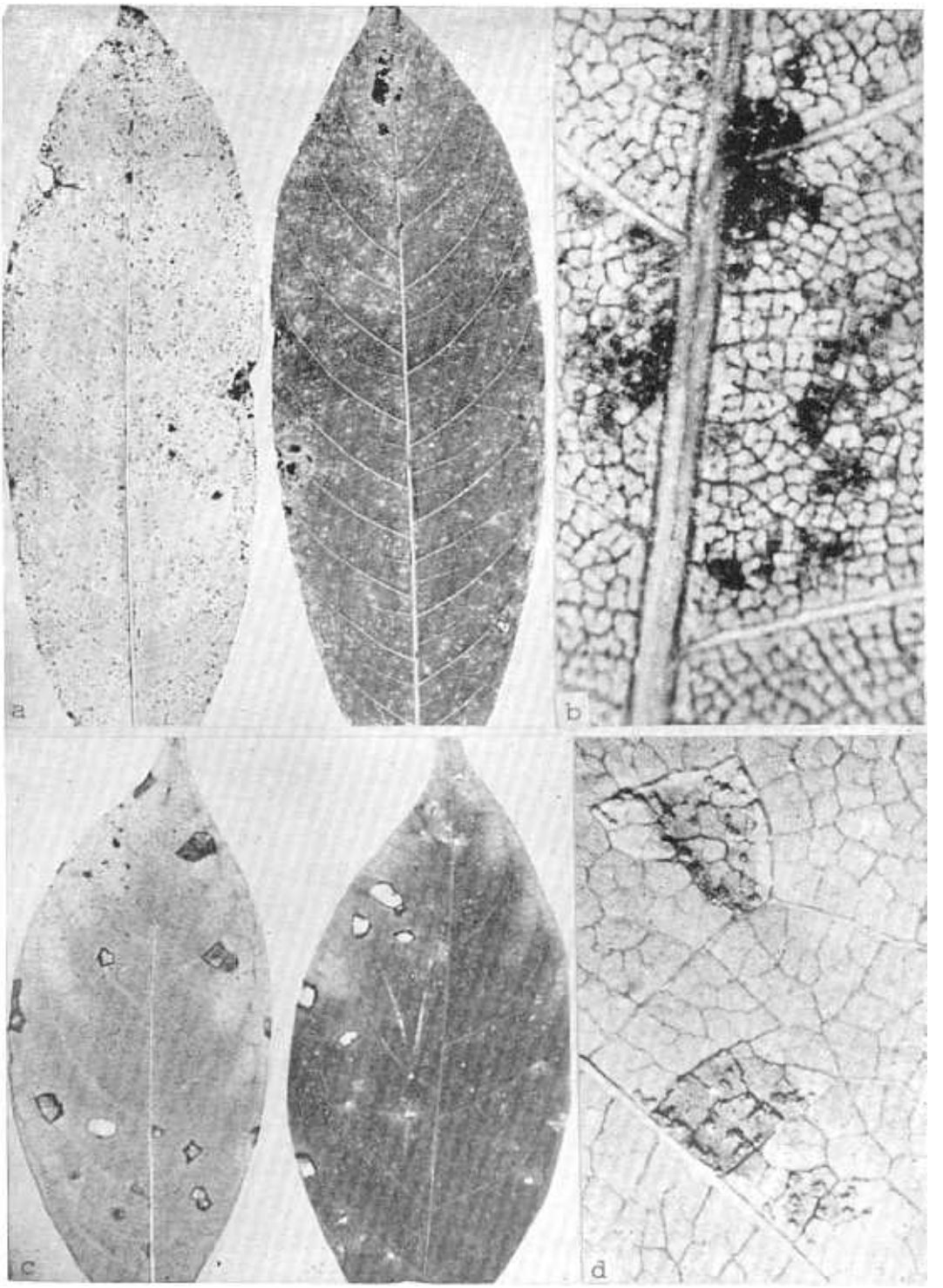


Fig. 14. Leaf rust of *Bombax ceiba* caused by *Uredo bombacis*. a, Symptoms of leaf rust on abaxial and adaxial surfaces; b, A magnified view of uredinia scattered on the abaxial surface; c and d, A different strain of *U. bombacis* localized in spots; d, A magnified view of grouped uredinia on the abaxial surface.

Control measures

Based on the studies conducted for the control of collar rot of ***Alianthus triphysa,*** two foliar sprays of Emisan-6 (0.005%a. i.) applied at a week's interval were found to be effective against leaf blight disease of ***B. ceiba.***

Discussion

Leaf blight of ***Bombax,*** recorded for the first time from India, appears to be a serious disease as it caused considerable mortality within a short period. Since high density of seedlings and high soil moisture facilitate manifestation and spread of the disease it is recommended to avoid these conditions in the nursery.

PLANTATION DISEASES

1. LEAF RUST

Occurrence and severity

Leaf rust of ***B. ceiba*** was observed throughout Kerala. The rust usually appeared towards the middle of South-West monsoon (July/August (Figs. 3a, b) and continued to attack healthy leaves till the time they were shed in January/February.

Severe infection was observed in young trees and a nursery at Peechi during 1982 and 1983. The incidence and severity of the disease varied depending upon locality and year of observation (Table 12). Generally the incidence of rust showed an increasing trend from 1980 to 1982. Only at Thundathil medium severity of rust was recorded during the three years of observation. In other plantations it varied from low to medium or vice–versa, except at Kannoth where it was absent.

Table 12. **Incidence and severity of *Uredo bombacis* in different plantations of *Bombax ceiba* surveyed during 1980 - 1982**

Sl. No.	Locality	1980			1981			1982		
		% incidence	DSI[a]	DSR[b]	% incidence	DSI	DSR	% incidence	DSI	DSR
1.	Kannoth	0		Nil			Nil	—	—	—
2.	Thundathil	38.19		M			M	75.11	1.26	M
3.	Kuttampuzha	77.27			78.52	0.92	L	83.25	1.15	M
4.	Mampazhathara	67.92	1.0	M	81.i3	1.79	M	39.62	0.83	L
6.	Choodal	31.40	0.97	L	68.96	1.36	M	70.25	1.20	M

aDSI, Disease Severity Index
bDSR, Disease Severity Rating
Observations not recorded

Symptoms

The rust infection first appeared only on leaflets of mature leaves. Later, as the top young leaves matured, the infection proceeded upwards and by December all the leaves got infected. Initially infection appeared as light greenish-yellow flecks on the upper surface during the rains. Uredinia developed in the form of light yellowish-orange blisters, 1-2 mm across, scattered on the lower surface (Figs. 14 a, b). Later, after the eruption, the pustules appeared bright-orange-brown in colour. Upper surface of leaf showed characteristic yellowish-orange flecking at the location of uredinia on the lower surface (Fig. 14 a). When the rust infection was severe (>25 uredinia cm-2) the whole leaflet turned yellow and defoliated prematurely.

During December/January, possibly a different strain of the rust was noticed causing angular necrotic spots (Fig. 14 c), dull whitish-grey to light brown in colour. Unlike the other strain in this case the uredinia were grouped (Fig. 14 d). The same leaf was found to be attacked by both types of rust strains.

Etiology

Uredo bombacis Petch (IMI 293349-50). Only the uredinial stage was observed on ***B. ceiba*** (Figs. 15 a-c).

Discussion

Though ***U. bombacis*** has been recorded on ***B. ceiba*** from Balehonnur, Karnataka (Bakshi, 1975) and Sri Lanka (Spaulding, 1961) not much details are available about its host-parasite relationships. This is the first report of *U. bombacis* from Kerala.

As the severe rust infection resulting in extensive premature defoliation has usually been observed in nurseries and young plantations of ***B. ceiba,*** especially in the latter case when grown with teak, it may affect the growth of trees considerably. By opening the canopy and removing the weed growth, which will help in reducing high humidity in young plantations, the rust incidence may be brought down considerably.

2. MYROTHECIUM LEAF SPOT

Occurrence and severity

Myrothecium leaf spot, a serious widespread disease of ***B. ceiba*** was observed at Choodal, Mampazhathara, Thundathil, Kuttampuzha, Mullaringad (Kothamangalam Div.) and Kannoth. The disease usually appeared towards the end of the monsoon during August/September (Figs. 3a, b) and continued to affect fresh foliage till November/December.

The incidence of the disease was quite high in all the plantations but the severity varied greately ffom locality to locality and year to year (Table 13) and it showed a decreasing trend as plants grew older. Severe infection resulting in premature defoliation was recorded at Choodal and Mampazhathara during 1980. Later, the severity in these plantations came down to medium and low during 1982, respectively. At Kuttampuzha, however, the disease severity was medium throughaut and at Thundathil it was medium in 1980 and 1981 and low during 1982.

Table 13. **Incidence and severity of Myrothecium leaf spot in different plantations of *Bombax ceiba* in Kerala surveyed during 1980- 1982**

Sl. No.	Locality	1980		1981				1982		
		% incidence	DSI[a]	DSR[b]	% incidence	DSI	DSR	% incidence	DSI	DSR
1.	Kannoth	95.80	1.03	M	83.52	0.85	L	—	—	—
2.	Thundathil	98.61	1.41	M	98.56	1.35	M	82.58	0.90	L
3.	Kuttampuzha	77.27	1.45	M	68.52	1:55	M	88.06	1.26	M
4.	Mampazhathara	100.00	2.94	S	98.11	1.61	M	70.88	0.98	L
5.	Choodal	95.04	2.18	S	98.85	1.47	M	'99.'25	1.60	M

[a]DSI, Disease Severity Index
[b]DSlt, Disease Severity Rating
—, Observations not recorded

Symptoms

Initially, amphigenous, minute, circular light brown spots . with dark brown margin developed on leaflets of any leaf irrespective of maturity but more on older ones. Sporodochia developed on both the surfaces, usually more on the lower surface, at the periphery of spots. The sporodochia produced abundant olive-green conidial mass which later turned dark in colour. The fungus invaded the adjoining green tissues and formed large necrotic brown areas (Fig. 16 a). As the spots enlarged, more concentric rings of sporodochia were added (Fig. 16b). The sporodochia of the inner rings became more or less inactive producing fewer conidia as compared to those situated in the outer ones. Thus, each leaflet developed one to four large spots, circular ,to oval with uniform to irregular dark brown margin, 15-25 mm in diam, which eventually coalesced to give rise to large necrotic area. The severely affected leaflets dried up and defoliated prematurely.

Etiology

Myrothecium roridum Tode ex Fr. (IMI 246401).

Pathogenicity

Pathogenicity of the isolate was confirmed on detached leaflets of ***B. ceibu,*** floated on **5** ppm of benzimidazole solution in large petri dishes. A conidial suspension, containing 25-35 conidia per drop of Pasteur pipette, was prepared from a I0-day-old culture of ***M. roridum*** in sterile distilled water. Five drops of this suspension were placed each on the adaxial and abaxial surfaces of leaflets, separately. Appropriate controls were also maintained. Observations were recorded daily for the appearance of symptoms.

Characteristic necrotic spots of the disease developed on the third day of inoculation on the lower surface and fifth day on the upper surface. Within two days numerous sporodochia appeared, which produced abundant conidia. M. ***roridum*** was re-isolated in pure culture from the necrotic spots.

Discussion

M. roridum was considered earlier to be a saprophyte. Taubenhaus (1935) and Preston (1936) first showed that ***M. roridum*** was parasitic on ***Antirrhinum*** and ***Viola.*** Later Fergus (1957) concluded from his studies that M. ***roridum*** is a widespread facultative parasite capable of invading plants that have been either wounded or exposed to certain environmental conditions such as high humidity. Severe infection in ***Bombax*** plantations situated in humid areas of the State confirms the latter observation. The disease is of serious consequences in young plantations (< 5-year-old), especially those in humid tracts with thick weed growth. As severe infection results in large-scale premature defoliation, possibly the growth of trees will be affected.

This is the first record of the disease from Kerala. Earlier it has been reported only from Jaldapara Sanctuary, West Bengal (Bakshi et al., 1972) and Maharashtra (Pawar and Thirumulachar, 1970).

3. COLLETOTRICHUM LEAF SPOT

Occurrence

This disease was observed in 18-month-old saplings in a nursery at Peechi (Kerala Forest Research Institute Campus) and in a young plantation at Choodal. Infection was severe at Peechi while at Choodal the disease appeared to be unimportant. The disease was recorded during September-November.

Symptoms

The disease was characterized by circular, 1-2 mm diam., dark black leaf spots with a narrow yellowish green border on the upper surface of leaflets (Fig. 16 c). On

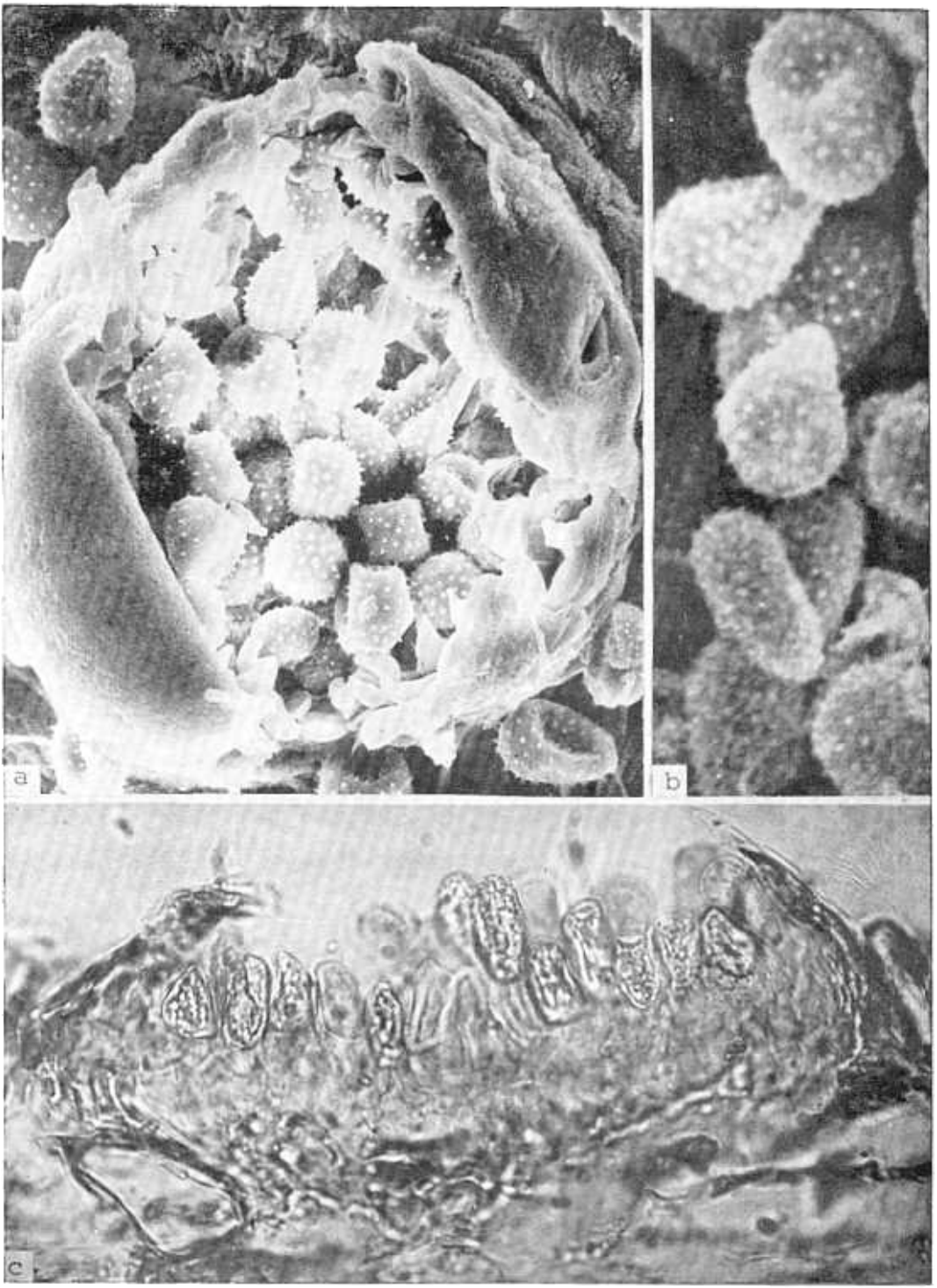


Fig. 15. Leaf rust of *Bombax ceiba* caused by *Uredo bombacis*. a, SEM of a uredinium (670 X). Note the ruptured peridium; b, SEM of urediniospores (1000 X); c, A vertical section through uredinium. Note the presence of a few paraphyses at the periphery.

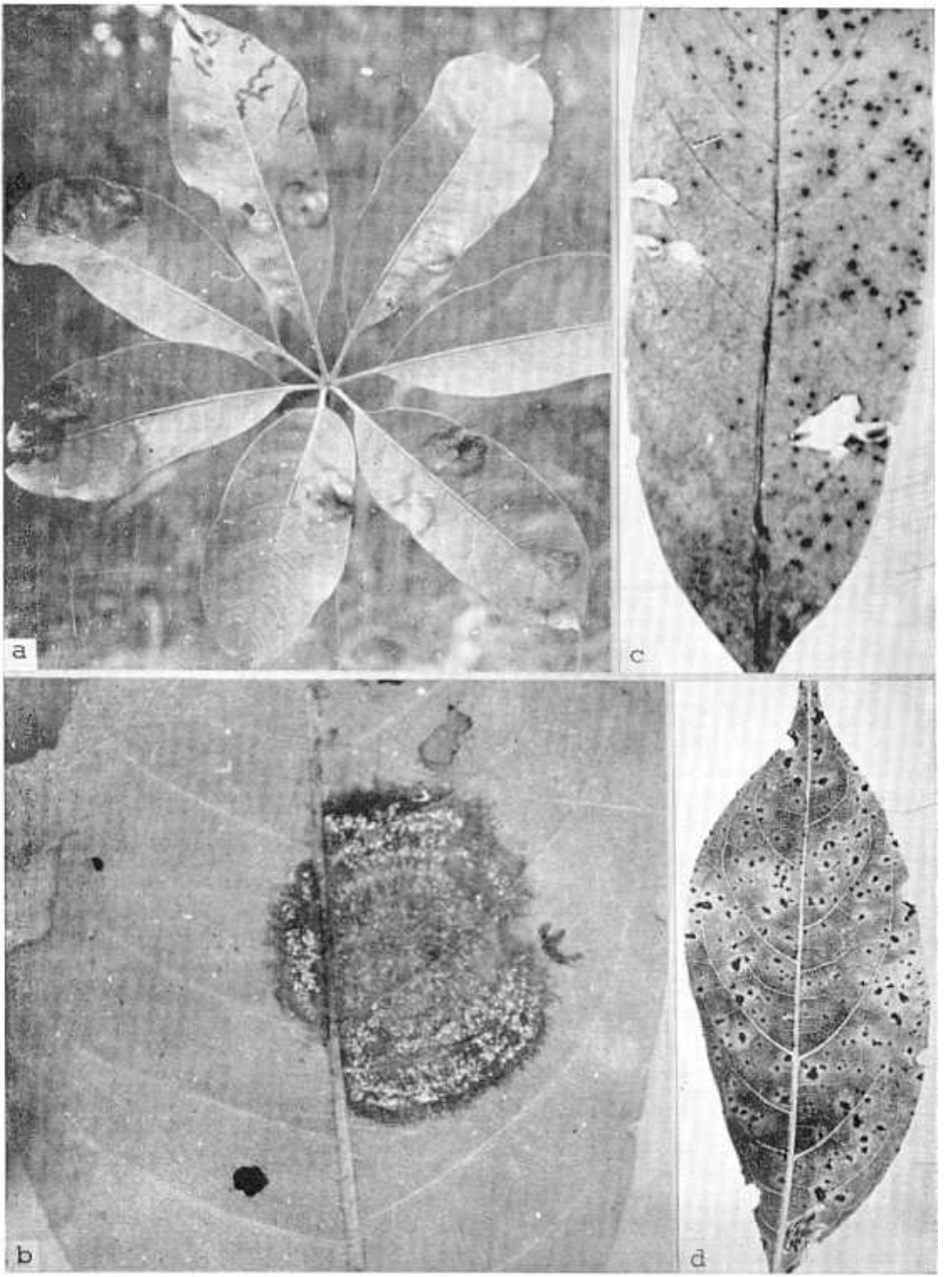


Fig. 16. Leaf spot diseases of *Bombax ceiba*. a, A leaf severely infected with *Myrothecium roridum*; b, A magnified view of a Myrothecium leaf spot. Note the concentric rings of sporodochia; c, Leaf spots caused by *Cercospora bombacina*; d, Leaf spots caused by *Colletotrichum gloeosporioides*.

the lower surface the spots "appeared dull grey in colour. Under high humid conditions, occasionally conidia were produced on the under surface of the spots. The necrotic area of the old spots got detached and formed shot-holes.

Etiology

Colletotrichum glbeosporioides (Penz.) Sacc.

Pathogenicity test

The pathogenicity of the isolate was confirmed on detached leaves obtained from 6-month-old seedlings. Conidial suspension, containing about ten conidia per drop of Pasteur pipette, was prepared by flooding 7-day-old culture of ***C. gloeosporioides*** with sterile distilled water. Small droplets of this suspension were placed at five marked areas on the adaxial and abaxial surfaces of ten leaflets, separately. The inoculated leaves were transferred to a humidity chamber maintained at >95% r.h. During the incubation period the temperature varied between 23 to 27°C. The inoculated leaves were observed daily for the development of symptoms. Approprite controls were also maintained.

Characteristic symptoms of the disease appeared after four to five days of incubation. The infected leaves produced shot-holes after five to six days, The pathogen was reisolated from the necrotic spots.

Discussion

C. gloeosporioides is worldwide in distribution affecting a large number of cultivated as well as wild plants, especially in warm-humid tropical climate. It is known to cause die-back, leaf spots, seedling blight, leaf blight, etc. of several hosts (von Arx, 1957;.Mordue, 1971). Colletotrichum leaf spot of ***B. ceiba*** is not widespread and appears to be unimportant., This is the first report of ***C. gloeosporioides*** causing leaf spot on ***B. ceiba.***

4. CERCOSPORA LEAF SPOT

Occurrence and severity

The disease, observed during October/November, was recorded only from two plantations at Choodal and Kannoth: The severity of the leaf spot was low at Kannoth while it was severe at Choodal.

Symptoms

The infection occurred on young as well as mature leaves as both were found to be equally susceptible to disease. Initially small pale brown spots appeared on the leaflets which turned into cream coloured necrotic areas with dark brown margins and

light pale halo around (Fig. 16 d); severe foliage infection in a nursery at Peechi resulted in early defoliation.

Etiology

From the naturally produced asexual fructifications (conidia) on the leaf spots the pathogen was identified as ***Cercospora bombacina*** T. S. & K. Ramakrishnan (IMI 290732).

Discussion

Though Cercospora leaf spot disease does not appear to be of common occurrence, severe infection can cause premature defoliation as observed in an isolated tree at Choodal. The disease has been earlier reported from Kerala (Butler and Bisby, 1960: Vasudeva, 1963).

5. PINK DISEASE

Occurrence

Pink disease of ***B. ceiba*** was found affecting young trees (4-year-old) in one plantation at Mampazhathara. The disease was recorded during September at the end of South-West monsoon (Figs. 3a, b).

Symptoms

The infection, characterised by pink encrustation over a canker, occurred somewhere on the upper half part of the stem. The stem above the canker rarely died due to incomplete girdling of phloem.

Etiology

Corticium salmonicolor Berk. &Br.

Only cobweb, pustule and pink stages were observed on the affected stem.

Discussion

C. salmonicolor, a pathogen of common occurrence in warm-humid tropical climate has a wide host range (Browne, 1965). Though during the survey it was recorded only at Mampazhathara it shows the susceptible nature of ***Bombax*** to pink disease. However, killing of the infected stem rarely possibly indicates some degree of resistance in ***B. ceiba*** to ***C. salmonicolor.*** ***B. ceiba*** is a new host record for ***C. salmonicolor*** from India.

6. PHANEROGAMIC PARASITE

Occurrence

Infestation of trees with a phanerogamic parasite was recorded at Choodal, Thundathil and Peechi (Kerala Forest Research Institute Campus). Except at Peechi,

where upto 3-5 clumps per tree were noticed, elsewhere there were only one to two clumps with <2 per cent incidence in the plantations.

Parasite

Dendrophthoe falcuta (L. f.) Ettingsh.

Discussion

D. falcata has been reported on ***B. ceiba*** from many countries (Browne, 1968). Observations indicate that though the incidence of mistletoe infestation was high on teak, it was quite low on ***B. ceiba*** trees in the same plantation. This possibly indicates unsuitability of ***Bombax*** for mistletoe infestation.

GENERAL DISCUSSION

During the course of the survey a total of 8 parasitic diseases including one phanerogamic parasite were recorded on ***B. ceiba*** (Table 14); So far there are no records of any nursery disease of ***B. ceiba.*** Leaf blight and collar rot caused high mortality affecting the stocking considerably. These diseases occurred exclusively in nurseries whereas the leaf rust and Colletotrichum leaf spot were prevalent in nurseries as well as plantations.

Table 14. **Check list of diseases of *Bombax ceiba* recorded in Kerala**

Sl. No.	Disease	Pathogen/Parasite	New Pathogen record *Bombax ceiba*	First record of pathogen from India
	A. NURSERY			
1.	**Collar rot**	***Rhizoctonia solani* Kuhn state of *Thanatephorus cucumeris* (Frank.) Donk**	+	–
2.	**Leaf blight**	***Corticium rolfsii* Curzi**	+	–
3.	**Leaf rust**	***Uredo bombacis* Petch**	–	–
4.	**Colletotrichum leaf spot**	***Colletotrichum gloeosporioides* (Penz.) Sacc.**	+	–
	B. PLANTATION			
1.	**Leaf rust**	***Uredo bombacis* Petch**	–	–
2.	**Myrothecium leaf spot**	***Myrothecium roridum* Tode ex Fr.**	–	–
3.	**Colletotrichum leaf spot**	***Colletotrichum gloeosporioides* (Penz.) Sacc.**	+	–
4.	**Cercospora leaf spot**	***Cercospora bombacina* T. S. & K. Ramakrishnan**	–	–
5.	**Pink disease**	***Corticium salmonicolor* Berk. & Br.**	+	–
6.	**Phanerogamic parasite**	***Dendrophthoe falceta* (L. f.) Ettingsh.**	–	–

In plantations six diseases were recorded. Only the leaf rust and Myrothecium leaf spot were of widespread occurrehce, the latter being a serious foliar disease causing premature defoliation. The other three diseases, Colletotrichum leaf spot, Cercospora leaf spot, and pink disease recorded for the first time on ***B. ceiba,*** were not common and occurred in low incidence.

AILANTHUS TRIPHYSA

Ailanthus triphysa (Dennst.) Alston occurs naturally in South East Asia. It was introduced as a plantation species in Kerala during early 1960s for its soft wood used in matchwood industry. ***A. triphysa*** is raised either in pure plantations or mixed with ***Bombax*** or teak under the category of 'soft wood plantations'. Occasionally, in some teak plantations the casualties are replaced with ***Ailanthus.*** Due to increasing demand of ***Ailanthus*** it is commonly grown in home-steads, farm yards, etc. Now it is also being popularised under social forestry programme in the State.

A list of plantations of ***A. triphysa*** surveyed during the course of this study is provided in Table 15.

Table 15. List of representative plantations of *Ailanthus triphysa* surveyed for incidence and severity of diseases during 1980-1982.

Sl. No.	Locality	Forest Divn.	Altitude (m above msl)	Area of plantation (ha)	No. of observation plots surveyed	*Year* of planting	Age of treesat survey in 1980 (years)
1.	Kannoth[a]	Wynad	200	5.0	3	1974	6
2.	Kuttampuzha[a]	Malayattoor	80	10.5	5	1976	4

[a] **Mixed 'soft wood' plantations of *A. triphysa* and *B. ceiba***

NURSERY DISEASES

Shot-hole disease caused by ***Colletotrichum*** state of ***Glornerella cingulata*** and sooty mould by ***Meliola ailanthii,*** which were recorded both in nurseries and plantations are described under plantation diseases.

1. DAMPING-OFF

Occnrrence

Damping-off of A. ***triphysa*** seedlings was observed at Peechi (Trichur Div.) and Thirunelli (Wynad Div.) At both places the disease occurred within two weeks of germination of seeds, when the first pair of leaves was just emerging, and caused upto 50-60 per cent mortality of seedlings. Dark shade over the seedbeds and excess watering favoured the disease development.

The disease appeared in the form of irregular patches in seedbeds. These patches enlarged rapidly from the periphery affecting the neighbouring, healthy seedlings under high soil moisture regimes.

Symptoms

Initially water-soaked lesions, measuring 5- 10 mm across, appeared on the hypocotyl near the ground level. The lesions turned brown in colour and the affected area got shrunken due to rapid collapse of cells, resulting into a prominent constriction (Fig. 17a). At this stage the seedlings fell over the ground and eventually died.

Etiology

Pytkium sp.

Control measures

As the disease occurs during warm weather under high soil moisture and dark shade, it is advisable to minimize watering frequency and quantity and open up the shade panda1 to get dispersed light as soon as the damping-off is noticed. This will facilitate in checking the rapid spread of the disease.

At Peechi the damping-off of ***A. triphysa*** was effectively controlled by two soil drenches of Dithane M-45 (0.05% and 0.02% a. i.) applied at a week's interval.

Discussion

Pythium is a common damping-off pathogen capable of causing large-scale mortality of young seedlings under favourable conditions. Damping-off of *A. triphysa* is a new disease record.

2. COLLARROT

Occurrence

This is the most widespread and serious disease among all the nursery diseases of *A. triphysa* recorded in Kerala. Severe infection was observed at Pattikad, Erumapetti (Trichur Div.), Periya, Kannoth (Wynad Div.), Kalladikode, Chandranagar, Erumayoor (Palghat Social Forestry Div.), Neriamangalam (Idukki Social Forestry Div.), Adirapally, Kollathirumede (Vazhachal Div.), Chinnar (Munnar Div.) and Koovappadv and Kodanad (Malayattoor Div.).

The disease was first noticed affecting one-month-old seedlings in small irregular patches which spread fast from the periphery damaging large areas of the seedbeds within a few days (Fig. 17b). The disease often continued to affect even 3- to 4-month-old seedlings causing 30-60% mortality during April/May, if seedlings had not been pricked out into containers or proper control measures were not adopted earlier.

Symptoms

First symptom of the disease was the appearance of water-soaked lesions at the collar region. These lesions developed into light brown necrotic area and the tissue got decayed, which resulted in a constriction at the collar region and consequently death of seedlings (Fig. 17c). Young seedlings (I-month-old) usually collapsed from the decayed region and fell over the ground; old seedlings (>2-month-old) showed only wilting.

Occasionally infection of hypocotyl region in emerging seedlings was also observed. Affected seedlings either failed to emerge or emerged only partially, which died later.

Etiology

Rhizoctonia solani Kuhn state of ***Thanatephorus cucumeris*** (Frank.) Donk (IMI 267022).

Pathogenicity

For testing pathogenicity of the isolate, 1-month-old inoculum of *R. solani*, raised on corn meal agar, was dried and powdered in a waring blender. One gram of this inoculum, containing abundant microsclerotia was mixed thoroughly with 2 Kg of sterile soil in a sterile aluminium tray (30 x 30 x 5 cm.) The infested soil was incubated for about a week. Healthy 1-month-old seedlings, grown in pasteurised soil, were transplanted in trays containing the infested soil. All trays were transferred to a humidity chamber where r. h. was >95 per cent and temperature ranged between 26-31°C. The seedlings were watered with measured quantity of water. Observations on the development of symptoms were recorded every day.

On the second day, the mycelium was seen growing on the root collar from the soil, which caused elongated water .soaked lesions. Subsequently typical symptoms of collar rot were observed on all seedlings transplanted in the infested soil. Death of seedlings occurred from fourth day onwards.

Control measures

As the disease caused high mortality of seedlings in various nurseries, studies were taken up on chemical control measures for ensuring healthy stock. Some of the commonly available fungicides, such as Captan, Vitavax, Bavistin and Emisan-6 were evaluated at 0.025, 0.05, 0.1 and 0.2% a. i. for their efficacy against *R. solani* using poison food technique and soil method. In the case of Emisan-6 a concentration of 0.01% a. i. was also used. The concentration was considered effective when the growth of the test fungus was inhibited completely in culture. The results are summarised in Table 16.

Table 16. Efficacy of various fungicides in inhibiting growth of *R. solani.*

Fungicide	Concentration % (a.i.)	Diameter growth of colony (mm)[a]	
		Poison food technique	Soil method
Bavistin	0.025	0	63.0
	0.05	0	0
	0.1	0	0
	0.2	0	0
Vitavax	0.025	0	28.0
	0.05	0	0
	0.1	0	0
	0.2	0	0
Captan	0.025	26.1	67.0
	0.05	27.3	66.0
	0.1	19.1	60.8
	0.2	17.6,	60.0
Emisan-6	0.01	0	0
	0.025	0	0
	0.05	0	0
	0.1	0	0
	0.2	0	0
control	Nil	85.0	90.0

[a] Mean of three observations

Of the five fungicides tested Emisan-6 was the most effective in inhibiting the growth completely in both the techniques. Bavistin and Vitavax were also effective at all concentrations, except at 0.025, where some growth occurred. Captan was not effective at any of the concentrations used.

Due to economic reasons and availability, Emisan-6 was tested for its efficacy in controlling the collar rot disease in nursery trials. Since the effective concentration, 0.025% (a.i.) caused phytotoxicity in young seedlings, lower concentrations were evaluated. In trials the disease was effectively controlled by two applications of 0.005% (a.i.) of Emisan-6 given as soil drench at the rate of 30 litres of solution per standard bed at an interval of 10-15 days depending upon the age of seedlings.

Since the disease is manifested under high soil moisture it is recommended to avoid overwatering and high seedling density in seedbeds.

Discussion

Rhizoctonia solani is world wide in distribution and is known to be pathogenic to a large number of plants (Baker, 1970). This is the first report of *R. solani* causing cdlar rot in seedlings of *A. triphysa*. The fungicides used in this study have been reported earlier to control *R. solani* on different hosts (Borum and Sinclair, 1968; Kataria and Grover, 1978; Leach and Garber, 1970; Taneja and Grover, 1982). In the present study Emisan-6 (MEMC) was found to be highly effective in both *in vitro* and *in vivo* trials. On the contrary, earlier Kataria and Grover (1978) have reported MEMC as less efficient in controlling damping-off of mung bean caused by *R. solani*. This discrepancy in the effectiveness of MEMC on *R. solani* may be due to strain differences of the pathogen, besides host plant, season and location (Kataria and Gover, 1976).

As a prophylactic measure for controlling the collar rot disease it is recommended to drench the nursery beds with Emisan-6 (0.005% a. i.) or any other preparation of MEMC when the seedlings are l-week-old.

3. SEEDLING BLIGHT

Occurrence

The seedling blight disease, prevalent after the onset of South-West monsoon (Figs. 3a,b), was observed in 4-to 6-month-old seedlings. The survey of the affected nurseries revealed that the incidence of the disease was high where seedlings older than 4-month-old were pricked into containers, though in some nurseries the disease was also found in seedbeds. The incidence of the disease was negligible or absent where 1-to 2-month-old seedlings were pricked. High mortality of seedlings was recorded at Peruvannamuzhi (Kozhikode Div.), Vaikkom (Ernakulam Social Forestry Div.) and Kalladikode (Palghat Div.).

Symptoms

Initially the infection appeared on the stem near the apex in the form of elongated brown spots, which soon coalesced to form large necrotic area, Thereafter, the shoots showed typical symptoms of seedling blight i. e., wilting and drying up of leaves followed by death of the terminal bud (Fig. 17 d). The necrosis extended downwards which eventually killed the seedling. During the monsoon, on the affected stem the pathogen produced numerous splash dispersed oonidia in acervuli, which helped in the spread of infection.

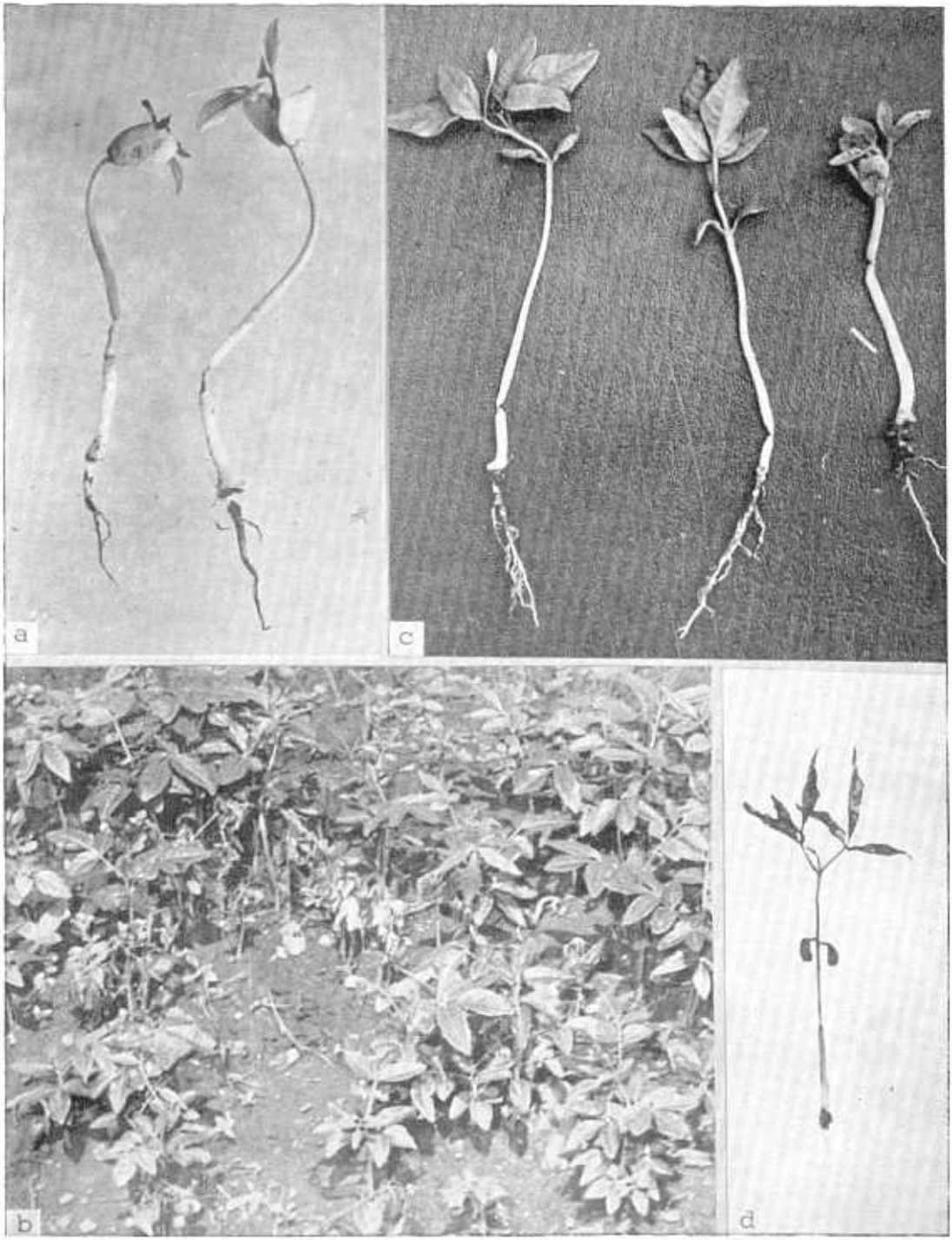


Fig. 17. Seedling diseases of *Ailanthus triphysa*. a, Damping-off of 1-week-old seedlings caused by *Pythium* sp.; b, Seedlings in a seedbed affected with collar rot caused by *Rhizoctonia solani*; c, Seedlings affected with collar rot; d, A seedling affected with blight caused by *Colletotrichum dematium*.

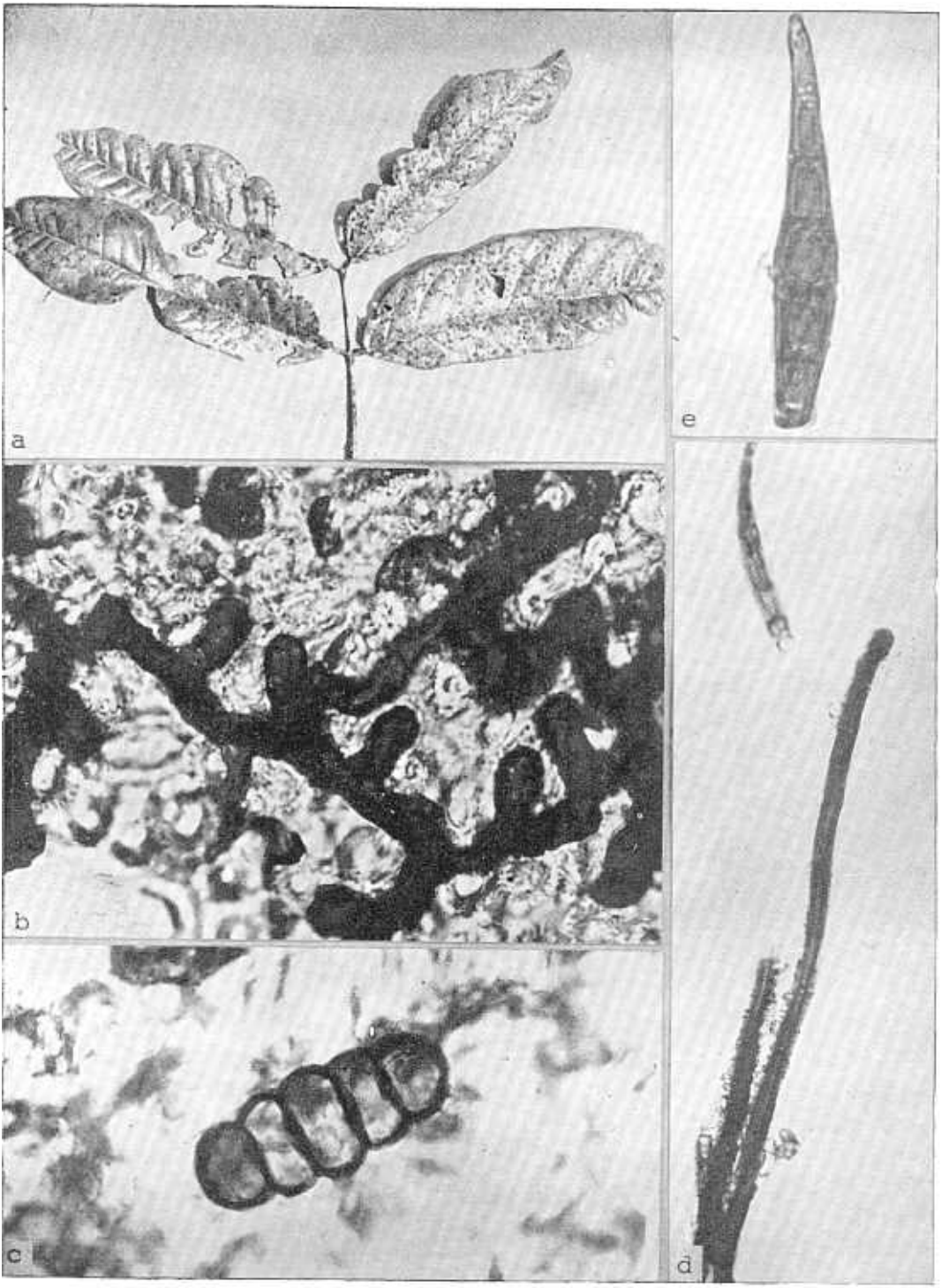


Fig. 18. Sooty mould of *Ailanthus triphysa*. a, Adaxial surface of leaflets severely infected with *Meliola ailanthii*; b, Hyphopodia on the mycelium; c, A conidium of *M. ailanthii* sp. nov. (760 X); d and e, Conidiophore (230 X) and conidia (760 X) of *Spiropes capensis*, a hyperparasite of *M. ailanthii*.

Etiology

Colletotrichum dematium (Pers. ex Fr.) Grove (IMI 260692).

Pathogenicity

Seedlings of *A. triphysa* were raised in the nursery at Peechi. Three-month-old seedlings were pricked out into polythene containers and kept in shade for establishment for about a week: Before inoculation the seedlings were washed thoroughly with sterile water to remove soil particles. Twenty container seedlings, ten each wounded on the stem with the help of a sterile needlk and unwounded, were transferred to a humidity chamber (>95% r. h)and sprayed with sterile distilled water. The following day seedlings were inoculated with a conidial suspension (20-25 conidia per drop of the Pasteur pipette) using a fine atomizer. The suspension was prepared from a 10-day-old culture of ***C. dematium.*** Appropriate controls (wounded and unwounded) were also maintained.

The wounded seedlings developed dark lesions on the stem, three to four days after inoculation. Soon these lesions coalesced and caused decay extending upto the apex. Within 8-10 days all the seedlings showed symptoms of seedling blight. In the unwounded set blight developed only in three seedlings after 15-17 days of incubation.

Control measures

Foliar spray of Fytolan or Thiride (0.02% a.i.) applied twice at a week's interval was found to be effective in controlling t4e disease. During the rainy season it is recommended to use systemic fungicide, Bavistin (0.01% a.i.) instead of Fytolan or Thiride.

Development of the disease can be avoided to a considerable extent by means of proper nursery practices, such as raising the nursery at proper time, avoiding crowding of seedlings and pricking out 45-to 60-day-old seedlings into containers.

Discussion

Colletotrichum dematium recorded on 218 hosts from 37 countries, is considered to be usually saprophytic. It is commonly found in temperate climate and less so in the tropics (Sutton, 1980). The pathogenicity tests on *A. triphysa* confirm the pathogenic .nature of ***C. dematium,*** which is a new pathogen record for the host.

4. BACTERIAL LEAF SPOT

Occurrence

Though the bacterial leaf spot disease was found in almost all the nurseries surveyed, it did not appear to cause any concern as the incidence and severity were

low. However, at Peruvannamuzhi (Kozhikode Div.) the disease caused severe foliage infection in 5-month-old container seedlings resulting in defoliation during the South-West monsoon. Almost 75 per cent of the container plants were found to be affected with bacterial leaf spot.

Symptoms

The first symptom of the disease was the appearance of water-soaked translucent amphigenous round to irregular lesions, which soon turned light brown. The lesions enlarged and coalesced to give rise larger irregular spots, which occasionally covered a considerable part of the lamina. The spots were shiny and sticky to touch and the necrotic tissue of the spots became thin and somewhat elastic. Severely infected leaflets were defoliated prematurely and seen sticking on other healthy leaves or stem.

Etiology

Pseudomonas sp. (possibly ***P. solanacearum*** (E. F. Smith) E. F. Smith)

The colony on nutrient agar off-white, slow growing, producing diffusible pigment; gram negative, rod-shaped, non-fluorescent.

Pathogenicity

The pathogenic nature of the bacterial isolate was confirmed on 2- and 5-month-old container seedlings of A. triphysa. Bacterial suspension with a concentration of ca. 10^{10} bacteria ml^{-1} was prepared by flooding a 4-day-old culture with sterile distilled water. The foliage of the seedlings was washed twice thoroughly with sterile distilled water. They were transferred to a humidity chamber (> 95% r. h.) and sprayed with sterile distilled water using a fine atomizer. Five seedlings were inoculated with the bacterial suspension by spraying it on upper and lower surfaces of the foliage. In another set of five seedlings, the lower surface of young and mature leaves were injected with the bacterial suspension using a fine micro-hypodermic syringe. All seedlings were incubated in the humidity chamber and observations recorded daily on the appearance of the symptoms.

Typical wafer-soaked bacterial leaf lesions developed on the third day in the injected seedlings and after 6-7 days of incubation in sprayed ones. Young leaves and young seedlings were found to be more susceptible than the old ones as the size of the spots was much larger in the former than in latter; also, old seedlings took longer time to develop the spots as compared to young ones.

Control measures

The bacterial leaf spot disease was controlled by a foliar application of Plantamycin (0.01% a. i.).

PLANTATION DISEASES

1. SOOTYMOULD

Occurrence and severity

The sooty mould was quite common in nurseries and plantations during hot and warm period following North-East monsoon (Figs. 3a, b). Infection of medium severity was observed in plantations at Kuttampuzha (1980, 1982) and Kannoth (1982) (Table 17) and in nurseries at Periya (Wynad Div.) and Kollathirumede (Vazhachal Div.). The incidence of the disease was generally high even when the severity was low, except at Kuttampuzha during 1981. Though the disease is of minor importance, it may become of serious concern in nurseries where it can cause defoliation due to complete coverage of leaf lamina with the black mouldy growth,

Table 17. **Incidence and severity of sooty mould caused by *Meliola ailanthii* in two plantations of *Ailanthus triphysa* surveyed during 1980-1982**

Sl. No.	Locality	1980			1981			1982		
		% incidence	DSI[a]	DSR[b]	% incidence	DSI	DSR	incidence	DSI	DSR
1.	Kannoth	86.82	0.95	L	82.09	0 27	L	96.35	1.60	M
2.	Kuttampuzha	97.77	1.64	M	4.76	0.05	L	98.15	1.29	M

[a] **DSI, Disease Severity Index**

[b] **DSR, Disease Severity Rating**

—, Observations not recorded

Symptoms

The disease appeared in the form of superficial black irregular to round patches initially on the upper surface (Fig. 18a). Under warm and humid conditions these patches enlarged and coalesced to give rise to large patches. In severe cases the the infection also extended to the lower surface. Severely infected leaves turned yellow and defoliated prematurely.

Etiology

Meliola sp. (IMI 270190a). As it differs from other known species of ***Meliola*** it is described as a new species, ***Meliola ailanthii*** sp. nov. (Figs. 18b, c).

Hyperparasite

Spiropes capensis (Thum.) M.B.Ellis (IMI 270190 b), a hyperparasite of *M. ailanthii,* was frequently observed on most of the affected leaves (Figs, 18d, e).

Discussion

Sooty mould is a tropical fungus occurring on a wide variety of plants as a superficial net of dark mycelium. Usually it does not cause any harm, but when the entire leaf surface is covered some yellowing may be observed. In severe cases premature defoliation is caused, possibly because of inhibited photosynthetic activity.

2. SHOT-HOLE LEAF DISEASE

Occurrence

The disease, recorded throughout Kerala in nurseries as well as in plantations, was prevalent during June to November when high humidity (85-95 per cent r h.) and moderate temperature (22-25°C) occur (Figs. 3 a,b). The incidence of the disease varied greatly from one location to another (Table 18). Infection of medium severity was observed in a 6-year-old plantation at Kuttampuzha during 1982 where ca. 95 per cent of the trees had serious foliar damage due to extensive shot-holes; at Kannoth it occurred only at low severity during 1980 and was absent during 1981 and 1982 (Table 18). The affected leaflets were shed prematurely leaving the bare rachii attached to the trees. Young and mature leaves were equally affected.

Table 18. Incidence and severity of shot-hole disease caused by ***Colletotrichum gloeosporioides*** in two Plantations of ***Ailanthus triphysa*** surveyed during **1980-1982**

Sl. No.	Locality	1980			1981			1982		
		% incidence	DSI[a]	DSR[b]	% incidence	DSI	DSR	% incidence	DSI	DSR
1.	Kannoth	**41.36**	**0.945**	**L**	0.0	0.0	Nil	0.0	***0.0***	Nil
2.	Kuttampuzha	59.80	**0.61**	**L**	**0.0**	**0.0**	**Nil**	**94.83**	**1.33**	**M**

[a]DSI; Disease Severity Index
[b]DSR, Disease Severity Rating
–, Observations not recorded

Symptoms

Initially symptoms developed on the leaflets as dark green areas lined with a yellowish green margin (Fig. 19a). The colour of these areas changed to light yellowish to reddish orange and finally light brown (Fig. 19b). The necrotic areas of the leaf became thin and got detached very easily either by wind or due to impact of rain drops, thus leaving a prominent hole in the lamina (Figs. 19c,d). A sevefely infected leaflet got completely deformed with irregular cuts due to separation of a large part of the lamina after the infection (Figs.19 e,f).

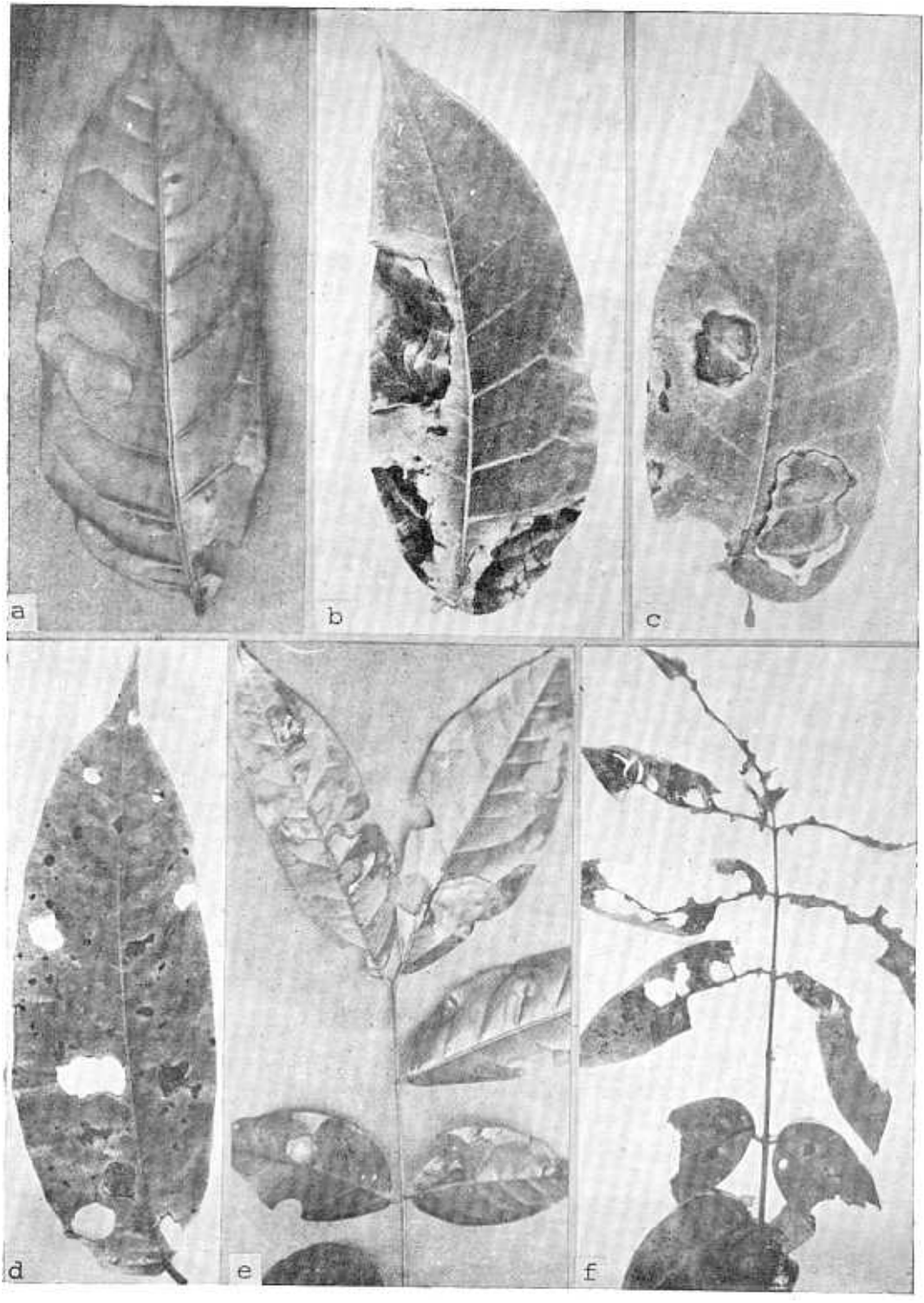


Fig. 19. Shot-hole disease of *Ailanthus triphysa* caused by *Colletotrichum* state of *Glomerella cingulata*. a, A leaflet showing initial infection in the form of a leaf spot with marked outline; b, c and d, Leaf spots turn necrotic and get detached; e, A leaf showing typical shot-holes; f, A severely infected leaf.

Fig. 20. Stem diseases of *Ailanthus triphysa*. a and b, Pink disease caused by *Corticium salmonicolor*. Note profuse growth of pink stage over the canker in b; c and d, Stem canker caused by *Botryodiplodia theobromae*; e, Initial symptom of stem canker; f, Oozing of dark gummy substance from the canker area.

Etiology

Colletotrichum state of ***Glomerella cingulata*** (Stonem.) Spauld. & Schrenk (IMI 278253).

Pathogenicity

Pathogenicity of the isolate was tested on detched leaves floated on 5 ppm solution of benzimidazole in sterile petri dishes, as well as on leaves of 4-month-old container plants. Ten leaflets each were inoculated on the adaxial and abaxial surfaces by placing small droplets of the conidial suspension (20-25 conidia per drop) prepared from a 7-day-old culture. The petri dishes were incubated at 25 + 2°C in 16 h light cycle. The inoculated plants were transferred to a humidity chamber maintained at > 95 per cent r. h.

Symptoms of the disease appeared after three to five days of incubation both on detached leaves and leaves of test plants. Characteristic spots leading to shot-hole developed after five days, from which the pathogen was reisolated.

Discussion

Colletotrichum spp. are world-wide in distribution, causing a variety of diseases on various cultivated and wild plants (von Arx, 1957). This is the first report of ***Colletotrichum*** state of ***Glomerella cingulata*** causing shot-hole in *A triphysa.*

3. PINK DISEASE

Occurrence

The pink disease of *A. triphysa* was recorded only at Kuttampuzha during 1980 and 1981 when the incidence was 25 and 97 per cent and the severity low and medium respectively (Table 19). In severe cases the top of the diseased tree died due to complete girdling of phloem.

Table *19.* **Incidence and severity of pink disease caused by *Corticium salmonicolor* in two plantations of *Ailanthus triphysa* surveyed during 1980-1982**

Sl. No.	Locality	1980			1981			1982		
		% incidence	DSI[a]	DSR[b]	% incidence	DSI	DSR	% incidence	DSI	DSR
1.	Kannoth	0.0	0.0	Nil	0.0	0.0	Nil	0.0	0.0	Nil
2.	Kuttampuzha	25.3	0.28	L	97.40	1.79	M	0.0	0.0	Nil

[a]DSI, Disease Severity Index

[b]DSR, Disease Severity Rating

–, Observations not recorded

Symptoms

The disease was characterised by a canker formed on the upper half of the stem, which resulted in the girdling of phloem thus killing the stem above the canker (Figs. 20 a, b). The stem got constricted at the canker region and bark showed longitudinal splitting.

Etiology

Corticium salmonicolor Berk. & Br. Only cobweb, pustule and perfect (pink) stages were observed. This is the first record of pink disease on *A. triphysa.*

4. STEM CANKER AND DIE-BACK

Occurrence

The disease was observed only in Trichur area in a plantation (4year-old, 40.98 ha) at Pothuchadi, Paravattani, Mala (Trichur Div.) and in a homestead at Mullakkara (Figs. 20 c, d). At Pothuchadi the disease was spreading in patches, killing the infected plants. However, at Mullakkara where the trees were 2-year-old, no mortality occurred. Infection was also recorded on the stem of young seedlings which were killed outright.

Symptoms

The symptoms of stem canker at Pothuchadi appeared to be significantly different from those at Paravattany. At Paravattany initially small longitudinal cracks appeared on the main stem (Fig. 20 e) from which occasionally a dark gummy substance oozed out (Fig. 20 f). These cracks developed into large cankers, measuring 30-60 cm in length. The tissue under the canker turned greyish. Due to the infection numerous epicormic shoots developed just below the canker. The epicormic shoots also got infected in due course due to spreading canker, on which numerous fructifications of the causal organism produced.

At Pothuchadi, the infection started near the ground at the root collar and rapidly spread upwards (Figs. 21c, d). Due to the infection, which was several millimeters deep, the, plants-showed decline symptoms with aweak crown having fewer and yellowed 1eaves. The weakened main shoot as well as the branches got infected and died slowly and gradually. As a result, numerous epicormic shoots were produced (Fig. 21 a). In the meantime the basal stem canker had also spread downwards decaying the root system. Complete girdling of the outer bark killed the trees outright (Fig. 21 b).

Etiology

Botryodiplodia theobromae Pat.

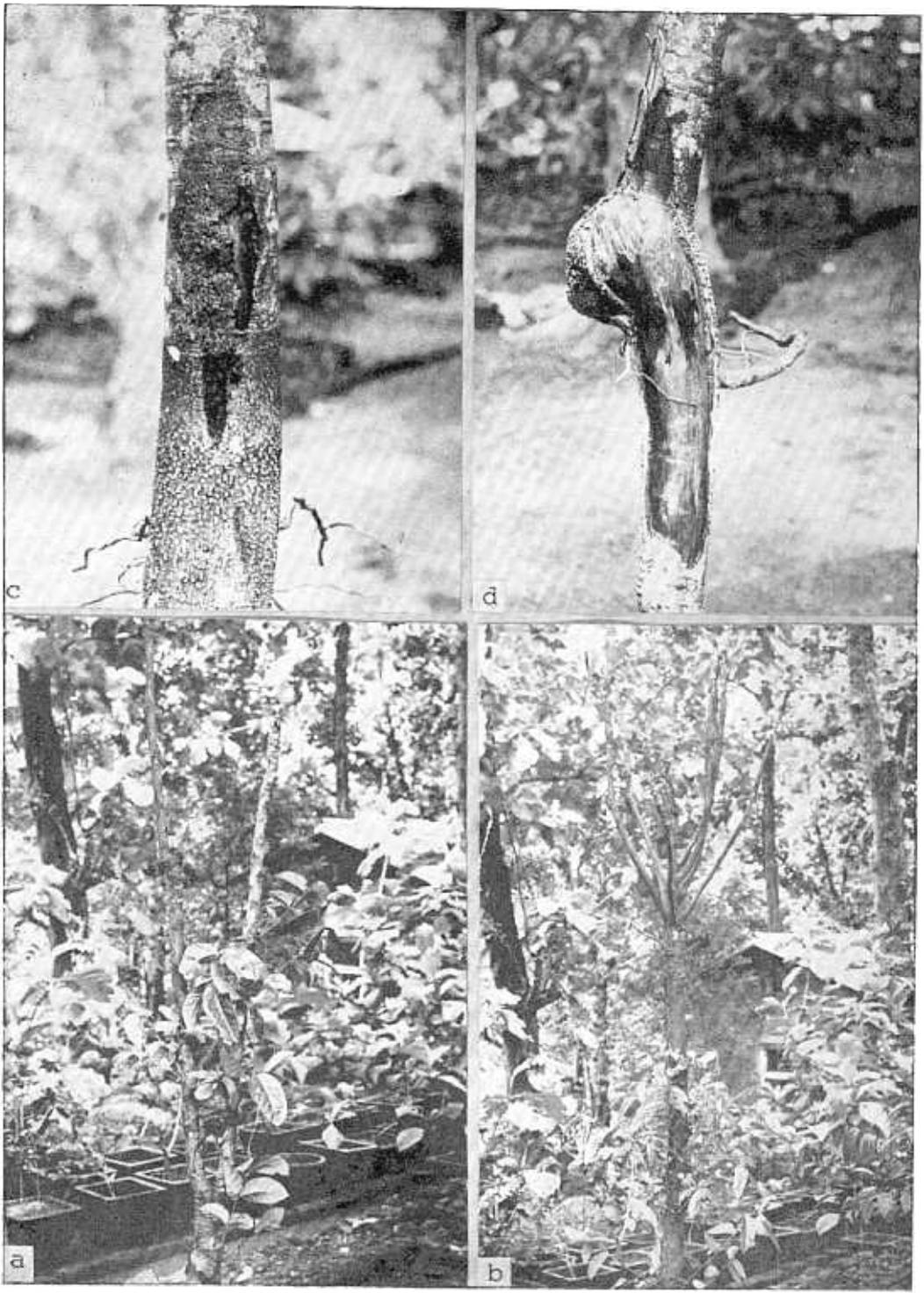


Fig. 21 Die-back of *Ailanthus triphysa* caused by *Botryodiplodia theobromae*. a and b, Die-back of terminal shoots and development of epicormic shoots; c, Initial canker at collar area near the ground; d, Canker extending to root causing decay of the latter.

Pathogenicity

The pathogenicity of the isolate was confirmed in wound inoculations on 2-year old ***A. triphysa*** plants.

Discussion

Stem canker and die-back caused by ***B. theobromae,*** a new disease of ***A. triphysa,*** appeared to be a serious disease as it killed the affected plants. Since ***B. theobromae*** is known to be a weak wound parasite, the possible way of manifestation of the disease and its role in killing the trees needs detailed investigation. The role of the leftover stem of tapioca, (grown for two years as a taungya), which forms a good substrate for the saprophytic growth of ***B. theobromue,*** also warrants further studies.

DISEASE OF UNKNOWN ETIOLOGY

1. MOSAIC

A mosaic disease, similar to mosaic virus disease, was noticed in nurseries. Incidence of the disease was very low and no stunting or abnormal growth of seedlings was observed. The affected seedlings gradually became weak and dried up.

Symptoms

The leaves of the affected seedlings had off-white to yellow, irregular patches. These chlorotic patches could even be found on the youngest leaves.

Discussion

Though the disease appeared to be caused by a virus, in the absence of transmission studies the etiology of the disease is uncertain

GENERAL DISCUSSION

No records are available on diseases of ***A. triphysu.*** During the course of the survey a total of nine diseases were recorded, eight in nursery and four in plantation, three (sooty mould, shot-hole disease and stem canker), being common to both (Table 20). Collar rot was the most prevalent and destructive disease in nurseries resulting in heavy mortality of seedlings. The results of the chemical control trials show that the disease can be controlled effectively by applying Emisan-6 once or twice depending upon the severity and age of seedlings. In nurseries no root disease was observed.

Among the plantation diseases, a foliar shot-hole disease and stem canker may pose serious problems in humid areas by causing extensive defoliation (Kuttampuzha) and mortality (Pothuchadi) respectively.

All the nine diseases described here are new records for ***A. triphysa.***

Table 20. Checklist of diseases of ***Ailanthus triphysa*** recorded in Kerala

Sl, No.	Disease	Pathogen(s)	New pathogen record for *A. triphysa*	First record of pathogen from India
	A. *NURSERY*			
1.	Damping-off	***Pythium*** sp.	+	—
2.	Collar rot	***Rhizoctonia solani*** Kuhn state of ***Thanatephorus cucumeris*** (Frank.) Donk	+	—
3.	Seedling blight	***Colletotrichum dematium*** (Pers. ex Fr. Grove	+	—
4.	Seedling stem infection	***Botryodiplodia theobromae*** Pat.	+	—
5.	Bacterial leaf spot	***Pseudomonas*** sp. (possibly ***P. solanacearum)***	+	—
6.	Shot-hole	***Colletotrichum*** state of ***Glomerella cingulata*** (Stonem.) Spauld. & Shrenk	+	—
7.	Sooty mould	***Meliola ailanthii*** sp. nov.	+	+
8.	Mosaic	Unknown etiology	−	—
	B. *PLANTATION*			
1.	Sooty mould	***Meliola ailanthii*** sp. nov. and its hyperparasite, ***Spiropes capensis*** (Thum.) M. B. Ellis	+	+
2.	.Shot-hole	***Colletotrichum*** state of ***Glomerella cingulata***	+	—
3.	Pink disease	***Corticium salmonicolor*** Berk. & Br.	+	—
4.	Stem canker and die-back	***Botryodiplodia theobromae***	+	—

GMELINA ARBOREA

Gmelina arborea L., a large to medium sized tree found in deciduous and moist-deciduous forests, is a hardwood native to India, Burma, Sri Lanka and other parts of South East Asia. During early 1970 s it was introduced in plantation forestry in Kerala for its valuable timber. So far ca. 600 ha have been raised under G. ***arborea*** in the State, most of the plantations being in Central and Southern Forest Circles.

A list of plantations surveyed is provided in Table 21.

Table 21. **List of representative plantations of *Gmelina arboreo* surveyed for incidence and severity of diseases during 1980-1982.**

Sl. No.	Locality	Altitude (m above msl)	Forest Divn.	Area of plantation (ha)	No. of obervation plots surveyed	Year of planting	Age of trees at survey in 1980 (years)
1.	Kottappara	50	Malayattoor	15.0	3	1977	3
2.	Onthupacha	150	Punalur	17.0	3	1977	3

NURSERY DISEASES

Colletotrichum leaf spot disease, observed both in nursery and plantation, is dealt with under plantation diseases.

1. STEM INFECTION

Occurrence

The disease, observed in 3-to-4-month-old seedlings in nurseries a Arippa (Kerala Forest Development Corp., Trivandrum) and Adhirapally (Vazhachal Div.), caused 10 to 30 per cent mortality, highest being at Arippa. The disease occurred under warm and humid conditions (April/May), especially when the seedlings were crowded and it continued to affect till the onset of monsoon (June/July) (Figs. 3a, b).

Symptoms

Initially light brown flecks appeared on lower part of the stem, which soon changed to dark brown discolouration (Fig. 22a) often extending upto the entire length of seedling. At this stage the seedling showed wilting with flaccid and droopy

leaves. The affected seedlings failed to survive. Numerous pycnidia developed on the dead stem, which produced spore mass in creamy white ooze.

Etiology

Phoma nebulosa (Pers. ex S. F. Gray) Berk. (IMI 260690).

Control measures

As the disease is manifested under conditions of overcrowding of seedlings and excessive watering of seedbeds all the affected seedlings should be pulled out and destroyed and the watering regulated to a bare minimum. This will reduce further spread of the disease and the source of secondary inoculum, the splash dispersed spores. Two to three foliar sprays of Dithane M-45 (0.05% a. i.) applied at weekly interval were found to be effective against stem infection of ***G. arborea*** at Arippa.

Discussion

P. nebulosa, recorded for the first time from India, is a new pathogen record for ***G. arborea.*** It has earlier been recorded from U. K., Austria and Belgium on a number of hosts (Sutton, 1980).

2. SEEDLING BLIGHT

Occurrence

Seedling blight of ***G. arborea*** was recorded in nurseries at Peechi (Kerala Forest Res. Inst. Campus) and Arippa (Kerala Forest Dev. Corp., Trivandrum), where it caused large-scale mortality (ca. 70 per cent) in seedbeds and containers. The disease was noticed in 5-to 6-month-old seedlings during May/June after premonsoon showers. After the initial appearance of the disease in patches, it spread rapidly covering the entire seedbed, thus causing appreciable loss of nursery stock.

Symptoms

The stem of the affected seedlings showed prominent dark brown discolouration near the tip (Fig. 22 b) which extended under high humidity upto the ground level and also spread to petioles and leaves. The infection of stem and leaves resulted in blighting of shoots. Subsequently the stem and leaves of diseased seedlings were decayed on which a large number of acervuli developed producing abundant conidia in pink gelatinous mass. The infected seedlings died outright.

Etiology

Colletotrichum state of ***Glomerella cingulata*** (Stonem.) Spauld. & Shrenk and ***Fusarium solani*** (Mart.) Sacc. (IMI 260689).

Initially the infection was caused by ***Colletotrichum*** and later the affected stem colonized by ***F.solani*** which promoted the decay of seedlings.

Control measures

At Peechi the disease was effectively controlled by two applications of Bavistin (0.025% and 0.01% a.i.) given at weekly interval.

Discussion

Colletotrichum state of ***Glomerella cingulata*** and ***Fusarium solani*** are pathogens with wide host range causing varying types of diseases. This is the first report of their occurrence on ***G. arborea.***

PLANTATION DISEASES

1. COLLETOTRICHUM LEAF SPOT

Occurrence and severity

The disease was common both in nurseries at Peechi (Kerala Forest Res. Inst. Campus), Arippa (Kerala Forest Development Corp. (KFDC), Trivandrum) and in plantations at Arippa (KFDC), Onthupacha and Kottappara. Severe infection causing extensive defoliation (upto three-fourth of leaves of a seedling) was observed in 4-month-old container seedlings at Arippa. The severity of the disease was recorded as medium during 1980 and 1981 at Onthupacha; in 1982 it was low. At Kottapara low disease severity was recorded during 1980 and in later observations no disease was found (Table 22). Hence, as the trees grew older the incidence and severity of the disease declined.

Table **22.** **Incidence and severity of leaf spot caused by *Colletotrichum* state of *Glomerella cingulata* in two plantations of *Gmelina arborea* surveyed during 1980-1982**

Sl. No.	Locality	1980			1981			1982		
		% incidence	DSI[a]	DSR[b]	% incidence	DSI	DSR	% incidence	DSI	DSR
1.	Kottappara	35.63	0.36	L	0.0	0.0	Nil	0.0	0.0	Nil
2.	Onthupacha	0.0	0.0	Nil	24.44	0.21	L	33.87	0.36	L

Symptoms

Initially, brown small spots, 1-2 mm across, surrounded by light yellow halo appeared on lower mature leaves. Under high humidity the spots enlarged and became round to angular in outline, with light and dark brown concentric bands (Fig. 22 c). Severely infected leaves turned yellow and defoliated prematurely. Gradually the upper leaves also got infected and defoliated, thus leaving the bare stem with a few apical juvenile leaves.

Etiology

Colletotrichum state of ***Glomerella cingulata*** (Stonem.) Spauld. & Schrenk.

Pathogenicity

The pathogenicity of the isolate was confirmed on detached mature leaves of ***G. arborea,*** floated on benzimidazole solution (10 ppm). The leaves were surface sterilized in 0.25 per cent sodium hypochlorite solution and their abaxial surfaces inoculated with conidial suspension (10-15 conidia per drop of Pasteur pipette), prepared from a 10-day-old culture. The leaves were incubated at 25 + 2°C in large petri dishes under 16 h light period. Characteristic brown leaf spots developed seven days after inoculation, from which the pathogen was reisolated.

Control measures

In plantations the control of Colletotrichum leaf spot disease was not possible due to economic considerations. However, in nurseries the disease could be controlled. The disease attaining serious proportion could be evaded by avoiding crowding of seedlings which form conducive micro-environment for the disease development and its further spread from splash dispersed conidia, In nursery at Arippathe disease was controlled effectively by two to three foliar sprays of Dithane M-45 (0.05% a. i.) applied at week's interval or two spraysof Bavistin (0.02% a. i.).

2. CORYNESPORA LEAF SPOT

Occurrence

This foliar disease was observed during the monsoon (June-September) in 2-year-old outplanted saplings at Peechi (Kerala Forest Res. Inst. Campus). The disease, affecting only the mature leaves was recorded in low incidence.

Symptoms

The initial symptom was the appearance of irregular pale green water-soaked lesions which soon turned dark brownish-black spots with light brown centres(Fig. 22 d). Later these spots enlarged and coalesced to give rise to large necrotic area which led to premature defoliation.

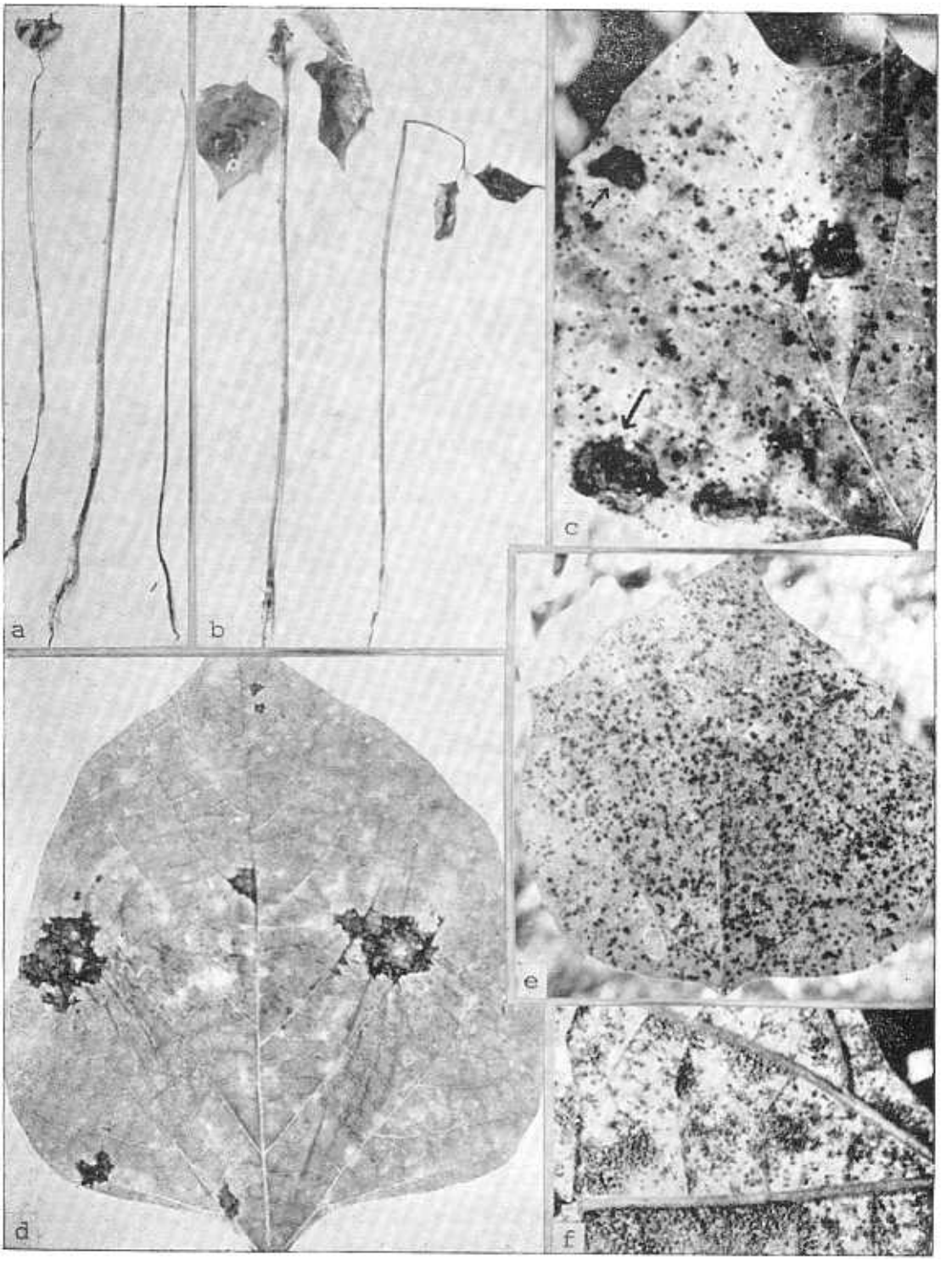


Fig. 22. Nursery and foliage diseases of *Gmelina arborea*. a, Seedling stem infection caused by *Phoma nebulosa*; b, Seedling blight caused by *Colletotrichum* state of *Glomerella cingulata* and *Fusarium solani*; c, Leaf spots caused by *Colletotrichum* state of *G. cingulata* (marked with an arrow); d, Leaf spot caused by *Corynespora cassiicola*; e and f, Leaf spots caused by *Pseudocercospora ranjita*.

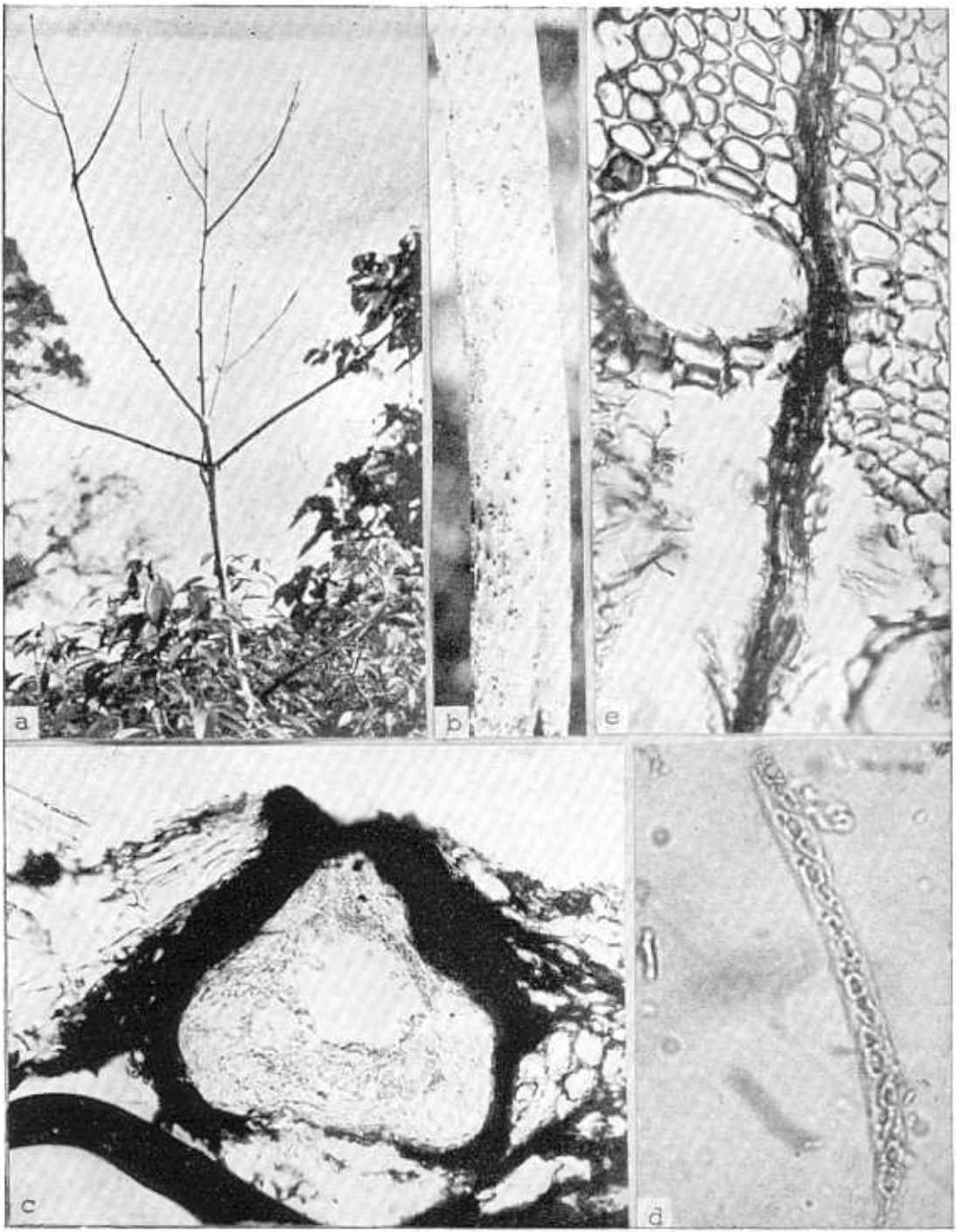


Fig. 23. Die-back of *Gmelina arborea* caused by *Griphosphaeria gmelinae* sp. nov. a, A young tree showing die-back. Note tapioca crop in the foreground; b, A twig showing numerous black ascomata of *G. gmelinae*; c, A vertical section through the perithecium (250 X); d, An ascus containing 8 bi-celled ascospores (760 X); e, Dark brown septate mycelium in the medullary rays of the wood of the affected stem (160 X).

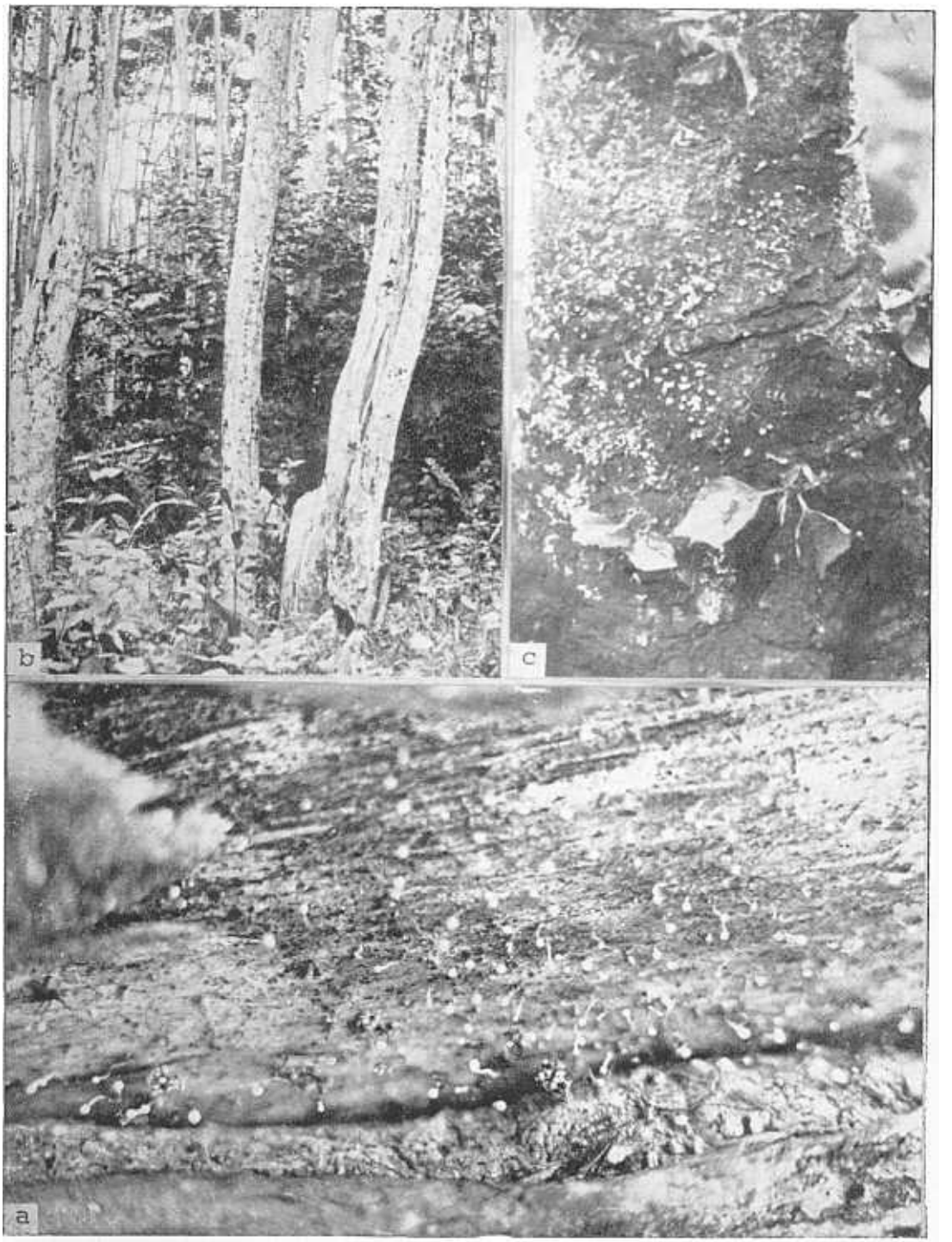


Fig. 24. Stem diseases of *Gmelina arborea*. a, Numerous conidiomata of *Thyronectria pseudotricha* on the canker; b, Partial stem decay caused by *Lentinus squarrosulus*; c, Stem rot caused by *Sclerotium* sp. Note abundant sclerotia and development of epicormic shoots at the base of the stem.

Etiology

Corynespora cassiicola (Berk. & M. A. Curtis) Wei (IMI 280237).

Pathogenicity

Pathogenicity of C. cassiicola was tested on leaves of 5-month-old container seedlings. Conidial suspension of the fungus, containing 5-6 conidia per drop of Pasteur pipette was prepared from a 7-day-old culture. The suspension was sprayed on abaxial and adaxial surfaces of leaves. The inoculated seedlings were transferred to a humidity chamber maintained at > 95% r. h. Typical leaf spots appeared on the third day of inoculation. The pathogen was reisolated from the leaf spots.

Discussion

This is the first report of ***C. cassiicola*** on ***G. arborea*** from India. Earlier, the pathogen has been recorded on ***Cassia*** sp. (Tamil Nadu), ***Carica papaya*** (Kerala), ***Corchorus capsularis*** **(Assam)** and ***Lycopersicum esculentum*** (Orissa) (Wei, 1950; Subramanian, 1952; Ellis, 1957).

3. PSEUDOCERCOSPORA LEAF SPOT

Occurrence and severity

Pseudocercospora leaf spot, a serious foliage disease, was found to be prevalent at Arippa (Kerala Forest Dev. Corp., Trivandrum), Onthupacha and Kottappara. The leaf spots appeared usually after the monsoon during the dry weather and caused considerable damage to the foliage due to premature defoliation. Severe infection with ca. 75 per cent of the leaves affected was recorded in a 3-year-old plantation at Arippa. However, at Kottappara and Onthupacha the severity of the disease was low (Table 23); incidence varied from 5 to 35 per cent.

Sl. NO.	Locality	1980			1981			1982		
		% incidence	DSI[a]	DSR[b]	% incidence	DSI	DSR	% incidence	DSI	DSR
1.	Kottappara	0.0	0.0	Nil	0.0	0.0	Nil	5.0	0.08	L
2.	Onthupacha	35.0	0.38	L	21.25	0.21	L	0.0	0.0	Nil

[a] DSI, Disease Severity Index
[b] DSR, Disease Severity Rating

Symptoms

Brownish-black circular to irregular spots bearing numerous conidia were confined initially on the abaxial surface (Fig. 22e). Due to profuse sporulation the spots resembled sooty mould. On the adaxial surface the spots appeared as light yellowish green patches. Smaller spots coalesced to form larger ones (Fig. 22 f). In severe cases the spots were also developed on the adaxial surface and petioles, Severely infected leaves turned yellow and fell prematurely.

Etiology

Pseudocercospora ranjita (Chaudhury) Deighton (IMI 269020).

Discussion

Leaf spot caused by ***P. ranjita*** has earlier been recorded from Lahing, Assain (Tandon and Chandra, 1963-1964). Elsewhere, it occurs in Kenya, Uganda, Brazil (Gibson, 1975) and Philippines (Quiniones and Dayan, 1981). This is the first report of ***P. ranjita*** on ***G. arborea*** from Kerala.

4. DIE-BACK

Occurrence

A serious die-back of ***G. arborea*** was observed in a 2-year-old 10 ha plantation at Arippa (Kerala Forest Development Corp., Trivandrum) and Onthupacha. The disease caused extensive die-back of branches and consequently death of trees; ca. 85 per cent of the trees were affected of which 50 per cent died. However, in the representative plantations the disease severity was either low as at Onthupatha or absent (Kottappara). At Onthupacha, the disease recorded only in 1981 and 1982, increased in incidence from 24 to 33 per cent during this period (Table 24).

Sl. NO.	Locality	1980			1981			1982		
		% incidence	DSI[a]	DSR[b]	% incidence	DSI	DSR	% incidence	DSI	DSR
1.	Kottappara	0.0	0.0	Nil	0.0	0 0	Nil	0.0	0.0	Nil
2.	Onthupacha	0.0	0.0	Nil	24.44	0.21	L	33.87	0.36	L

[a]DSI, Disease Severity Index

[b]DSR, Disease Severity Rating

Symptoms

Initially, the tender branches on top of the tree started dying from tip downwards. Gradually the infection spread to main stem killing it outright (Fig. 23 a). As a result of die-back some epicormic shoots developed from the basal part of the stem. But these shoots also died because of downward spread of infection in the stem. The wood of the affected trees showed light to dark brown discolouration. Numerous black coloured ascomata developed on the dead branches (Fig. 23 b).

Etiology

The pathogen causing die-back was identified as ***Griphosphaeria*** Hohn. (IMI 261570). It was assigned to a new species, ***G. gmelinae*** sp. nov. as it was found to be different in morphological details from other reported species (Figs. 23 c, d).

In the wood the fungus produced extensive dark brown septate mycelium, which mostly transversed through the parenchymatous medullary rays (Fig. 23 e) and attacked nearby vessels and fibres; affected cell walls showed browning.

Discussion

The die-back disease was found only in young plantations of ***G. arborea*** at Onthupacha and Arippa, whereas all the other nearby older plantations (5- to 7-year-old) were unaffected. In both the plantations a tall variety of tapioca was grown as a taungya crop. It is not understood whether it had some role to play in the manifestation of the disease. Since the disease caused extensive mortality during short period at Arippa it was considered as a potentially dangerous disease with possibility of spreading to other plantations. Hence, the KFDC was advised to clear-fell the plantation immediately and replant the area with some other species as a measure to contain the disease.

This is the first record of ***Griphosphaeria*** from India and die-back, a new disease record for ***G. arborea.***

5. THYRONECTRIA STEM CANKER

Occurrence

The disease was observed only at Kottappara in low incidence (< 1 per cent) during October 1982. During the observation period no mortality of the affected trees was observed.

Symptoms

Usually infection occurred on one side of the stem as a depression on which numerous anamorphs (conidial state) of the causal organism were produced. The

tissue of the depressed area developed into a canker. As the fungus invaded surrounding healthy tissues, the canker increased in length and width. During the dry period the bark got splitted and peeled off, exposing the dead wood. In the following monsoon anamorphs developed on the exposed dead wood (Fig. 24 a).

Etiology

Conidial state of ***Thyronectria pseudotricha*** (Schw.) Seeler.

Discussion

T. pseudotricha is a pathogen of wide host range. This is the first report of its occurrence on ***G. arborea.***

6. PINK DISEASE

Occurrence and severity

Low severity of pink disease was recorded during September/ October in both the plantations of ***G. arhorea*** at Kottappara and Onthupacha (Table 25). The incidence of the disease was higher at Kottappara (24 to 27 per cent) than at Onthupacha (4 to 7 per cent). Mostly the disease was confined to branches and occasionally it occurred on the main stem. Generally, no mortality of branch or the main stem was observed, except in one instance of a tender branch in a plantation at Kottappara.

1.	**Kottappara**	24.13	0.24	L	27.05	0.67	L	0.0	0.0	Nil
2.	**Onthupacha**	4.94	0.44	L	5.00	0.56	L	7.22	**0.83**	L

Symptoms

The pink disease was characterised by a canker on the branches or the upper part of the main stem. Death of shoots was observed rarely. Pink stage (perfect state) of the fungus was observed on the affected area.

Etiology

Corticium salmonicolor Berk. & Br.

Only cob web, pustule and pink (perfect) stages were observed on the affected area.

Discussion

The low incidence of pink disease in ***G. arborea*** plantations and formation of callus over the cankers possibly indicate its somewhat moderately resistant nature, as nearby plantations of E. ***tereticornis*** had ca. 90 per cent incidence with severe infection. Nevertheless, considering its potential to spread in epidemic proportion, ***Gmelina*** plantations need to be monitored regularly for the incidence of pink disease. This is the first report of pink disease on ***G. arborea.***

7. STEM DECAY

Occurence

Stem decay in living trees of ***G. arborea*** was noticed in a plantation at Kottappara. The disease appears to be unimportant as the incidence was < 1 per cent. No mortality of trees was observed.

Symptoms

Decay started on one side of the stem at the of the tree and proceeded upwards,(Fig. 24 b). The affected ,area became soft and pulpy. During the rainy season sporophores of the fungus were produced in clusters throughout the decayed area, but more at the base.

Etiology

Lentinus squarrosulus Mont.

Discussion

Generally infection by decay fungi in living trees takes place either through some mechanical injury or fire damaged tissue. After the pathogen is established on the injured wood it infects the healthy wood and causes decay, which may kill the trees if girdled completely. Though during the survey period no death of the affected trees was noticed, conidering that some trees had atleast half of the stem diameter decayed there is a possibility that they will be killed in due course.

This is the first occurrence of ***L. squarrosuluss*** ***G. arborea*** from India. Earlier, it has been reported from Nilgiri, Tamil Nadu on unidentified dead trunks (Natarajan and Manjula, 1976).

8. STEM ROT

Occurrence

Stem rot of a standing tree, was recorded a tKottappara during the monsoon of 1983. The tree was partially dead due to the infection.

Symptoms

The infection occurred at the base of the stem in the form of profuse mycelial growth accompanied with the formation of numerous fructifications (Fig. 24 c). The affected part of the stem became soft and showed sign of rotting. The surrounding healthy tissues produced epicormic shoots (Fig. 24 c).

Etiology

Sclerotium sp.

Discussion

Sclerotium is a common soil-borne pathogen causing stem rot in seedlings of many host plants (Browne, 1968). This is possibly the first report of ***Sclerotium*** causing stem rot in a tree. As the, disease was recorded in a single tree only it appears to be unimportant.

GENERAL DISCUSSION

So far only a few diseases have been reported on ***G. arborea*** from India., of these only one disease i.e., Pseudocercospora leaf spot was found in Kerala. Including this, a total of ten diseases were recorded in nurseries and plantations (Table 26); allare new records for the State and except Pseudocercospora leaf spot all are new diseases of ***G. arborea.***

In nurseries no damping-off, root rot or wilt were observed. Only three diseases viz. stem infection, seedling blight and Colletotrichum leaf spot were recorded The former two diseases were of serious nature as they caused high mortality of seedlings in seedbeds as well as in containers. The leaf spot, though caused severe premature defoliation, did not cause any mortality. It was found both in nurseries and plantations.

Eight diseases, three of foliage and five of stern were recorded in plantations, of these thoughPseudocercospora leaf spot caused premature defoliation insome plantations, only 'die-back paused by ***Griphosphaeria gmelinae*** was the most serious diseases, which resulted in heavy mortality in plantation at Arippa.

The stem infection and seedling blight diseases in nursery were controlled effectively by applicationof appropriate fungicides. However, control of die-back was not attempted due to economic considerations.

Table 26. Checklist of diseases of *Gmelina arborea* recorded in Kerala

Sl. No.	Disease	Pathogen(s)	New pathogen record for *G. arborea*	First record of pathogen from India
	A. *NURSERY*			
1.	Stem infection	***Phoma nebulosa*** (Pers. ex S. F. Gray) Berk.	+	+
2.	Seedling blight	i. ***Colletotrichum*** state of ***Glomerella cingulata*** (Stonem); Spauld. & Shrenk	+	–
		ii. ***Fusarium solani*** (Mart.) Sacc.	+	–
3.	Colletotrichum leaf spot	***Colletotrichum*** state of ***Glomerella cingulata***	+	–
	B. ***PLANTATION***			
1.	Colletotrichum leaf spot	***Colletotrichum*** state of ***Glomerella cinguleta***	+	–
2.	Corynespora leaf spot	***Corynespora cassiicola*** (Berk. & MA. C urtis) Wei	+	–
3.	Pseudocercospora leaf spot	***Pseudocercospora ranjita*** (Chaudhury) Deighton	–	-
4.	Die-back	***Griphosphaeria gmelinae*** sp. **nov.**	+	+
5.	Thyronectria stem canker	***Thyronectria pseudotricha*** (Schw.) Seeler	+	+
6.	Pink disease	***Corticium salmonicolor*** Berk. & Br.	+	–
7.	Stemdecay	***Lentinus squarrosulus*** Mont.	+	–
8.	Stem rot	***Sclerotium* sp.**	+	?

DALBERGIA LATIFOLIA

Dalbergia latifolia Roxb., a large deciduous tree, occurs scattered in mixed deciduous forests of Western Ghats; occasionally, it is found gregarious in patches. Regeneration of ***D. latifolia*** is quite common through root suckers, Attempts to raise rosewood in plantations have not been very successful mainly due to tall weeds which smother the slow growing seedlings. During the course of this study two plantations situated at Kannoth and Begur and a regeneration plot at Begur in Wynad Div. and other isolated trees in various localities were surveyed.

DISEASES

Only four foliar diseases were recorded on root-suckers and young and mature trees of ***D. latifolia.*** All the diseases are new host records

1. PHYSALOSPORA LEAF SPOT

Occurrence

Though this disease was ,recorded throughout Kerala it was found to be more common in northern parts. The leaf spots usually appeared in September/October just after the South-West monsoon (Figs. 3a, b).

Symptoms

Leaves of all the ages were found to be susceptible to infection. The leaf tissue of the affected area became yellowish-green and gradually turned into an amphigenous necrotic spot. Black, shiny dot-like ascomata developed in groups on the adaxial surface of these spots (Figs. 25 a,b). In severe cases five to eight such leaf spots could be present on a single leaflet.

Etiology

Physalospora sp. Niessl. (IMI 286904).

Since this species differs from the other known ones in morphological characters, it was designated as ***P. dalbergiae*** sp. nov. (Figs. 25 c,d).

Discussion

Physalospora is known to cause varying types of diseases, such as leaf spots, twig blight, die-back, stem canker and fruit rots, on different hosts. Common fungicides (Dithane M-45, Captan and Fytolan) have been found to be effective in controlling ***Physalospora*** diseases. Leaf spot caused by ***P. dalbergiae*** is a new disease record for ***D. latifolia.***

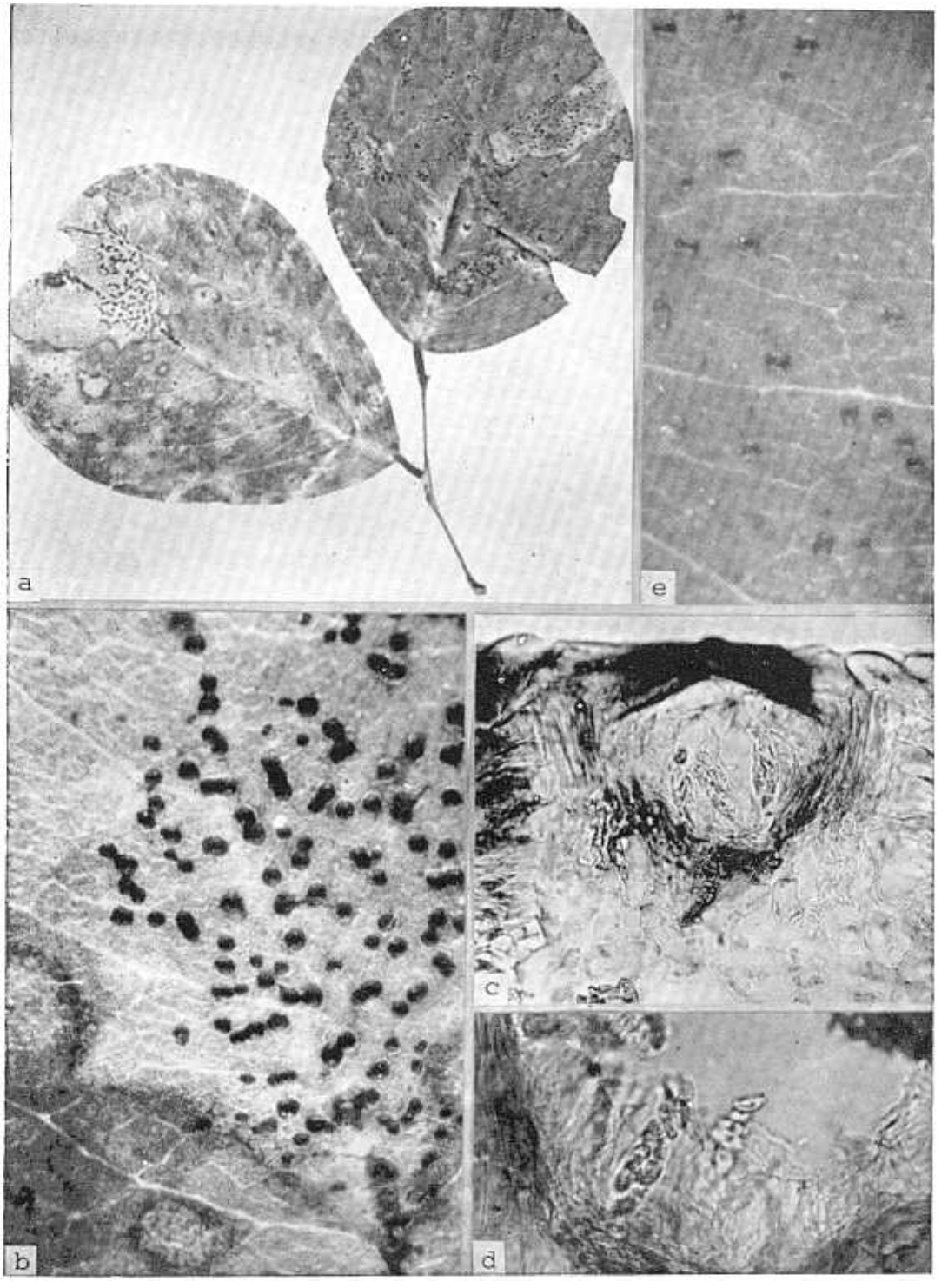


Fig. 25. Leaf spot diseases of a *Dalbergia latifolia*. a, Leaflets showing dual infection by *Physalospora dalbergiae* sp. nov. and *Phyllachora dalbergiae;* b, A leaf spot caused by *Physalospora dalbergiae*. Note abundant shiny dark coloured perithecia in the necrotic tissue; c, A vertical section of perithecium. Note prominent dark coloured clypeus (250 X); d, A magnified view of the perithecium to show biseriate 2-celled ascospores in the ascus (400 X); e, Ascomata of *Phyllachora dalbergiae*. Note the absence of necrotic tissue as compared to *Physalospora dalbergiae*.

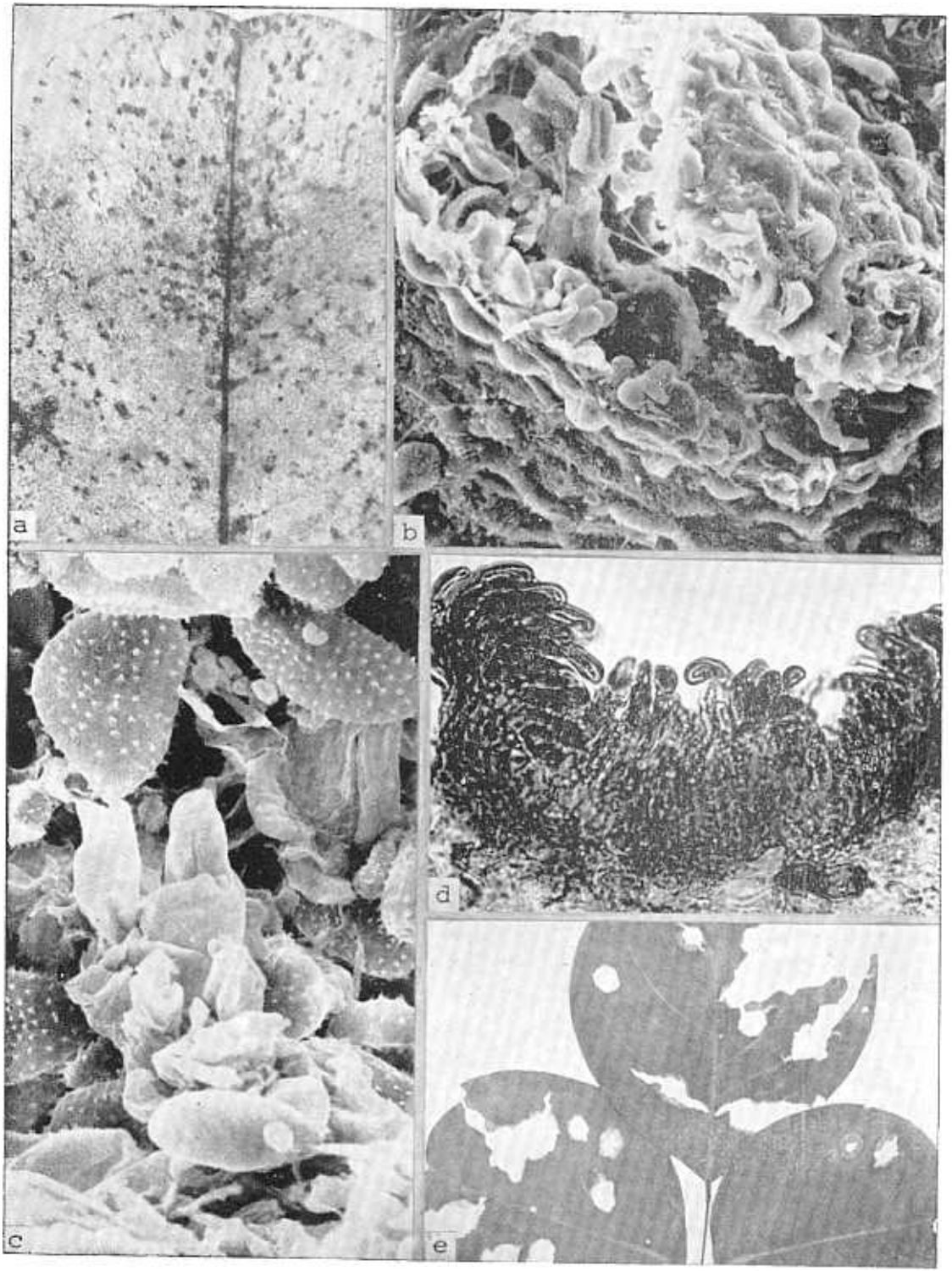


Fig. 26. Foliage diseases of *Dalbergia latifolia*. a, A part of the leaflet severely infected with leaf rust, *Uredo sissoo;* b, SEM of a uredinium (360X); c, SEM of urediniospores and paraphyses (1100X); d, A vertical section through a uredinium to show abundant marginal paraphyses; e, Leaf spots caused by *Colletotrichum* state of *Glomerella cingulata*.

2. PHYLLACHORA LEAF SPOT

Occurrence

The disease was recorded mostly Wynad plateau at Thettroad, Begur, Periya (Wynad Div.), Chethaleth and Mavinhalla (Kozkikode Div.) and Feechi. The incidence' of the disease was very low and it usually occured along with Physalospora leaf spot.

Symptoms

Initially, yellowish-green flecks developed on the upper surface of leaves; where shiny black, cushiony ascomata developed singly or in clustures (Figs. 25 a, e): The affected area of the leaf turned pale and gradually to light brown colour.

Etiology

Phyllachora dalbergiae Niessl. (IMI 293352).

Discussion

P. dalbergiae has earlier been recorded on ***Dalbergia sissoo*** from Calcutta and Pusa, Bihar (Saccardo, 1883). This is the first record of its occurrence on ***D. latifolia.*** Since this disease is uncommon and occurs in low incidence, it appears.to be unimportant (Brown, 1968).

3. LEAF RUST

Occurrence

The'leaf rust was widespread and observed during October-April. Premature defoliation due to severe rust infection on root suckers was recorded at Vattappoil (Wynad Div.). In general the disease appears'to be of minor importance.

Symptoms,

Minute, sparsely scattered uredeniosori (Fig. 26a), rusty brown in colour,< appeared during October/November on the abaxial surface of leaflets. Only in the case of severe infection some light greenish-yellow flecks developed **on** the adaxial surface. Severely rusted leaflets turned yellow and got defoliated prematurely.

Etiology

Uredo sissoo Syd. & Butler (IMI 273438) (Figs. 26 b - d).

Discussion

Uredo is widespread on ***D. sissoo*** in North India (Bakshi and Sujan Singh 1967). This is the first record of ***U. sissoo*** on ***D. latifolia*** from Indiia. The rust may be of serious concern only in young root suckers where it causes some premature defoliation.

4. COLLETOTRICHUM LEAF SPOT

Occurrence

The disease was recorded during the South-West monsoon (July-September) in Wynad Plateau at Chethaleth (Kozhikode Div.) and Begur (Wynad Div.), Peechi (Kerala For. Res. Inst. Campus), Kottappara and Pezhad (Malayattoor Div.). Severe infection caused considerable damage to the leaves by forming shot-holes; premature defoliation was observed occasionally.

Symptoms

Young as well as mature leaves were equally susceptible to infection. Small brown amphigenous necrotic spots, 1-2 mm. across, appeared scattered over the leaflets, but commonly along the margin and near the tip. Later, these spots coalesced to from large irregular reddish brown spots surrounded by light greenish yellow border. The necrotic tissue was often shed leaving shot-holes in the lamina (Fig. 26 e).

Etiology

Colletotrichum state of ***Glomerella cingulata*** (Stonem.) Spauld. & Shrenk.

Pathogenicity

Pathogenicity of the isolate was tested on detached leaflets of ***D. latifolia,*** floated on 5 ppm solution of gibberellic acid. The leaflets were inoculated on abaxial and adaxial surfaces with a conidial suspension (20-25 conidia per drop of Pasteur pipette) of ***Colletotrichum*** (7-day-old) at five places and incubated at 25 + 2°C under 16 h light cycle.

On the fifth day of inoculation greyish flecks developed which soon turned into reddish brown spots. The pathogen was reisolated from the spots confirming the pathogenic nature of the isolate.

Discussion

Colletotrichum leaf spot is a new disease recorded for ***D. latifolia.***

GENERAL DISCUSSION

Only four diseases, all affecting the foliage, were recorded on root suckers and young and mature trees of ***D. latifolia*** (Table 27). Leaf spots caused by ***Physalospora*** and ***colletotrichum*** are new diseases while for leaf rust and Phyllachora leaf spot ***D. latifolia*** is a new host, being reported earlier on ***D. sissoo.*** No stem decay fungi such as ***Polyporus gilvus*** (Schw.) Fries and ***Ganoderma lucidum*** (Leyss.) Karst. reported earlier by Bakshi (1975) were observed in Kerala.

None of the diseases recorded were serious though some of them caused premature defoliation.

Table 27. Checklist of diseases of Dalbergia latifolia recorded in Kerala

Sl. NO,	Disease	Pathogen	New pathogen record for *D. latifolia*	First record of pathogen from India
1.	**Physalospora leaf spot** *	***Physalospora dalbergiae* sp. nov.**	+	+
2.	**Phyllachora leaf spot** *	***Phyllachora dalbergiae* Niessl.**	+	–
3.	**Leaf rust***	***Uredo sissoo*** Syd. & Butler	+	–
4.	**Colletotrichum leaf spot** *	***Colletotrichum*** State of ***Glomerella cingulata*** (Stonem.) Spauld. & Shrenk	+	–

+ Diseases also recorded on seedlings and root suckers.

OCHROMA PYRAMIDALE

Ochroma pyramidale (Cav. ex Lam.) Urb., a native of tropical America, yields one of the lightest wood used in aviation industry, rafts, etc. Small-scale plantations of **O. *pyramidale*** were raised in Kerala during the seventies. Since then the demand of balsawood has declined considerably and hence, planting of ***O. pyramidale*** has been discontinued.

A list of the balsa plantations surveyed is given in Table 28.

Table 28. List of representative plantations of ***Ochroma pyramidale*** surveyed for incidence and severity of diseases during **1980-1982.**

Sl. NO.	Locality	Altitude (m above msl)	Forest Divn.	of plantation (ha)	No.of observation plots surveyed	Year of planting	Age of trees at survey in 1980 (years)
1.	Erampadam	50	Nilambur	10.0	5	1978	2
2.	Kondodi (Vayakara)	60	Konni	5.0	3	1978	2

PLANTATION DISEASES

No diseases were observed in nursery. In plantations, a serious die-back disease was recorded causing upto 50-60% mortality. Occasionally trees were also found to be infested with ***Dendrophthoe*** sp. Besides, a disease of unknown etiology causing deformity of shoots such as stunting of internodes and littling of leaves was also observed.

1. DIE-BACK

Occurrence and severity

The die-back was observed in all balsa growing areas of the State. It was most prevalent in plantations at Erampadam and Kondodi, the highest incidence (>98 per cent) being at Erampadam where ca. 60 per cent of the affected trees died (Table 29). In both the plantations where the severity remained medium, the incidence increased from 1980 to 1981.

Symptoms

The disease was characterised by crinkling and stunting of foliage and die-back, and often bunchy appearance of shoots. The infection, initiated either on the

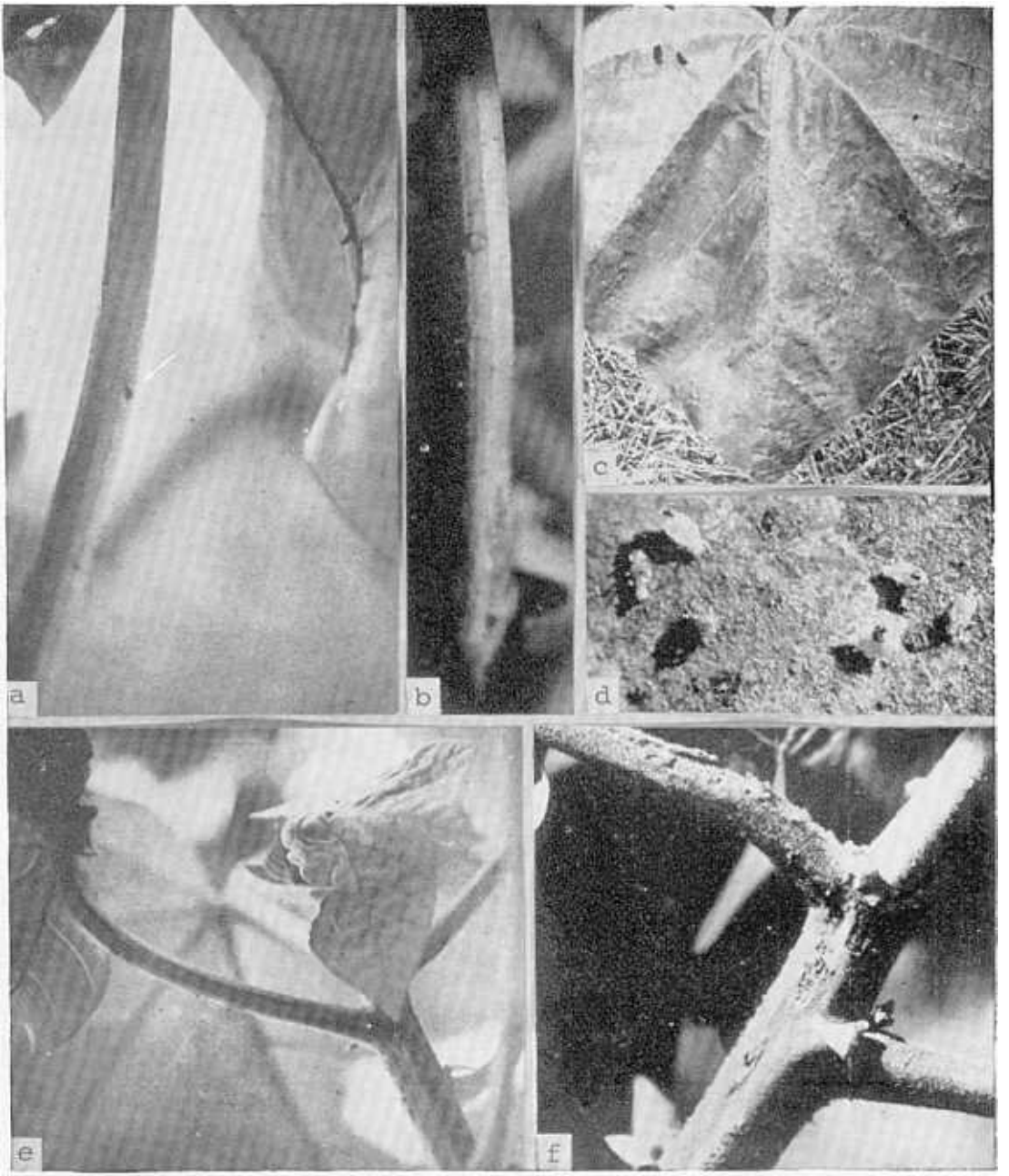


Fig. 27. Die-back of *Ochroma pyramidale*. a, An ant in the process of making an injury on the tender petiole (marked with an arrow); b, Oozing of mucilage at the site of the injury; c and d, Unidentified hemipteran bugs feeding on leaf; e, The injured area becomes brown as seen on the leaf lamina, petiole and stem; f, Browning develops into a canker on petiole and stem.

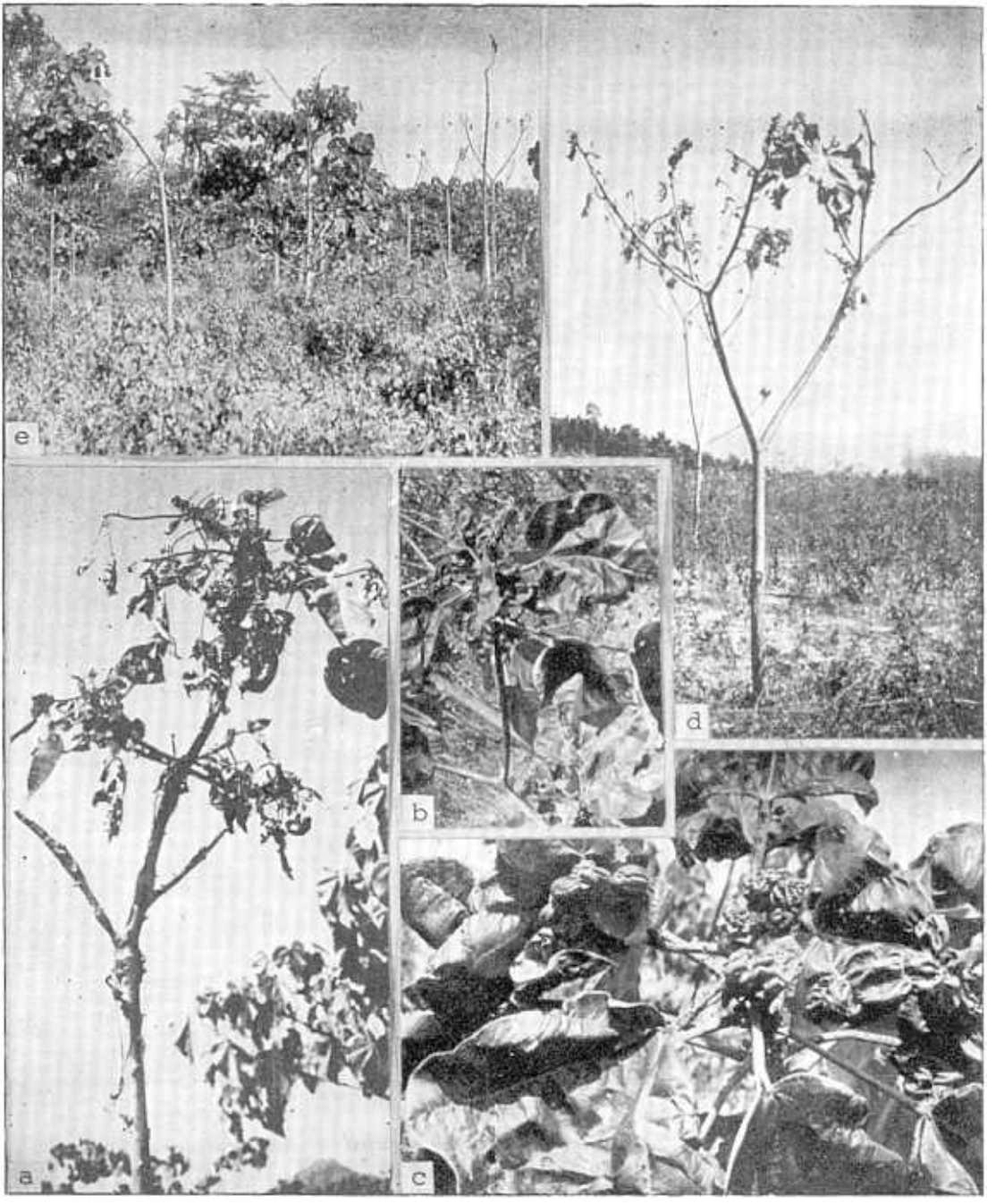


Fig. 28. Die-back of *Ochroma pyramidale*. a, Die-back of terminal shoot results into development of epicormic shoots which later also get infected; b and c, Crinkling of leaves; d, A partially dead tree. Note dead trees in the back ground; e, A view of the plantation at Erampadam showing large-scale mortality of trees.

tender stem near the apex, petiole or veins of leaves, was manifested through a series of wounds made by small red ants, or other sap feeding insects (Figs. 27 a–d), whose activity was found to be more during the dry period (December-March). The ants punctured the epidermis and consequently droplets of mucilage oozed out (Figs. 27 a, b) on which they fed. Within a week all the wounded areas turned necrotic and showed prominent browning (Fig. 27 e), which extended even to surrounding green tissues. These brown lesions developed into small cankers, which coalesced to form large cankerous areas (Fig. 27 f). The affected leaves showed extensive crinkling and stunting (Figs. 28 b, c). Often infection of petioles (Fig. 27 f) resulted in premature defoliation. In the initial stages only the tissues of outer bark i.e., cortex; phloem were killed but later it extended to xylem also. Due to the extensive cankers the apical bud whithered and died. As a result, the axillary buds sprouted and developed into shoots thus giving rise to bunchy appearance of shoots (Fig. 28a). Subsequently, the infection from the infected shoots proceeded downwards thus killing other healthy shoots. Occasionally a few apparently healthy shoots survived for sometime (Fig. 28 d) but eventually they were also killed, thus killing the whole tree (Fig. 28 e). It took about two years to kill the trees.

Table 29. **Incidence and severity of die-back caused by *Calonectria rigidiuscula* and *Fusarium moniliforme* in two plantations of *Ochroma pyramidale* surveyed during 1980-1982**

Sl. No.	Locality	1980			1981			1982		
		% incidence	DSI[a]	DSR[b]	% incidence	DSI	DSR	% incidence	DSI	DSR
1.	Erampadam	94.08	1.93	M	98.25	2.78	,M	—	—	—
2.	Kondodi	84.69	1.24	M	87.38	2.41	M	—	—	—

Over the canker causal organisms sporulated profusely, which appeared in the form of white-pinkish powdery mass. The ants were seen in the crevices of the cankers of dead trees and carrying the spore mass. Possibly, ants feeding on mucilage, produced on tender shoots, transfer the inoculum to wounds thus spreading the infection.

Etiology

Calonectria rigidiuscula (Berk. & Br.) Sacc. (IMI 257549) conidial state, ***Fusarium decemcellulare*** Brick. and ***Eusarium moniliforme*** var. ***subglutinans*** Wollenw. &

Reink. (IMI 257550). In contrast to most ***Fusarium*** species, ***E. decemcellulare*** is usually referred to in the literature under the name of its perfect) state, ***C. rigidiuscula.***

Pathogenicity

The pathogenicity of the isolates was confirmed on side branches of 4-year-old healthy trees growing in Banana Res. Farm at Kannara of Kerala Agricultural University. Twenty tender shoots, ten each wounded and unwounded, were inoculated either separately or with both the fungal organisms. For wound inoculation the stem near the apex was pricked 20 times using a sterile needle. A disc, 5mm in diam., bearing mycelium and conidia, taken from a 15-day-old culture of the test fungus, was placed mycelial side facing the injury. This was covered with moist sterile cotton swab,which was fastened lightly with twine so that the disc was not displaced from the wounded site. The inoculated shoot was enclosed in a large polythene bag, the inner surface of which was sprayed with sterile water. The shoots without wound were inoculated the same way, except for the pricks. Observations on development of the disease were recorded regularly.

No disease developed in unwounded shoots even after a year. The disease first appeared in six wound inoculated shoots where both the test fungi were used together. The wounds developed small dark brown cankers which later extended upto the terminal bud. The shoots were killed within six months of inoculation. In the case of ***C. rigidiuscula*** only three shoots developed cankers and one shoot died after six months. None of the shoots inoculated with ***F. moniliforme*** developed any disease. This possibly indicates a secondary role of the latter pathogen in the development of the disease. From the dead shoots the respective pathogen(s) were re-isolated in pure culture.

Discussion

Pathogenicity tests confirm the role of both the fungi, when inoculated together, in causing die-back of ***O. pyramidale.*** The main pathogen appears to be ***C. rigidiuscula*** and ***E. moniliforme*** possibly facilitates the disease development in the presence of former due to synergestic effects. However, the low percentage of infection in inoculated shoots may possibly be attributed to improper conditions (accumulation of excessive moisture inside the polythene bag due to transpiration of leaves) during the process of infection and subsequently disease development.

Both, ***C. rigidiuscula*** and ***F. rnoniliforme,*** are tropical species pathogenic to plants belonging to many families (Booth, 1971). ***C. rigidiuscula*** mainly a wound (insect injury or other minor diseases) parasite, is known to cause die-back in a number of dicotyledonous plants such as ***Ceibapentandra*** in Ghana (Piening, 1962); 'Robusta' coffee in Ivory Coast(Meiffren and Belin, 1960), ***Hevea brasiliensis*** in Congo

(Zaire) (Spaulding, 1961) and cocoa in Cameroon, Ghana, Ivory Coast and Nigeria (Thresh, 1960).

A similar example, where ants or other sap feeding insects play an important r d e in infection, is cocoa. In cocoa, die-back caused by ***C. rigidiuscula*** is also through capsid injury (Thresh, 1960). The use of suitable insecticide combined with copper spray has given good control o 'Robusta' coffee canker in Ivory Coast (Meiffren and Belin, 1960) and of cocoa die-back in Cameroon, Ghana, Ivory Coast andNigeria.

This is a new disease record for ***O. pyramidale.***

DISEASE OF UNKNOWN ETIOLOGY

1. MALFORMATION OF SHOOTS

Occurrence

The disease was recorded at Erampadam and Kerala Forest Res. Inst. Campus. The affected shoots did not die.

Symptoms

The affected plants produced two distinct types of abnormalities. In one type, the shoots became stunted due to shortening of internodes and smalling of leaves (Figs. 29a, c). The latter did not show any crinkling or vein bending. Instead of normal branching, the main stem produced stunted shoots giving a bushy appearance to the plant.

The other type did not show any stunting, but only malformation of foliage (Fig. 29 b). The affected leaves became very small, leathery and formed cuplike structures due to prominent vein-bending (Fig. 29 d); some puckering was also observed.

Etiology

The disease is possibly caused either by a virus or a. mycoplasmalike organism. For establishing identity of the causal organism detailed investigations are needed.

GENERAL DISCUSSION

No disease was observed in the nursery. Of the three diseases recorded in plantations (Table 30), die-back manifested through the ants and sap feeding insects was most serious causing upto 60 per cent mortality. The disease caused by two fungal pathogens is a new record for balsa. Occasionally trees at Erampadam were found to be infested with ***Dendrophthoe*** sp. The incidence of disease of unknown etiology, malformation of shoots, was also very low.

Table 30. **Checklist of diseases of *Ochroma pyramidale* recorded in Kerala**

Sl. No.	Disease	Pathogen(s) / Angiospermic parasite	New pathogen record for *O. pyramidale*	First record of pathogen from India
1.	Die-back	(i) *Calonectria rigidiuscula* (Berk. & Br.) Sacc.	+	+
		(ii) *Fusarium moniliforme* var. *subglutinans* Wollenw. & Reink.	+	–
2.	Mistletoe	*Dendrophthoe* sp.	+	–
3.	Malformation of shoots	Unknown etiology	–	–

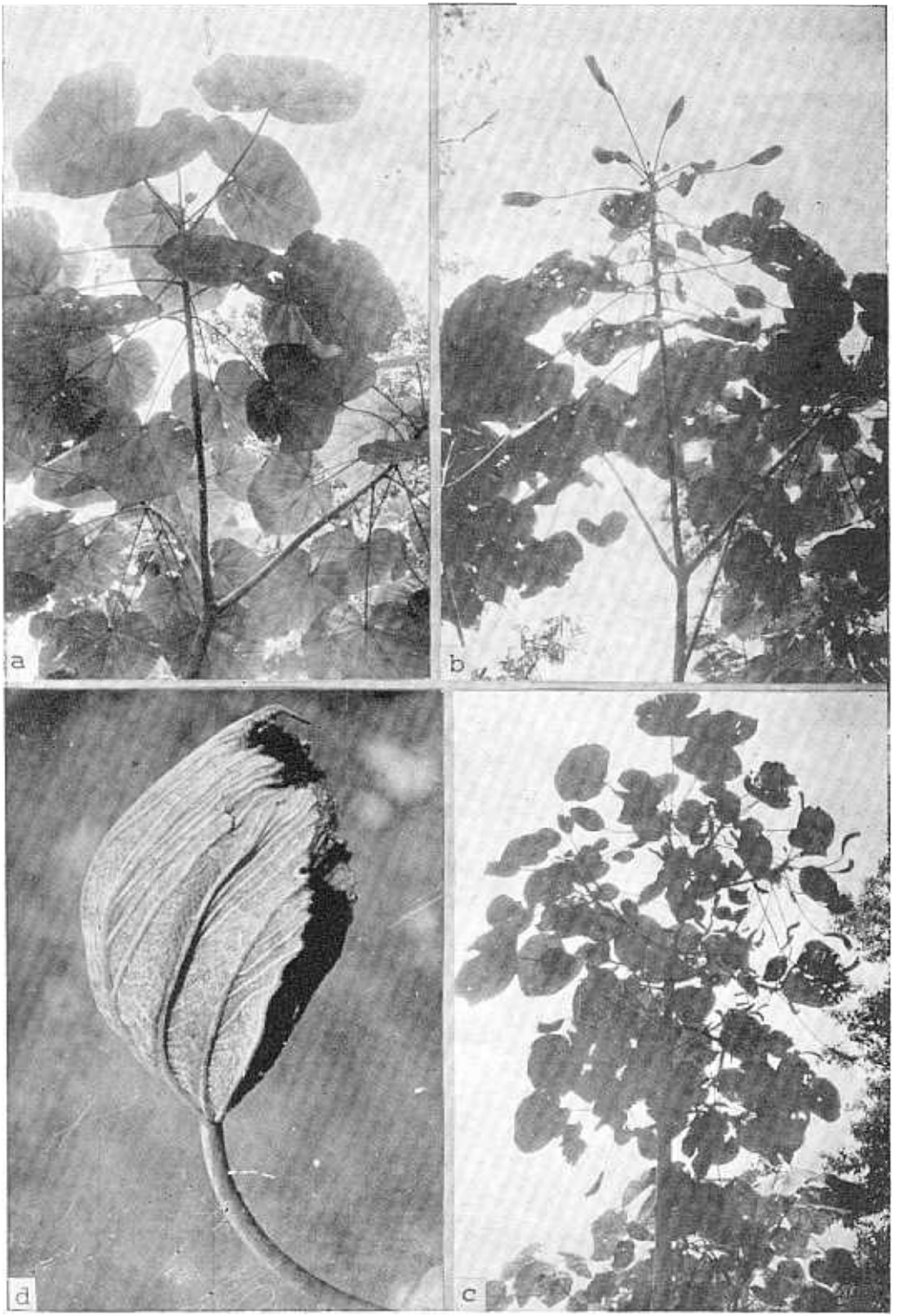


Fig. 29. Malformation of shoots of *Ochroma pyramidale*. a, A healthy plant; b, A tree affected with malformation of leaves; c, A tree showing stunting of internodes, littling of leaves and malformation of leaves; d, A malformed cup-like leaf.

EUCALYPTUS

Eucalypts have emerged as the most widely planted exotic forest tree species in Kerala because of their fast growth, adaptability and important end uses in pulp and paper industries. In Kerala, ***Eucalyptus*** spp. have been planted on a limited scale at high altitudes 1000-1500m above msl) for several decades. During 1961-1967 appreciable areas of grasslands in high lands of Pamba and Munnar (1000-1200 m above msl) were converted to plantations of ***E. grandis*** Hills ex Maiden, seeds of which were obtained from neighbouring Nilgiri Hills, Tamil Nadu. Subsequently ***E. grandis*** and ***E. tereticornis*** Sm. were planted intensively in clear-felled areas of natural forests of low land regions to meet the requirements of pulp and paper industries in the State. So far about 38,000 ha have been planted with eucaIypts; ***E. grandis*** 14,779 ha and ***E. tereticornis,*** 23,797 ha (Karunakaran, 1982).

A list of plantations of ***Eucalyptus*** spp. surveyed is given in Table 3I.

NURSERY DISEASES

Phaeoseptoria leaf spot and little leaf diseases, also recorded in nurseries, are described in detail under plantation diseases,

Diseases in seedbeds

As most of the nursery diseases are soil-borne, potential pathogens affect the eucalypt seeds immediately after they begin to germinate. Various diseases, namely damping-off, web blight, seedling blight, seedling wilt, root-rot and leaf spots appeared almost in succession. Occurrence of web blight, seedling blight and seedling wilt overlapped. Except for the seedling blight, the other diseases continued to affect the seedlings till they were pricked out into polythene containers. Damping-off, web blight and seedling blight were the major widespread diseases,

1. DAMPING-OFF

Damping-off, the first disease to appear in seedbeds can cause considerable loss before and after the emergence of seedlings of ***E. grandis*** as well as ***E. tereticornis.***

a. Pre-emergence damping-off

Occurrence

The disease was recorded in ***E.grandis*** at Periya (Wynad Div.) where the seedbeds were over watered and a dark shade was provided in the nursery. Due to the disease only about 20 per cent of the seedlings emerged.

Table 31. List of representative plantations of ***Eucalyptus*** spp. surveyed for disease occurrence during 1980-1982

Sl. No.	Locality	(m	Forest	Area of plantat-	No. of observation plots surveyed	Year of planting	Age of trees at survey in 1980 (years)
	E. grandis						
1.	Thrissillery	**850.0**	Wynad	**54.67**	**8**	**1977**	**3**
2.	Mavinhalla (S. Battery)	**850.0**	Kozhikode	62.85	**5**	**1977**	**3**
3.	Noolpuzha	**925.0**	„	—	**3**	**1978**	**2**
4.	Papathishola	**152.5**	Munnar	—	**5**	**1978**	**2**
5.	Mullachal	**175.0**	KFDC * Trivandrum	9.0	**3**	**1978**	**2**
	E. tereticornis						
6.	Varavoor	125.0	Trichur	102.0	5	**1976**	**4**
7.	Kottappara	**50.0**	Malayattoor	—	5	**1977**	3
8.	Pezhad	**50.0**	Malayattoor	—	3	**1978**	2
9.	Anakulam	160.0	Punalur	**106.85**	5	**1977**	3
10.	Mullachal	**175.0**	KFDC Trivandrum	**10.0**	3	**1978**	2
	E. globulus						
11.	Silent Valley	2040.0	Munnar	3.0	3	**1978**	2

Symptoms

The disease was usually characterised by rotting of the just emerged radicle and subsequently the cotyledons inside the seedcoat. It was noticed within 2 to 3 days of sowing.

Etiology

Rhizoctonia solani Kuhn.

Discussion

High soil moisture and dark shade provided its the nurseries contributed to the development of pre-emergence damping-off. Pre-emergence damping-off, though uncommon, often goes undetected because it is misidentified as failure of germination of “poor seeds”.

b. Post-emergence damping-off

Occurrence

Post-emergence damping-off, which occurred more commonly than the pre-emergence disease, appeared within a week of emergence of seedlings. The disease spread rapidly with increasing soil moisture, often resulting from excessive watering of beds. Mortality of seedlings was observed for the first two to three weeks only. It appears that thereafter the seedlings developed resistance.

Symptoms

The infection caused a collapse of the stem tissues marked by a water-soaked constricted area at the soil level causing the seedlings to fall over; such seedlings failed to survive. The damping-off usually occurred roughly in circular patches (Fig. 30 a) in which the most recently dead seedlings were on the periphery.

Etiology

A total of nine fungi were isolated from damped-off seedlings:

R. solani Kuhn. state of ***Thanatephorus cucumeris*** (Frank.) Donk on ***E. grandis*** and ***E. tereticornis.***

ii. ***Pythium deliense*** Meurs. (IMI 267018, 281614) on ***E. grandis*** and ***E. tereticornis.***

iii. ***P. myriotylum*** Drechsler (IMI 268319, 268320) on ***E. tessellaris*** and ***E. grandis.***

iv. ***P. spinosum*** Saw. (IMI 276611,276608, 276609, 276605) on ***E. grandis.***

v. ***Cylindrocladium quinqueseptatum*** Boedijn & Reitsma (IMI 246473) on ***E. grandis.***

vi. ***C. ilicicola*** (Hawley) Boedijn & Reitsma (TMI 250214, 250215) on ***E. tereticornis*** and ***E. grandis.***

vii. ***C. floridanum*** Sobers & Seymore (IMI 250219) on ***Eucalyptus*** hybrid 'FRI-4'.

viii. ***C. parvum*** Anderson (IMI 250221) on ***E. grandis*** and ***E. tereticornis.***

ix. ***Fusarium oxysporum*** Schlecht. (IMI 269013) on ***E. grandis*** and ***E. tereticornis.***

Pathogenicity

Pathogenicity of all the isolates was confirmed as outlined under seedling blight.

Control measures

Remedial measures for damping–off have been standardized in nursery trials (Anon., 1984). As soon as the disease is noticed watering of beds should be reduced to a bare minimum. It can even be stopped for a day or two depending upon the climatic conditions of the area. This minimises the incidence and spread of damping-off. If dense thatching is used, it should be made sparse to allow some scattered light over the beds.

For controlling the disease Bavistin (0.01 % a.i.), Dithane M-45 (0.01% a.) and Emisan-6 (0.0025% a. i.) should be applied separately as soil drench at an interval of four hours, in place of normal watering. After the treatment, watering should be regulated (both quantity and frequency) to prevent build up of excess soil moisture.

Discussion

Damping-off of eucalypt seedlings, probably one of the most serious of all nursery diseases, appears to be a complex since it is caused by a number of pathogens. ***R. solani*** was the main pathogen responsible for damping-off followed by ***Cylindrocladium, Pythium*** and ***Fusarium. R. solani*** has already been considered as the important cause of damping-off in nurseries Of other species in India and elsewhere; ***Pythium*** and ***Fusarium*** are recorded less commonly (Ram Reddy, 1969). ***Rhizoctonia*** spp. are less specific in their behaviour and are reported to be favoured sometimes by high moisture and sometimes by low, perhaps because water and oxygen requirements are strain dependent (Vaartaja and Morgan, 1961). ***Cylindrocladium*** spp. which cause several kinds of diseases among eucalypt seedlings, are also known to be associated with damping-off (Cox, 1954). Among ***Cylindrocladium*** species C. ***scoparium*** has been reported to cause damping-off of eucalypt seedlings, besides causing other diseases in Argentina (Arruda, 1943), Brazil (Batista, 1951), India (Bakshi, 1967) and Japan (Tereshita and Takai, 1955). Also it has caused a total loss of seedlings in some nurseries in Latin America (Reis and Hodges, 1975). ***C. quinqueseptatum*** which was the major species in Kerala has earlier been reported only from Brazil (Figueiredo and Namekata, 1967). C. ***ilicicola, C. floridanum*** and ***C. parvum*** have been reported for the first time to cause damping-off of ***Eucalyptus*** seedlings in India by Sharma and Mohanan (1982).

Although two species of ***Pythium*** i.e., ***P. deliense*** (on ***E. tereticornis*** and ***E. grandis)*** and ***P. myriotylum*** (E. ***tessellaris)*** were found to be associated with damping-off of eucalypt seedlings, their occurrence was uncommon. These observations are supported by those of Roth and Ricker (1943) who found that damping-off due to ***Pythium*** sp. occurs less frequently in well drained soils and at high **soil** temperatures during the early periods of seedling growth.

Occasionally ***Fusarium oxysporum*** was also isolated from damped-off eucalypt seedlings. Although Gibson (1975) reports appreciable loss from damping-off of ***E. citriodora*** by ***Fusarium*** sp., its role as a primary pathogen is uncertain (Cox, 1954; Vaartaja and Cram, 1956). Cox (1954) concluded that ***Fusarium*** though freely isolated from damped-off pine seedlings, appeared to be a secondary invader found on seedlings showing advance decay. Similarly, Vaartaja and Cram (1956) found that ***Fusarium*** spp. were commonly associated with damping-off of Scots pine and

caragana but pathogenicity trials indicated only ***R. solani*** and ***Pythium*** to be the main pathogens.

Hawkins and Harvey (1919) and McClure and Robbins (1942) have shown that resistance of young seedlings to infection is correlated with the difficulty of puncturing the cell-wall. this is largely due to the amount of secondary thickening and lignification. Damping-off fungi are, therefore, mainly restricted in their attack of young tissues such as root tips and outright killing of the host will occur only where the hypocotyl region is still succulent and unlignified. The fungi that cause damping-off do not usually attack seedlings unless conditions for their development are favourable or the conditions for the growth of seedlings are poor (Leach, 1947; Griffin, 1958; Rowan *et al.*, 1972) Excessive soil moisture due to overwatering, excessive shade, high seedling density and high organic soil contents are the main factors contributing to initiation and spread of damping-off (Roth and Ricker, 1943; Vaartaja, 1952; Gibson, 1956).

Pre-sowing insecticidal treatments are also known to play some role in the occurrence and development of diseases. Damping-off of cauliflower caused by ***Pythium*** and Rhizoctonda is greatly enhanced by the pre-sowing treatments with Chlordane (1, 2, 3, 4, 5, 6, 7, 8, 8-Octachloro-3a, 4, 7, 7a tetrahydro-4, 7-methano-indane) (Grossmann and Steckhan, 1960). Our observations (Sharma and Mohanan. 1981 b) confirm these findings. The incidence of disease caused by ***R. solani*** and ***Pythium,*** in Chlordane treated beds (pre-sowing) of I98I· 1982 chemical control trials for ***Cylindrocladium*** in eucalypt seedlings, was significantly higher as compared to the untreated ones conducted during 1980-1981.

2. CYLlNDROCLADIUM COTYLEDON INFECTION

Occurrence

The disease was recorded on cotyledons of 2- to 3-week-old seedlings of *E. **grandis*** in Wynad plateau at Chandanathode (Wynad Div.), and Kodali (Kozhikode Div.). Severe infection resulted in mortality of young seedlings.

Symptoms

Minute circular, greyish water-soaked lesions appeared on cotyledons which expanded and turned as pale brown spots after drying. Under profuse watering conditions the spots enlarged further and covered the entire area of cotyledon. Often the infection spread to the tender stem killing the seedlings outright.

Etiology

Cylindrocladium quinqueseptatum Boedijn & Reitsma

Control measures

A foliar drench of Bavistin 0.01% (a. i.) is found to be effective in controlling cotyledon infection.

3. WEB BLIGHT

Occurrence

Web blight of eucalypt, usually occurring in irregular patches in seedbeds, was widespread in nurseries. Young seedlings were killed outright but older ones remained alive for some time before dying. The disease, generally appeared within two weeks of seedling emergence, was recorded in ***E. grandis*** and ***E. tereticornis.***

Symptoms

Initially the mycelium of the pathogen emerging from the soil grew up on the stem and over the leaves of a few seedlings (Fig. 30 b) and spread to others (Fig, 30c), invading the leaf tissues rapidly. The mycelial strands, which were initially hyaline, became light brown and branched extensively. Leaves of infected seedlings developed water-soaked lesions and wilted. Later. they became necrotic and dried up (Fig. 30d). The stem showed characteristic light greyish-black necrotic lesions. The pathogen often produced off-white to light brown irregular sclerotia on the affected stem and leaves of older seedlings (4-5 months old). The perfect stage, ***Thanatephorus cucumeris,*** occasionally developed on stems of diseased seedlings during the rainy season (June-August) with temperatures varying from 20 to 23°C and 90 to100% r. h.

Etiology

Rhizoctonia solani Kuhn. state of ***Thanatephorus cucumeris*** (Frank.) Donk (IMI 257894).

Pathogenicity

The pathogenicity of the isolate was tested by transplanting seedlings of E. ***grandis*** and ***E. tereticornis,*** raised in pasteurised soil, in separate trays where the soil had been mixed with a corn meal sand culture of the fungus containing abundant microsclerotia. All trays were kept at 24 to 27°C, some in a humid chamber at 95 per cent r. h., others in the laboratory at 65 to 80 per cent r. h. Damping-off occurred in all inoculated trays but web blight of the seedling foliage only developed at higher humidity. Both species of eucalypt were found to be equally susceptible. No root infection was detected from any of the affected seedlings.

Control measures

As soon as the disease is noticed the watering should be minimised. This will facilitate in checking the spread of the disease. Emisan-6 (0.005% a. i.) applied as soil drench is found to be highly effective in controlling web blight.

Discussion

The pathogen has a world-wide occurrence and is also a common damping-off pathogen. Web blight, which has been reported to occur commonly in other hosts, is a new record for ***E. tereticornis*** and ***E. grandis*** in India. Earlier, it has been reported only from Zaire, Africa (Anon., 1955). Since no root infection was observed in the affected seedlings this strain of ***R. solani*** may be an aerial one as also has been recorded by Baker (1970). Our field observations indicate that the disease is greatly influenced by nursery practices. Its spread is very rapid under high soil moisture conditions, especially when the seedlings are crowded and the r. h. is > 90%. Web blight may persist till the seedlings are pricked out into polythene containers. This way it could pose a potential threat to the stock of seedlings if proper nursery management practices related to watering quantity and frequency, and sowing rate are not followed.

4. SEEDLING BLIGHT

Occurrence

Seedling blight, usually observed in one-month-old seedlings of ***E. grandis*** and ***E. tereticornis*** may result in heavy mortality (Fig. 30 e). The disease is widespread in Kerala.

Symptoms

lnfection on the stern near the soil level, just above the root collar (collet) region, was the cause of seedling blight (Fig. 30 f). The affected area became greyish brown killing the tissues and consequently such seedlings dried up. The symptoms produced by ***Cylindrocladium*** and ***Rhizoctonia*** were similar, except seedlings affected with the former showed profuse conidial growth under high humid conditions which helped in increasing the inoculum potential of the pathogen with consequent rapid spread of disease.

Etiology

i. ***Rhizoctonia solani***
ii. ***Cyiindrocladium quinqueseptatum***
iii. ***C. ilicicola***
iv. ***C. parvum***
v. ***C. clavatum*** Hodges & May (IMI 270185)
vi. ***C. camelliae*** Venkataramani & Venkataram (IMI 262979)
vii. ***C. scoparium*** Morg.

Pathogenicity

For testing the pathogenicity of the above isolates, cultures raised on corn mea? sand medium were blended and mixed separately with sterile soil in aluminium trays. ***E. grandis*** seedlings (8, 10 and 12-week-old), raised in sterile soil, were transplanted after washing the root system thoroughly with sterile water. First damping-off occurred in 8-week-old seedlings followed by seedling blight in 10- and 12-week-old seedlings. Typical infection of root collar was observed in wilted seedlings.

Control measures

Seedling blight of eucalypt seedlings is controlled effectively by Bavistin 0.0I% (a. i.) applied as foliar and soil drench (Anon., 1984). If the disease persists due to excessive soil moisture another application of the same fungicide may be given.

Discussion

Seedling mortality in eucalypt nurseries due to blight disease caused by ***Cylindrocladium*** is a serious problem, especially in warm humid areas of the State. Since only ***C. scoparium*** (Goa and Dehradun) and ***C. quinqueseptatum*** (Kerala and Dehradun) have been reported earlier to be the cause of this disease (Bakshi, 1976) association of ***C. ilicicola,*** C. ***camelliae, C. parvum, C. clavatum*** and ***R. solani*** with the disease is new record from India. ***C. camelliae*** has earlier been reported to cause root rot of ***Camellia sinensis*** (L.) O. Kuntze (Venkataramani, 1952; Venkataramani and Venkata Ram, I961), ***Myristica fragrans*** Houtt. (Rahman *et al.*, 1981) and leaf spot of ***Wisteria sinensis*** (Sims) Sw. (Reddy, 1975) in South India. ***C. clavatum*** causes root disease of ***E. saligna*** Sm., ***Araucaria angustifolia*** (Bert.) O.Kuntze and several species of ***Pinus*** in Brazil (Hodges and May, 1972), and has been reported as one of the several fungi causing disease of eucalypts in Kerala (Anon., 1984). Earlier, seedling blight by ***C. scoparium*** has been reported to cause large-scale mortality of eucalypts in Argentina, Japan and Java (Spaulding, 1961) and by ***Fusarium oxysporum*** Schl. in USA (Hepting, 1971).

5. **CONIELLA SEEDLING** BLIGHT

Occurrence

The disease was observed to affect 1- to 2-month-old seedlings of ***E. tereticornis*** at Perikuth (Kothamangalam Div.) and Kamaramkudy (Punalur Div.). At perikuth the disease was quite severe as it killed more than 25 per cent of the seedlings.

Symptoms

Initially the infection appeared on the leaf tips in the form of browning, which gradually extended and covered the entire leaf. Numerous black dot like fructifications developed on the necrotic area. From the leaf the infection spread to the

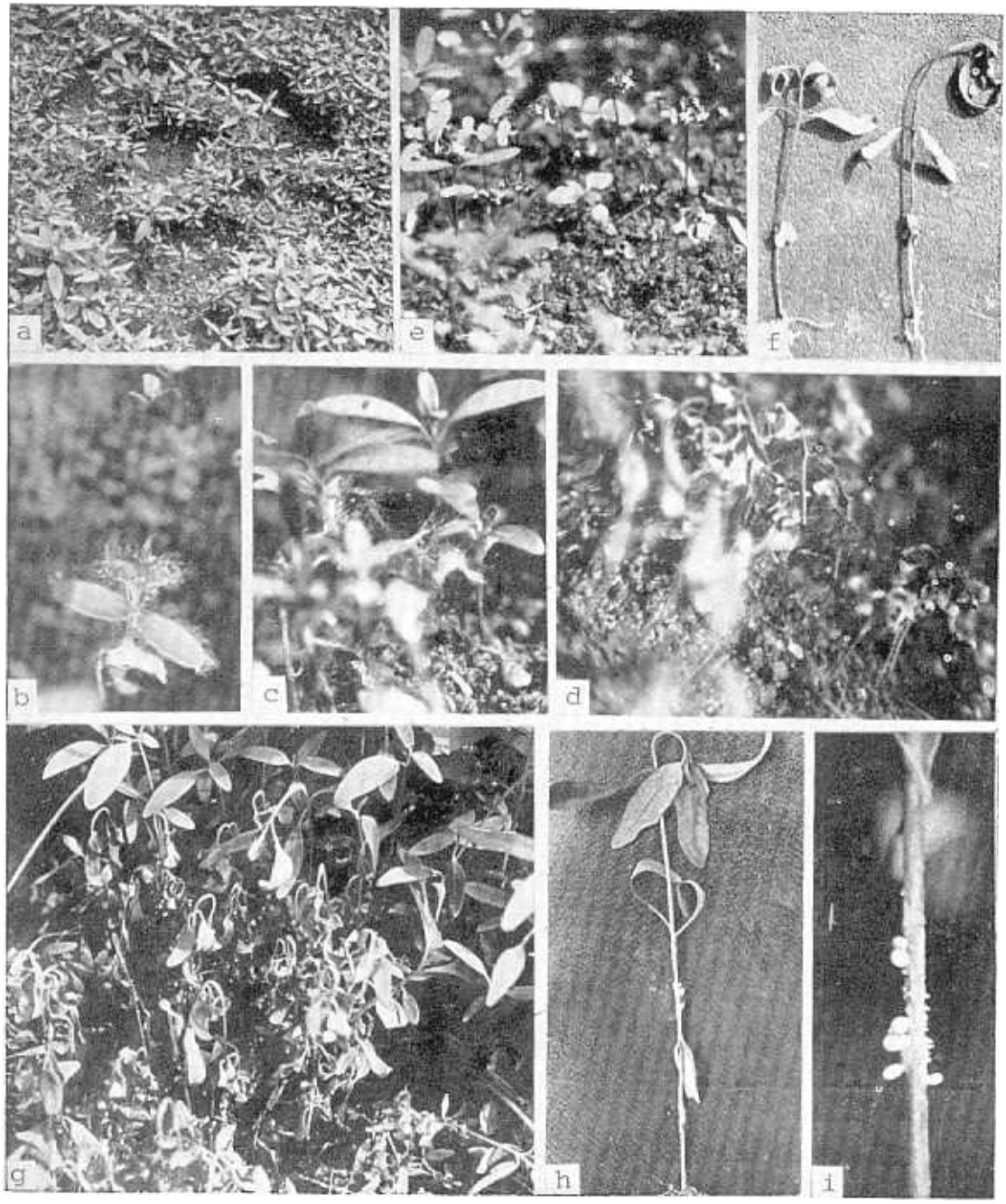


Fig. 30. Seedling diseases of *Eucalyptus*. a, Empty patches in a seedbed of *E. tereticornis* (30-day-old) due to damping-off caused by *Rhizoctonia, Pythium* and *Cylindrocladium;* b, c and d, Different stages of development of web blight of *E. grandis* caused by *Rhizoctonia solani*; e, 35-day-old seedlings of *E. tereticornis* in a seedbed affected with seedling blight caused by *Cylindrocladium*; f, Seedlings of *E. tereticornis* showing characteristic symptoms of seedling blight. Note that roots of the affected seedlings remain healthy; g, A patch of 60-day-old seedlings of *E. grandis* affected with seedling wilt caused by *Sclerotium rolfsii* ; h, A wilted seedling of *E. grandis* showing sclerotia on the stem; i, A magnified view of the affected part of stem to show sclerotia in different stages of growth and mycelium.

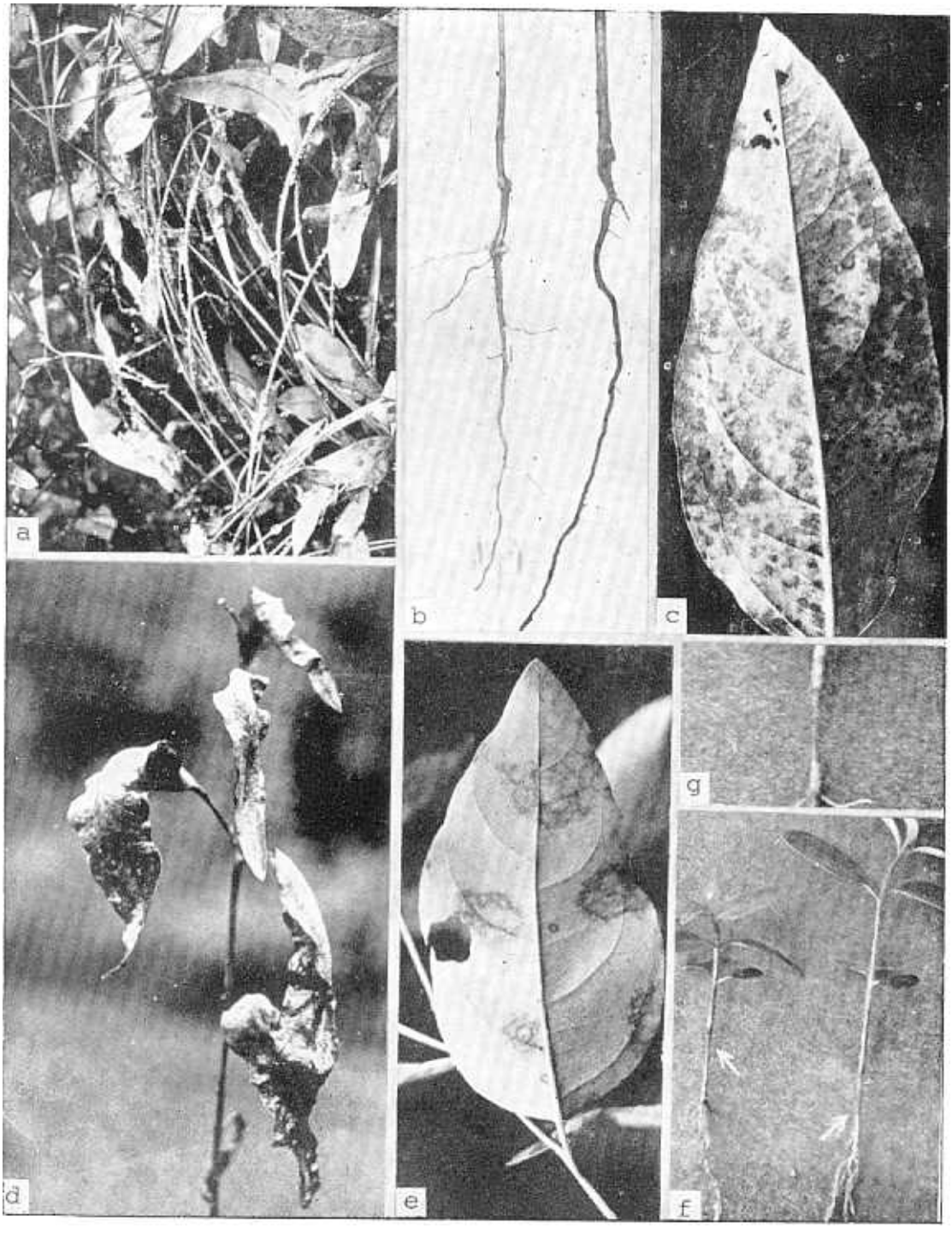


Fig. 31. Seedling diseases of *Eucalyptus*. a, 85-day-old seedlings of *E. tereticornis* in motherbed affected with *S. rolfsii*. Note abundant sclerotia on stem; b, 4-month-old seedlings showing root rot (right). A seedling on the left is healthy. Note fewer feeder roots on the dark coloured diseased root; c, Characteristic small greyish-black spots caused by *Cylindrocladium* on a leaf of *E. tereticornis*; d, A seedling of *E. tereticornis* showing leaf blight due to severe foliage infection of *Cylindrocladium*; e, A leaf of *E. grandis* with large water-soaked lesions as initial foliage symptom of *Cylindrocladium* infection during periods of high humidity; f and g, 2-month-old seedlings affected with collar rot caused by *Rhizoctonia solani*.

entire stem, thus killing the seedlings. Later, on the stem also abundant fructifications developed. Occasionally the infection was observed only on the stem.

Etiology

Coniella granati (Sacc.) Petrak & Syd. (IMI 280238).

Discussion

This is the first report of ***C. granati*** from India and also of its association with seedling blight of ***Eucalyptus.*** Elsewhere, ***C. granati*** is known to cause fruit rot of pomegranate (Hebert and Clayton, 1963).

6. **SEEDLING** WILT

Occurrence

The seedling wilt was recorded in 45- to 60-day-old seedlings of ***E. grandis*** and *E. **tereticornis*** at Chandanathode (Wynad Div.) and Neri amangalam (Kothamangalam Div.). The disease, occurring in patches (Fig. 30g), continues to affect fresh seedlings for 5-6 weeks, then gradually it stops with the ageing of seedlings. At this stage only the weft of mycelium may be seen on the lower portion of the stem.

Symptoms

The first sign of the disease was the formation of a white weft of mycelium at the base of stem, spreading up to the leaves which may entangle other nearby seedlings also. The general (physiological) wilting of seedlings was accompanied by the development of round, off-white sclerotia on the affected leaves and stem (Figs. 30h, i and 31 a). The sclerotia became light brown with age. Wilted seedlings turned brown and died. Usually the infection, which caused decay of the stem and consequent wilting of plants, was localised on stem and leaves; roots remained unaffected.

Etiology

Sclerotium rolfsii Sacc. state of ***Corticium rolfsii*** Curzi (IMI 269010, 160693).

Discussion

S. rolfsii is a common soil-borne pathogen and is known to parasitize seedlings of various tree species (Browne, 1968). This is the first report of ***S. rolfsii*** infecting eucalypt seedlings The seedling wilt disease was noticed only in two nurseries where seedbeds were treated separately with Bavistin (methyl-IH-benzimidazole-2 yl-carbamate) and Benlate (methyl - 1-(butylcarbamoyl) 2-benzimidazole carbamate) for controlling ***Cylindrocladium*** infection. Under these circumstances the development of this disease cannot be regarded to be a primary infection by ***S. rolfsii.*** A similar instance was recorded earlier when severe infection

of seedling blight caused by ***Cylindrocladium*** was the result of a treatment with methyl bromide to control the damping-off pathogens, ***Pythium*** and ***Rhizoctonia*** of ***Eucalyptus.*** This phenomenon where a fungicide applied to control a particular pathogen results in dominance. of some other pathogen and thus causes severe infection of plants has been reported also by Cockerill (1955)

7. · ROOT-ROT

Occurrence

This disease was recorded in seedlings (2- to 5-month-old) of ***E. grandis*** and ***E. tereticornis*** at Chandanathode (Wynad Div.), Peechi (KFRI Campus) and Uppukuzhi (Kothamangalam Div.), especially in those which remained in motherbeds for 6 to 7 months.

Symptoms

Root-rot caused slow wilting of seedlings in scattered patches, The first symptom was the change of pigmentation in apical leaves from normal green to light purple. Within a week, this change in pigmentation moved downwards rapidly and by the time all the leaves were affected, the apical portion of seedlings showed wilting resulting in death of the plants. The root system of such plants was found to be completely damaged due to rotting. The colour of the affected roots was dark brown instead of off-white (Fig. 31 b), or pale yellowish brown, the natural colour of roots. Generally, the infection was noticed starting from the feeder roots and later proceeding to the main root system. In some of the root rot specimens even the stem was found to be affected causing decay of the root collar zone.

In the case of ***Cylindrocladium*** root-rot, usuaily 2-month-old seedlings were affected. The infection caused discoloutation of roots, which often extended to the stem. The root system of the diseased seedlings got decayed completely.

Etiology

i. ***Cylindrocladium*** **curvatum** Boedijn & Reitsma
ii. ***Sclerotium rolfsii***
iii. ***Rhizoctonia solani***

Control measures

Root-rot is effectively controlled by drenching the affected seedbeds with Emisan-6 (0.005% a. i.) for ***S. rolfsii*** and ***R. solani*** or Bavistin (0.02% a. i.) for ***C. curvatum*** (Anon., 1984). The treatment may be repeated after a week if the disease persists.

Discussion

Root rot caused by ***C. curvatum,*** a new disease report, was not common as it was recorded only at Peechi during 1979-1980.

E. grandis, which was susceptible to seedling wilt caused by ***S. rolfsii*** at a younger stage, became affected with root-rot only when seedlings were 4- to 5-month: old, The cause of this delayed attack on roots was not clearly understood. The root-rots of ***Eucalyptus*** caused by ***S. rolfsii*** and ***R. solani*** are new disease reports.

Diseases of container seedlings

After the seedlings are transplanted into polythene containers, ***Cylindrocladium*** spp. pose the main threat to survival, especially after the pre-monsoon showers. These species cause various diseases such as stern infection, and leaf and shoot blights, They are often followed by the Phaeoseptoria leaf spot, which may cause severe leaf spotting resulting in premature defoliation.

8. CYLINDROCLADIUM LEAF SPOT

Occurrence

Leaf infection caused by ***Cylindrocladium*** spp. is one of the important and widespread diseases in eucalypt nurseries in Kerala. Usually the disease has been recorded to appear after the onset of the monsoon. Since under such conditions the pathogen produces abundant conidia on the affected leaves, which are disseminated by splashing of rain drops, the disease spreads very rapidly. At high elevations the disease appears even before the monsoon. Seedlings of both ***E. grandis*** and ***E. tereticornis*** have been found to be highly susceptible to Cylindrocladium leaf spot.

Symptoms

Leaf spots caused by ***Cylindrocladium*** appear first as minute, greyish-black water-soaked lesions on young as well as older leaves (Fig. 31, c). Later, smaller lesions coalesce to form large necrotic areas which on drying turn, brown giving typical blighted appearance (Fig. 31 d). Under high humidity, the initial symptoms are generally large greyish-black spots (Fig. 31 e), sometimes covering the entire leaf; abundant conidia may also be observed on the affected areas. Severe leaf infection causes leaf blight resulting in premature defoliation, which weakens the seedlings.

Etiology

i. ***Cylindrocladium quinqueseptatum***
ii. ***C. ilicicola***
iii. ***C. clavatum***
iv. ***C. camelliae***

Control measures

Bavistin 0.01% (a. i.) applied as a foliar drench is found to be highly effective in controlling leaf spot caused by ***Cylindrocladium.*** In case of severe infection a second treatment may be essential after a week.

Discussion

Leaf spot caused by ***C. quinqueseptatum*** has been reported by Bakshi (1975) and Sehgal *et al.* (1978) but ***C. ilicicola*** and ***C. clavatum*** are new records for ***E. grandis*** and ***E. tereticornis*** in India. Earlier, ***C. ilicicola*** has been reported from India on ***E. globulus*** by Reddy (1973). ***C. clavatum*** was first isolated from E. ***tereticornis*** nurseries at Kottappara, where it caused severe leaf and shoot blights. Later, it was recorded also from other nurseries at Pattikad, KFRI Campus, Mullaringad, Oonnukal (Kothamangalam), and Pezhad (Kottappara). During the monsoon of 1981, within a period of 15 to 20 days, a total of about 120,000 basketed seedlings were reported to have been killed due to leaf and shoot blights in the above nurseries. Although the mortality was due to the dual infection of ***C. clavatum*** and ***C.quinqueseptatum,*** the former was the dominant species which also appeared to be more aggressive.

Leaf infection of eucalypts caused by ***Cylindrocladium*** spp. is widespread in Brazil and curiously is not prevalent in plantations established from seed source of Australian origin. ***C. clavatum,*** the main cause of root–rot of eucalypts in Brazil, is also reported to be causing leaf infection of ***E. saligna*** (Hodges and May, 1972).

9. SEEDLING STEM INFECTION

A. Cylindrocladium stem infection

Qccurrcnce

Seedling stem infection of ***E. grandis*** and ***E. tereticornis,*** which may result in a canker, was frequently observed in 2- to 3-month-old seedlings. The disease was more prevalent in ***E. grandis*** in high ranges.

Symptoms

Initially the infection developed on the lower half of the stem (Fig, 32 a), and later it spread to upper parts as well (Fig. 32 b). The affected seedlings which primarily showed typical symptoms of physiological wilting, such as flaccidity of leaves and the apical shoot, were eventually killed. Under high humid-warm conditions abundant conidia of ***Cylindrocladium*** were observed on the infected stern (Fig. 32 c). Development of epicormic roots and shoots from the callus was frequently observed during continuous rainy periods.

Etiology

i. ***Cylindrocladium quinqueseptatum***
ii. ***C. iliciola***
iii. ***C. clavatum***

B. Rhizoctonia collar rot

Occurrence

This is not a common disease but it develops under high humidity and air stagnation due to crowding of seedlings. The disease was recorded in 2- to 3-month-old seedlings of ***E. grandis*** and ***E. tereticornis*** at Chandanathode (Wynad Div.) and of the latter species at Uppukuzhi (Kothamangalam Div.)

Symptoms

The infection appeared just near the ground level in the form of greyish water-soaked lesions on the stem of seedlings (Figs. 31 f, g). Stem discolouration was soon followed by splitting of the outer bark, stem girdling and callus formation. The affected seedlings, which showed typical symptoms of physiological wilting, failed to survive.

Etiology

Rhizoctonia solani

10. SHOOT BLIGHT

Occurrence

This is a common disease in seedlings of ***E. grandis*** and ***E. tereticornis*** after onset of the monsoon. The disease was more prevalent in high rainfall areas than in low rainfall. Shoot blight has been observed to cause over 50 per cent mortality in container seedlings.

Symptoms

Shoot blight of eucalypt seedlings was caused due to multiple infection by ***Cylindrocladium*** affecting the apical bud, stem (causing stem canker) and leaves. Generally, more than one species of ***Cylindrocladium*** were associated with the disease. Affected seedlings, which became leafless, were killed outright.

Etiology

i. ***Cylindrocladium quinqueseptatum***
ii. ***C. ilicicola***
iii. ***C. clavatum***

Control measures

Bavistin 0.01% (a. i.) applied as a foliar drench, is found to be highly effective in controlling leaf spot caused by ***Cylindrocladium*** (Anon., 1984). In case of severe infection a second treatment may be essential after a week.

Discussion

Unlike in Kerala, ***C. quinqueseptatum*** and ***C. ilicicola*** have been reported from Brazil as the cause of die-back of young plants (Figueiredo and Cruz, 1963; Figueiredo and Namekata, 1967), whereas ***C. clavatum*** causes lethal root disease of ***E. saligna. C. scoparium,*** which is probably the most important pathogen associated with die-back and defoliation of eucalypts in Brazil, is of minor significance in Kerala as it causes only leaf spot.

11. PHAEOSEPTORIA LEAF SPOT

Occurrence

The disease was prevalent in seedlings of ***E. tereticornis*** and ***E. grandis*** during the months of March-May. Severe infection caused premature defoliation of mature leaves thus affecting the growth of seedlings.

Symptoms

The infection appeared initially on the mature leaves (Fig. 32 d) as purple to brownish-purple amphigenous angular spots. The leaf spots gradually progressed upwards affecting even the youngest leaves (Fig. 32 e). In dry weather, when the spots turned necrotic, minute black pycnidia developed embedded in the leaf tissue. These pycnidia produced abundant conidia, usually in tendrils, which appeared as brownish-black woolly masses on both the leaf surfaces.

Etiology

Phaeoseptoria eucalypti (Hansf.) Walker

Discussion

The disease is discussed in detail along with the control measures under plantation diseases.

Possible control measures

It is clear from the above description that as nursery diseases in eucalypts are caused by several pathogenic fungi, it is not possible to control them by one fungicide or a specific combined application of different fungicides. Though control measure9 for some of the important diseases have been given at appropriate places, experience gained during this survey and chemical control trials conducted during 1980-I982 (Sharma and Mohanan, 1981 b) indicate that the nursery disease complex affecting ***Eucalyptus*** seedlings can be controlled effectively by an integrated approach i.e., proper management of the nursery together with some prophylactic application of fungicides.

Proper management of the nursery includes adequate shade with dispersed light, preferably with coir mat, medium density of seedlings in the bed (about 200 per 30 X30 cm area) and the right quantity of water per bed (30 - 40 litres at a time per standard bed, 12 X 1 . 2 m). The frequency of watering should range from 2 to 4 times a day depending on climaticconditions and growth stage of seedlings. These measures, if properly followed, will prevent the appearance of the disease to a considerable extent and also check the development of the disease into serious epidemic proportions. Prophylactic application of effective fungicides at the proper time will control the development of these diseases effectively. The details regarding schedule of fungicidal treatments and various nursery practices have been described in KFRI Information Bulletin No.6 (Anon., 1984).

PLANTATION DISEASES

A large number of pathogens 'were recorded causing leaf and stem diseases while the root diseases were only a few. Most of the serious diseases appeared within first three years of outplanting. A few were short lived such as Botryodiplodia stern canker while others continued to affect trees till the end of the rotation affecting the growth considerably and consequently yield. Heart rot, which usually develops at the<age of eight to ten years of growth was not recorded during the survey.

1. CYLINDROCLADIUM ROOT ROT

Occurrence

The disease was observed in a 9-month–old plantation of ***E. tereticornis*** at cheenkannipalli (Kozhikode Div.) during" January 1980. About 10 per cent of the plants were found to be affected with the diseases.

Symptoms

The leaves of the affected plants became flaccid and apical shoot showed drooping. The leaves turned brown and dried up. The root system of such plants was found to be completely rotted with pronounced brown discolouration (Fig. 33a), The affected plants failed to survive. On dead roots abundant mycelium and sporulation of the causal fungus could be ,observed.

Etiology

Calonectria floridana Sobers and its anamorph ***Cylindrocladium floridanum*** Sobers.and Seymour (IMI 250220,276603).

Discussion

Cylindrocladium floridanurn is known to cause mild leaf spotting in seedlings of ***Eucalyptus*** spp. in California, U. S.A. (Sobers and Seymour, 1967). This is the first

report of ***Calonectria floridana*** and its anamorph ***Cylindrocladium floridanum*** causing root rot of ***Eucalyptus.*** Unlike other ***Cylindrocladium*** species which cause foliar diseases during the wet season, occurrence of root rot caused by ***C. floridanurn*** during the dry period is quite interesting. If the roots of the affected plants are not properly examined this disease may be mistaken either for termite damage or Botryodiplodia stem canker. Considering the rare occurrence and low incidence of this disease it does not appear to be a serious problem.

2. CYLINDROCARPON ROOT ROT

Occurrence

The disease was observed in a 5-year-old plantation of ***E. grandis*** at Thrissillery and in a 1- to-2-year-old plantation of ***E. tereticornis*** at Thalakode, Neriamangalam (Kothamangalam Div.). The incidence of the disease at Thrissillery was <1 per cent whereas at Thalakode it was up to 20 per cent in certain areas of the plantation. This high incidence in the latter plantation was recorded during the dry period (December-April).

Symptoms

In ***E. grandis*** the initial symptom was wilting and drying up of leaves. The roots of the affected trees became discoloured and showed rotting. The infection generally spread upto the collar area and produced a canker. The affected trees died within 1-2 months. In ***E. tereticornis*** the leaves of the affected plants, especially the bottom ones, turned reddish purple in colour and dried up; no wilting of leaves was observed. The plants were killed within 2-3 weeks after the change in colour of the foliage was noticed. The roots of diseased trees showed browning and rotting (Fig. 33 b).

Etiology

Cylindrocarpon *lucidum* Booth (IMI 267023).

Discussion

Eucalyptus is a new host record for ***C. lucidum.*** It has earlier been reported on ***Acacia*** from Nilgiris (Venkataramani, 1951), and on ***Camellia*** sp. from Malayasia (Booth, 1966). C. ***lucidum*** is considered to be a weak pathogen as usually the entery in the host is through injury or wounds. However, in affected plants of ***Eucalyptus*** no injuries were observed which could serve as entry points for the pathogen. In a young ***E. tereticornis*** plantation, the disease appeared to be a potentially serious one as it killed upto ca. 20 per cent plants, indicating the susceptible nature of the host. The low incidence in ***E. grandis*** could be due to the age of the plants and/or the resistant nature of this species to the pathogen.

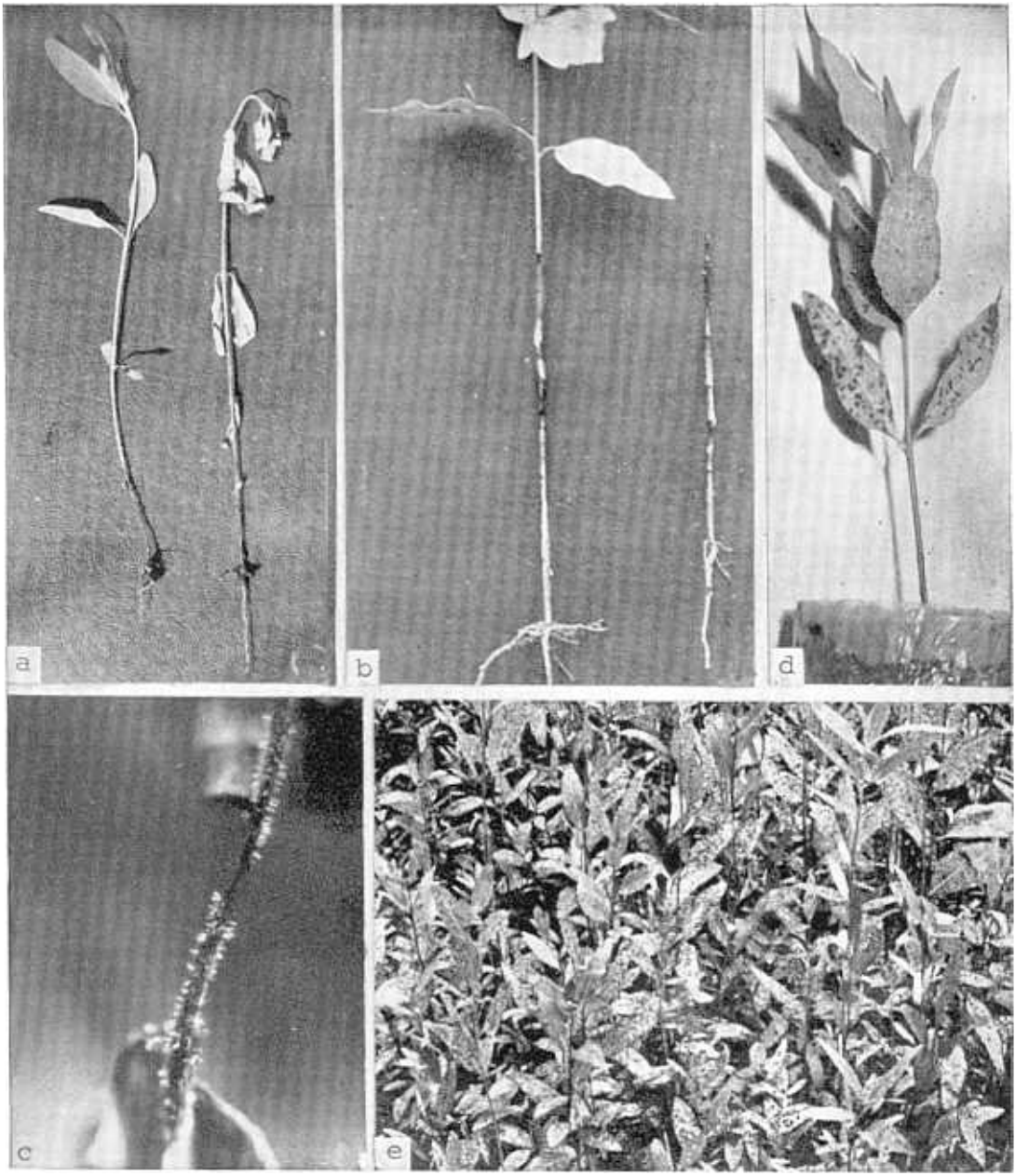


Fig. 32 Seedling diseases of *Eucalyptus*. a, 45-day-old seedlings of *E. tereticornis* showing wilting due to stem infection caused by *Cylindrocladium*; b, Stem infection of 90-day-old seedlings of *E. grandis* which results into a canker; c, Production of conidia of *Cylindrocladium* on the affected part of the stem which appears as white powdery mass; d, A 90-day-old container seedling of *E. tereticornis* infected with *Phaeoseptoria eucalypti*. Note severe infection in basal pair of leaves. The first pair of basal leaves had already defoliated prematurely; e, Seedlings of *E. tereticornis* in a motherbed showing severe foliage infection by *Phaeoseptoria*.

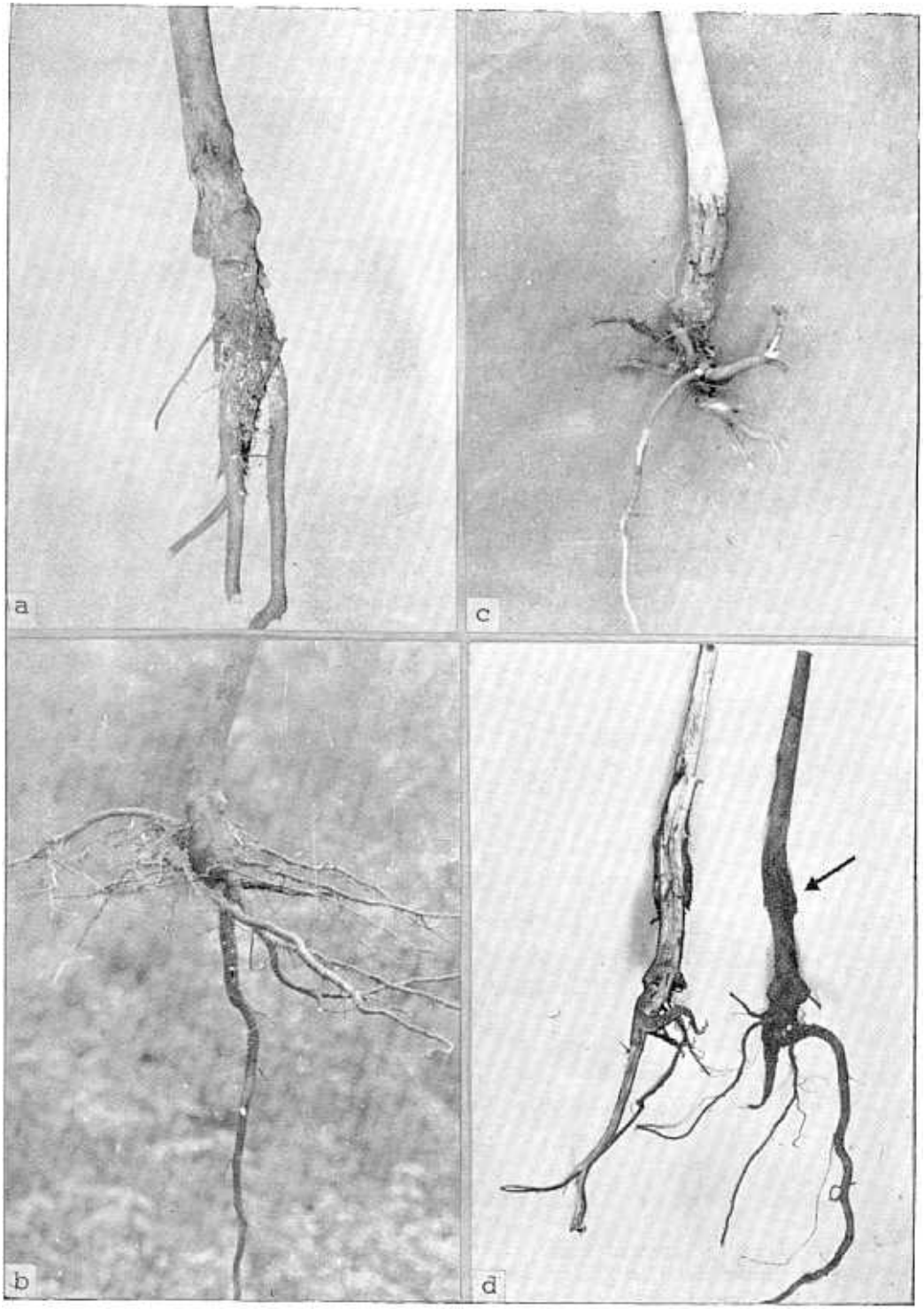


Fig. 33. Plantation diseases of *Eucalyptus*. a, Root rot of 9-month-old sapling of *E. tereticornis* caused by *Cylindrocladium floridanum*. Note abundant colonization by the pathogen on the root; b, Root rot of 1-year-old sapling caused by *Cylindrocarpon lucidum*; c and d, Stem (root collar) canker of *E. tereticornis* caused by *Botryodiplodia theobromae*. Root collar region of a partially wilted plant split open to show the damage to the tissue (d). Note typical swelling just above the canker (constricted area) and discolouration in the roots.

3. WILT

Occurrence

Wilt disease of ***E. grandis*** was recorded during the monsoon (August/September) in a 2-year-old plantation at Kaikutty, Nelliampathy (Nenmara Div.) and in 1- and 2-year-old plantations at Attappara, (Ranni Div.) and Pamba (Kerala For, Dev. Corp.), The incidence of the disease ranged between 10 and 25 per cent in various: plantations at Pamba, while in Nelliampathy, where ***E, grandis*** was grown as a roadside strip plantation (0.4 ha), ca. 35 per cent of the plants died.

symptoms

The affected plants showed characteristic symptoms of wilting. Initially the lower foliage became flaccid and dried up. Slowly and gradually the wilting proceeded upwards eventually killing the growing shoot, The wilted plants could be easily located in the plantations because of their dried up leaves. The vascular tissues of roots and stem of the diseased plants showed typical browning, characteristic of vascular wilt.

Etiology

F usarium oxysporum Schlecht.

Pathogenicity

Pathogenicity of the isolate was tested on 9-month-old seedlings of ***E. grandis*** raised in pasteurized soil. Ten seedlings were pulled out gently and the root system washed in sterile distilled water. Inoculation of roots was done by pricking and dipping them in a suspension of ***E. oxysporum,*** containing abundant conidia and mycelial fragments, for 5 minutes and transplanting seedlings back into the container. In another set of ten seedlings, 10 ml of suspension was poured over the container soil. Equal number of control seedlings were also maintained for both the set-ups: The seedlings were watered with equal quantity of water and observations on the appearance of disease symptoms recorded daily.

In root treatment, wilting was observed only in four seedlings after 10 days of incubation whereas in soil treated seedlings the success of infection was only 30 per cent and it toot more than a month. The roots of the affected seedlings showed characteristic browning in the vessel elements.

Discussion

Fusarium oxysporum, which has a worldwide distribution, is a soil-borne facultative parasite or many plants. It is disseminated through plant material and soil on implements, transplants, surface drainage, water and wind-borne spores

(Booth, 1971). Infection is mainly through vascular wounds and not via uninjured stems or roots or through the callus of healed wounds (Mc Clure, 1949, 1950).

The pathogenicity trials confirmed ***E. oxysporum*** as a wilt pathogen. The roots and stem of all the wilted seedlings consistently showed browning and abundant mycelium in the vessel elements.

4. BOTRYODIPLODIA STEM (ROOT COLLAR) CANKER

Occurrence and incidence

Mortality of 16-month-old ***Eucalyptus tereticornis*** in a plantation at Kottappara was brought to our notice by the Forest Department in November 1979, About 10 per cent of the plants in various compartments of a 1978 plantation were either dead or wilted. The disease was found at Noolpuzha and Thariode (Kozhikode Div.) and at Periya (Wynad Div.). Later, it was also recorded at Varavoor, Potta, Trichur (Kerala Forest Development Corp.), Kakkavayal (Kozhikode Div.) and Mullaringad (Kothamangalam Div.); in ***E. grandis,*** it was observed at Noolpuzha, Thariode and Periya.

As the fresh infection was mostly recorded during the hot, dry months of November-April (Figs. 3a, b), a survey of the incidence of the disease was conducted in young ***E. tereticornis*** and ***E. grandis*** plantations only during this period between 1979 and 1982. The assessment was made by counting the diseased plants in three] five plots of 100 plants selected at random in a plantation.

The disease was quite common in young plantations (Table 32). Of the ten plantations surveyed collar rot was not recorded only in two where no taungya was grown. The distribution of the disease did not show any pattern; it was observed either in patches or scattered at random all over the plot. There appeared to be no indication of plant-to-plant spread of the disease.

symptoms

Affected plants showed typical symptoms of physiological wilting i.e., droopy apical shoots with flaccid leaves. Within a day or two the wilted plants died. In all the diseased plants the root collar region was typically constrioted and compressed with irregular crevices (Fig. 33 c), where occasionally conidiomata of the pathogen were observed. Often the stem just above the canker was abnormally swollen. This seedling was the result of callus growth beneath the bark associated with the infection. Occasionally the splitting and rupturing of the outer bark at the canker resulted in girdling of the stem, and plants died.. The tissue in the canker region was brown and dead or dying; Sometimes the browning also extended to roots, causing root decay (Fig. 33 d).

Table 32. **Incidence of stem canker disease caused by *B. theobromae* in various plantations of *Eucalyptus* in Kerala during 1979-1982**

Eucalyptus species	Locality	Year of planting	Age of plants at survey (months)	No. of plots surveyed	Mean incidence of disease (%)
E. tereticornis	Kottappara	1978	16	3	10
	Varavoor	1978	18	5	5
	Elnad	1978	10	3	0
	Potta	1979	9	3	1
	Kakkavayal	1979	10	5	20
	Mullaringad	1981	11	5	12
E. grandis	Thariode	1979	9	3	10
	Noolpuzha	1979	9	3	1
	Mavinhalla	1980	9	5	0
	Periya	1980	10	3	2

Etiology

Botryodiplodia theobromae Pat. (Syn. ***Lasiodiplodia theobromae*** (Pat.) Griff & Maubla. (IMI 246479).

Pathogenicity

The pathogenicity of the isolate was tested during November 1981 using 12-month-old container plants of ***E. tereticornis*** and ***E. grandis.*** Plants were either wounded with a sterile scalpel near the root collar or unwounded, and inoculated with a 7 mm disc cut out from a culture of ***B. theobromae*** or uninoculated (control). Ten plants of each species with each combination of wounding and inoculation were used, the site of wounding/inoculation being covered with sterile moist absorbent cotton and tied with twine. Plants were observed for 75 days. After 60 days all plants of both species which had been wounded and inoculated had a typical canker. ***B. theobromae*** was re-isolated from the cankers, indicating the pathogenic nature of the isolate. ***E. tereticornis*** was damaged more than ***E. grandis,*** since the extent of canker was more in the former (mean length 3.8 cm) than in the latter (mean 2.5 cm). Most of the ***E. tereticornis*** plants showed dead tissue around the root collar region and wilted 75 days after inoculation and wounding. Only two unwounded inoculated saplings of ***E. tereticornis*** developed small cankers 1-2 cm long, and none of the ***E. grandis*** plantsshowed any symptom. All uninoculated saplings remained healthy.

Growth studies

Vegetative growth of ***B. theobromae*** was studied on potato dextrose agar (PDA), lima bean agar (LBA), malt agar and Czapek's dox agar (CDA) at $15 \pm 2°C$, $28 \pm 2°C$ and $38 \pm 2°C$. A disc, 4.5 mm diam, cut from the margin of the actively growing colony of the pathogen was transferred to the centre of the petri dish containing 15 ml of medium. There were three replications for each culture medium-temperature combination. Five observations on radial growth were recorded daily from each petri dish. At each observation the petri dishes were transferred back immediately to their respective temperatures.

Best growth was on PDA at 28^oC some growth occurred at 38^oC, but none at 15^oC (Table 33).

Evaluation of fungicides

Following poison-bait technique, 15 fungicides namely, Bavistin, Benlate, Calixin, Captan, Daconil, Difolatan, Demosan, Dithane M-45, Fytolan, Hexathir (Thiride), Kitazin, Polyram Combi, Saprol, Syllit and Tecto were evaluated to test their efficacy at 0.05, 0.1, 0.25 and 0.5% (a. i.) against ***B. theobromae.***

Total inhibition of growth was recorded at all concentrations of Bavistin and Tecto (Table 34). Though Benlate was also quite effective, some growth occurred at 0.05% on the 10th day of incubation. Another promising fungicide, Hexathir, also inhibited the growth completely but could not prevent the growth of the mycelium on the inoculated disc. Although there was no growth till the sixth day in Fytolan and Calixin, traces of growth was recorded on the tenth day. An interesting observation of a clear zone around the inoculated disc was recorded in all the petri dishes containing Calixin. The diameter of this clear zone increased with the increasing concentrations of Calixin.

Discussion

The stem canker disease caused by B. ***theobromae*** on ***E. tereticornis*** and ***E. grandis*** has been recorded for the first time in India. The survey indicates that it can pose a serious threat in the establishment of young plantations causing as much as 20 per cent mortality recorded at Kakkavayal (Thamarassery). Furthermore, the prevalence of this disease mostly in ***E. tereticornis*** plantations suggests that this species is more susceptible than ***E. grandis.*** This is also supported by the observations of pathogenicity trials.

B. theobromae is a weak parasite, and usually infects the host through wounds (Punithalingam, 1980). Termites are known to cause problems in the establishment of young eucalypt plantations, especially ***E. tereticornis,*** in Kerala (Nair and Varma,

Table 33. Growth (mm) of *B. theobromae* on different media incubated at 15, 28 and 38°C (Mean of 3 replications; SD and SE are given in bracket)

Days of incubation	Growth medium											
	Potato dextrose agar			Malt agar			Lima bean agar			Czapeck's dox agar		
	15*	28	38	15	28	38	15	as	38	15	28	38
1	0	2.09 (0.162+ 0.46)	2.44 (0.124± 0. 035)	0	1.925 (0.186± 0.053)	2.175 (O.322+ 0.093)	0	1.09 (0.0794± 0 22)	1.058 (0.079± 0.022)	0	1.016 (0.102+ 0.029)	2.475 (0.195± 0.056)
2	0	7.51 (0.157± 0.057)	4.20 (0.380± 0.107)	0	5.04 (0 .249+ **0.061)**	5.47 (0.2494± 0.071)	0	5.73 (0.344± 0.099)	5.09 (0.247+ **0.079)-**	0	7.10 (0.223+ 0. 064)	5.03 (0.369+ 0.106)-
3	0	9.00 *9.00*	7.47 (0. 293+ 0.084)	0	7.91 (0.284± 0.082)	7.49 (0.226 ± 0.065)	0	5.70 (0.333± 0 096)	7.47 (0.182± 0.052)	0	7.10 (0.219+ 0. 063)	7.65 (0.393± 0.113)

* Temperature °C

Table 34. Per cent growth inhibition over control of *B. theobromae* at four concentrations of different fungicides (mean of 15 observations from three replicates each and SE)

Fungicide	Period of incubation (in days)	Per cent concentration (a. i.)			
		0.05	0.1	0.25	0.5
1. Bavistin	4	100.00	100.00	100.00	100.00
	6	100.00	100.00	100.00	100.00
	10	100.00	100.00	100.00	100.00
2. Benlate	4	100.00	100.00	100.00	100.00
	6	100.00	100.00	100.00	100.00
	10	100.00	100.00	100.00	100.00
3. Calixin	4	100.00	100.00	100.00	100.00
	6	100.00	100.00	100.00	100.00
	10	95.21±0.07	98.54±0.13	100.00	100.00
4. DaconiI	4	100.00	100.00	100.00	100.00
	6	58.70±1.26	64.13±1.13	76.18±1.92	91.71±1.39
	10	36.26±1.27	90.57±1.66	42.55±2.19	63.69±3.41
5. Difolatan	4	100.00	100.00	100.00	100.00
	6	100.00	100.00	100.00	100.00
	10	91.46±0.39	90.33±0.45	90.81±1.36	93.41±0 72
6. Demosan	4	100.00	100.00	100.00	100.00
	6	80.57±0.71	86.91±0.32	98.05±0.23	100.00
	10	53.92±1.17	70.47±0.47	80.72±0.43	89.67±0.35
7. Captan	4	100.00	100.00	100.00	100.00
	6	100.00	100.00	100.00	100.00
	10	83.09±0.48	90.00±0.34	97.23±0.10	100.00
8. Dithane M-45	4	100.00	100.00	100.00	100.00
	6	100.00	100.00	100.00	100.00
	10	76.64±1.18	88.53±0.64	100.09	100.00
9. Fytolan	4	100.00	100.00	100.00	100.00
	6	100.00	100.00	100.00	100.00
	10	100.00(tr)	100.00 (tr)	100.00 (tr)	190.00(tr)

10. Hexathir (Thiride)	4	100.00	100.00	100.00	100.00
	6	100.00	100.00	100.00	100.00
	10	100.00	100.00	100.00	100.00
11. Kitazin	4	100.00	100.00	100.30	100.00
	6	94.96±0.32	96.l0+0.27	98.29± 0.16	100.00
	10	90.48+0 .32	90.20± 0.28	95.45± 0.30	100.00
12. Polyram Combi	4*	41.66±0.631*	75.06±1.10	100.00	100.00
	6*	36.70± 0.423*	45.30±0.75	96 00±0.30	100.00
	10	7.48±0. 16	14.05±0.00	63.00± 0.50	79.35±0.96
13. Saprol	4	100.00	100.00	100 00	100.00
	6	100.30	100.00	100.00	100.00
	10	92.0076±0.37	95.98±0.36	100.00	103.00
14. Syllit	4	100.00	100.00	100.00	100.00
	6	93.99±0.36	96.67±0.33	100.00	100.00
	10	90.08±0.49	90.97±0.47	100.00	100.00
15. Tecto	4	100.00	100.00	100.00	100.00
	6	100.00	100·00	100.00	100.00
	10	100.00	100.00	100.00	100.00

* Mean of 10 observations from two replicates; tr = traces of growth

1981). In some instances root injury caused by termites may have acted as entry points for the pathogen. It was interesting to note that in all the instances where the canker disease was prevalent, a taungya crop was also cultivated. It is likely that soil working for taungya may cause root injury facilitating infection by B. ***theobromae.*** This possibility is strengthened by the results obtained from the pathogenicity trials, which indicate that ***B. theobromae*** is a weak wound pathogen.

Canker disease occurs during the warm and dry weather in Kerala when the day temperature is high (35-38°C) and night temperature low (22-25°C). Large fluctuations in soil temperature may have caused some injury to the saplings near the ground level, through which infection may have occurred. This possibility is supported by earlier observations on infection by ***B. theobromae.*** Sun-scald and lack of moisture favour collar rot of rubber ***(Hevea brasiliensis*** Mull. Ag.) (Petch, 1921), and heat injury favours collar rot of groundnut ***(Arachis hypogaea*** L.) (McGuire and Cooper, 1965).

Growth of ***B. theobromae*** at high temperature indicates that the disease is favoured by warm weather as observed in the field. Though the pathogen grew even at 38°C the best growth was supported on PDA at 28 ± 2°C. Recently, Krupinsky (1982) has also shown that growth of ***B. hypodermia*** (Sacc.) Petr. and Syd., a canker pathogen of Siberian elm ***(Ulmus pumila*** L.), was best on PDA (Difco) at 25 ± 1°C among other media (PDA Difco), "homemade" PDA, yeast malt extract agar, V-8 juice agar, tested at 16,21, 22,25,26,27, 29, 31°C.

Of the 15 fungicides evaluated against ***B. theobromae,*** Bavistin and Tecto were most effective. Although, chemical control of a disease of this nature in forest plantations is not an economically feasible solution, a soil drench of either of these fungicides to plants with accidental injury may be useful in controlling the disease. Since the canker disease is manifested through wounds, weeding and other soil operations must be carried out carefully.

5. CRYPHONECTRPA STEM CANKER

Occurrence and severity

The disease was first recorded in Wynad Div. during 1980 in a 2-year-old plantation of ***E. grandis*** at Thrissillery and 3-and 5-year-old plantations at Mavinhalla. In the following year Cryphonectria cankers were also observed in 3-year-old ***E. grandis*** and ***E. tereticornis*** plantations in southern Kerala at Mullachal (Kerala Forest Development Corp., Trivandrum). Later the disease was also found in 4-year-old experimental plantations of *E.* ***tereticornis, E. citriodora*** Hook, ***E. torelliana*** F. Muell. at Kottappara and 10-year-old ***E. deglupta*** Bl. at Vazhachal (Vazhachal Div.). The latter species was the most affected. By 1983 the stem cankers were recorded in most of the ***E. grandis*** plantations (>3-year-old) situated in high ranges of Wynad District. However, the disease was not so common in ***E. tereticornis,*** grown at lower elevations; of the four plantations surveyed it was observed only in two, situated at Kottappara and Mullachal.

To study the distribution and incidence of the disease, a survey was conducted in nine plantations of ***E. grandis*** and ***E. tereticornis*** situated in various localities of the State (Fig. 34). Further eucalypt plantations of these and other species were also visited but no systematic survey was made in them. Regular observations were recorded in three to five observation plots of 20 x 20 plants selected at random in each of the representative plantations. However, in a 1977 ***E. grandis*** plantation at Mavinhalla where the disease incidence was high, observations were recorded from all the trees in four plots each of 25 x 25 plants (i.e., 625 trees per plot). This was done to study the spatial and temporal spread of the disease.

Observations on incidence and severity of the disease were recorded during September-October, just after the monsoon from 1980 to 1982. ***E. grandis*** plantations at Mavinhalla were visited on alternate months to study the symptoms and spread of the disease. The number and age of cankers per tree were recorded. Paint was used to mark the first appearance of cankers, so that their age could be recorded.

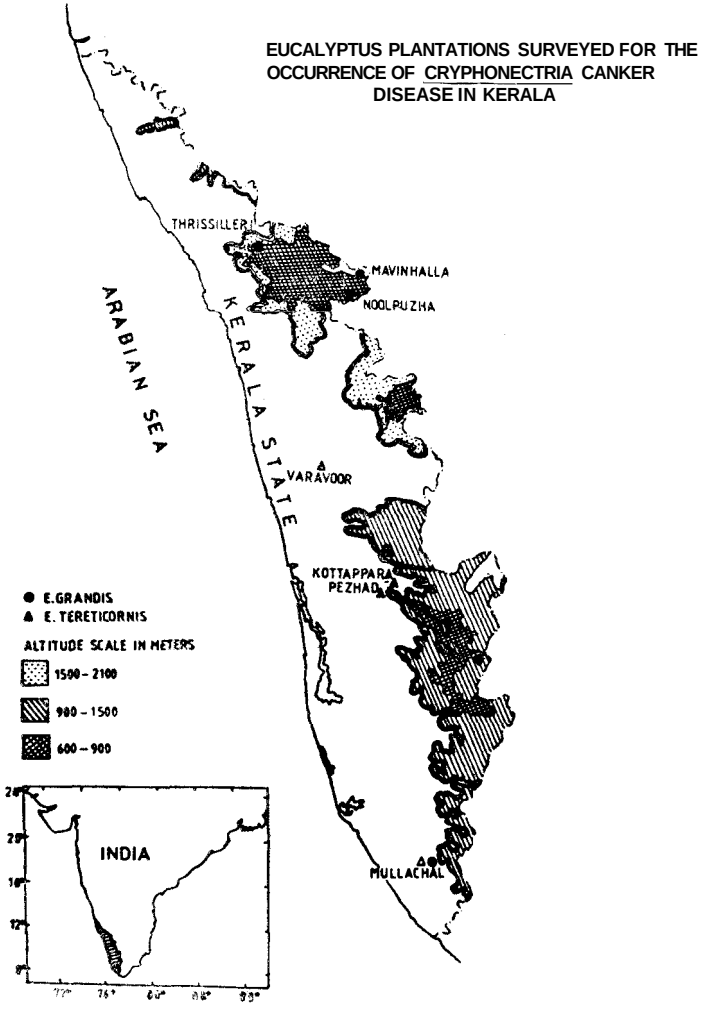


Fig. 34. ***Eucalyptus*** plantations surveyed for the occurrence of Cryphonectria stem canker **(C. *cubensis)*** disease in Kerala.

The data presented in Table 35 indicate that the incidence of the canker disease varied greatly between localities depending upon the species of ***Eucalyptus*** and climatic conditions. During the last four years of observations the disease has progressed rapidly in North at Mavinhalla and Thrissillery and South at Mullachal. Interestingly, plantations in northern areas are in higher elevations (av. 850 m above msl)

with high rainfall (>4500 mm p. a.) whereas those in southern areas are at lower elevations (50-200 m above msl with less rainfall upto 2000 mm p. a.) (Fig. 3). Although plantations in the central part of Kerala are at low elevations (<200 m above msl) they receive a rainfall of upto 3000 mm p. a. It appears from the disease incidence figures that ***E. tereticornis*** is a more resistant species than ***E. grandis.*** Thus, in adjoining plantations of these two species at Mullachal, where the incidence of canker disease was 12 per cent in ***E. grandis,*** it was well below I per cent in *E. **tereticornis***.

Table 35. **Incidence of stem canker caused by *Cryphonectria cubensis* in various eucalypt plantations situated in different parts of Kerala during 1980-1982**

Locality	*Eucalyptus* species & year of planting		Percent incidence of disease		
			1980	1981	1982
Mavinhalla	*E. grandis*	1975	0	0	21.0
	E. grandis	1977	0.80	7.5	27.4
Thrissillery	*E. grandis*	1977	3.0	4.2	-**
Mullachal	*E. grandis*	1978	0	—*	12
Noolpuzha	*E. grandis*	1979	0	0	-*
Kottappara	*E. tereticornis*	1976	0	0	-*
Varavoor	*E. tereticornis*	1976	0	0	0
Mullachal	*E, tereticornis*	1978	0	0	,-*
Pezhad	*E. tereticornis*	1979	0	0	0

* Very low disease incidence
** observations not recorded

The progress of disease in a *E. grandis* plantation (1977) at Mavinhalla (Wynad) presented in Table 36 indicates that the mean infection (of all four plots) increased from the initial 0.8 per cent in 1980 to 27.58 per cent during 1482. The number of cankers per tree increased more during 1981-'82 than in 1980-'81. The possibility of tree to tree spread of the disease is evident from Fig.35. Usually fresh cankers developed on healthy trees which were around or in the vicinity of already severely infected trees. Curiously the above ground stem cankers were more common than the basal ones. Of the total cankered trees, basal cankers were present only in 13.3 per cent as compared to 87.7 per cent on the trunk. Gummosis was frequently associated more with the older cankers than the younger ones. Mortality of trees

Table 36. Incidence of canker fungus, ***Cryphonectria cubensis,*** in four observation plots at Mavinhalla during 1980-1982

Observation Plot	Total No. of trees observed	Year of observation	No. of cankered trees: Above ground canker	Basal canker	Total	Percent trees infected	Percent trees with basal cankers	Cankers with gummosis	Percent gummosis	No. of trees dead	Percent mortality in affected area
I	457										
		1982	144	22	166	36.3	13.2	59	35.5	1	0.5
		1981	33	11	44	9.6	6.6	29	17.5		
		1980	0	0	0	0	0	0	0		
II	420										
		1982	115	6	121	28.8	4.9	45	37.2	2	1.3
		1981	34	0	34	0	0	29	24.0		
		1980	0	0	0	0	0	0	0		
III	540										
		1982	105	5	110	20.4	4.5	17	15.4	7	5.3
		1981	15	4	19	3.5	3.6	10	10.0		
		1980	2	0	2	0.4	0	0	0		
IV	459										
		1982	129	17	146	31.8	11.6	21	14.4	6	3.1
		1981	21	19	40	8.7	13.0	23	15.7		
		1980	1	7	8	1.7	4.8	6	4.1		

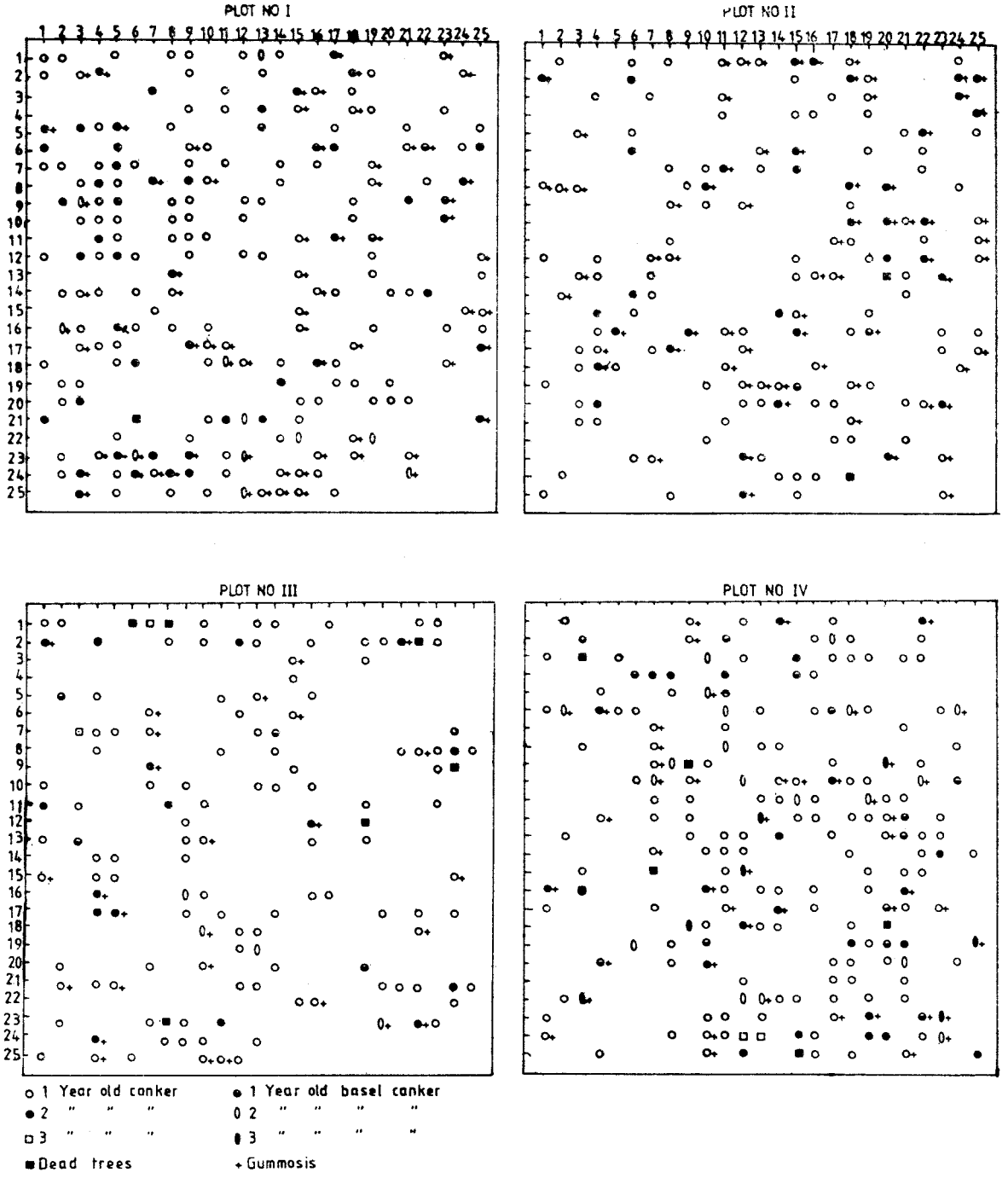


Fig. 35. Spread and severity of Cryphonectria stem canker disease (*C. cubensis*) in four observation plots.

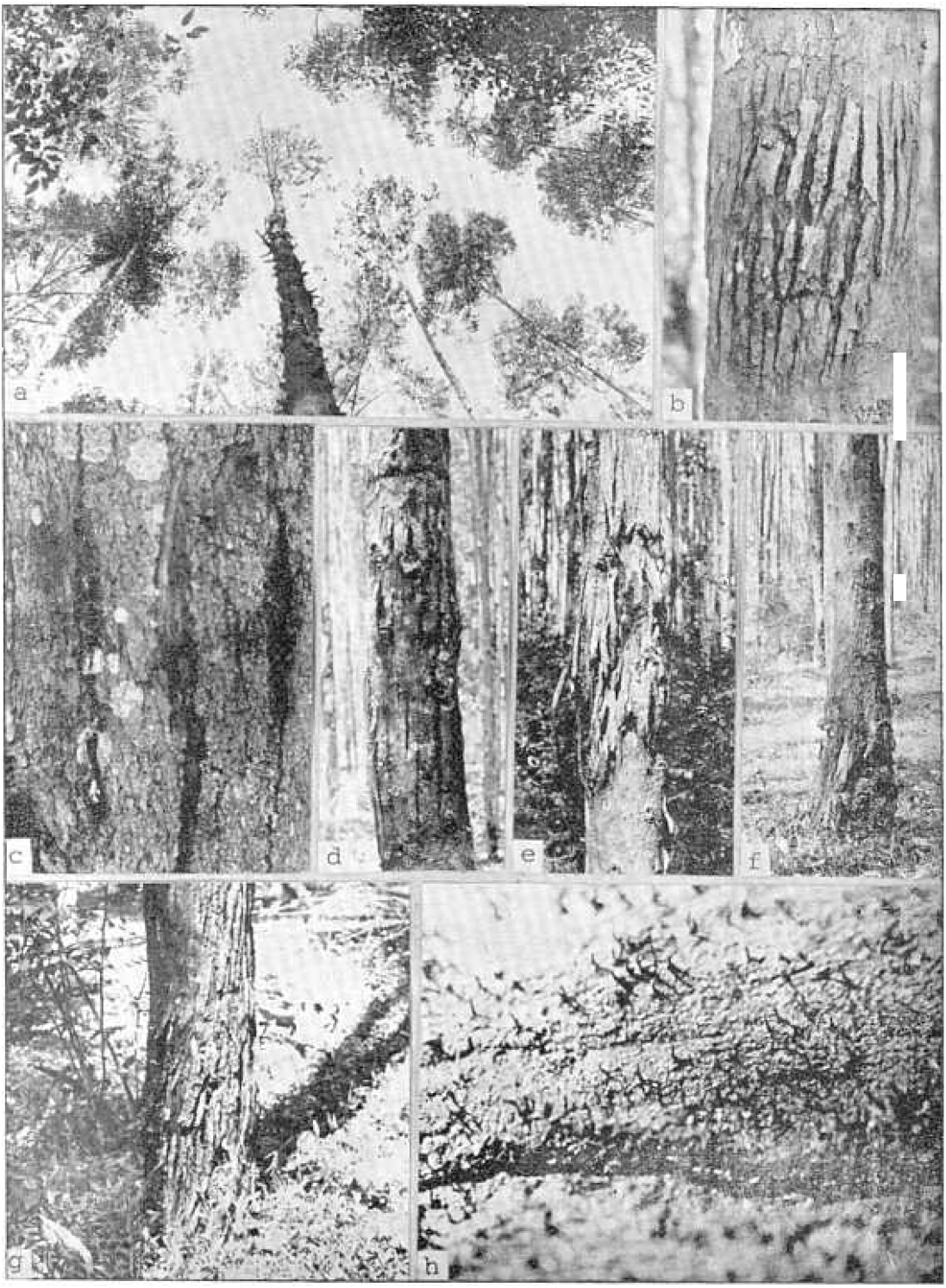


Fig. 36. Stem canker of *Eucalyptus grandis* caused by *Cryphonectria cubensis*. a, A view of plantation of *E. grandis* showing a dead tree in the centre and thinning of crown of other trees due to stem cankers; b, 1-year-old canker on 4-year-old tree; c, Exudation of kino from crevices formed on the canker; d, A 3-year-old canker; e, Stem canker more than 4-year-old on 8-year-old tree; f, A basal stem canker more than 4-year-old on 8-year-old tree; g, Confluent basal (3-year-old) and above ground (2-year-old) stem cankers on 8-year-old tree; h, Anamorph and teleomorph of *C. cubensis* on the dead bark.

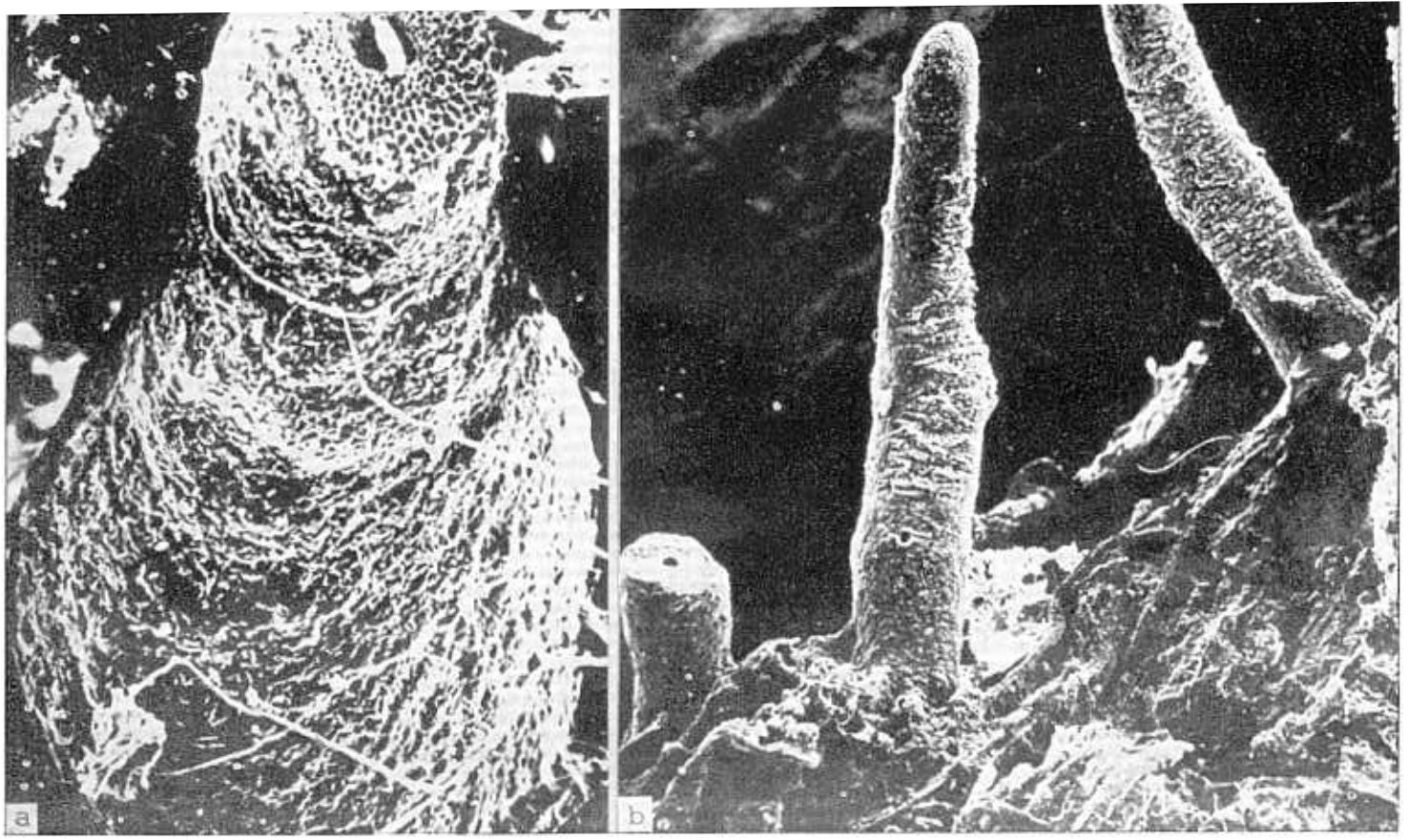


Fig. 37. Stem canker of *Eucalyptus grandis* caused by *Cryphonectria cubensis*; SEM of a pycnidium showing it being partially immersed in the bark (385X); b, SEM of ascocarps completely immersed in the bark (120X). Note the long protruding neck

was recorded in all the four plots with an average of 2.5 trees of the total affected ones. Large above ground cankers measuring more than a meter in length and basal cankers were occasionally responsible for the mortality of trees due to complete girdling of phloem (Fig. 36 a).

Symptoms

The first symptoms seen were slightly sunken, elongate areas measuring about 15-20 cm on the trunk either at the base or above ground, just after the South-West monsoon (Figs. 3a, b). The tissue beneath the depressed bark (inner bark) was brown and apparently killed. As canker developed during the dry period (December-April) (Figs. 3a, b) the bark showed vertical splitting, which increased in length and width with age (Fig. 36b). Generally at this time gummosis (oozing of kino) was observed in a few of the cankers (Fig. 36c). However, gummosis was commonly associated with the older cankers (2- to 3-year-old). The ruby coloured kino was usually washed down during the rains and imparted a distinct colour to the affected trees by which they could be recognised easily. Though the gummosis was fairly common in *E. **grandis*** it was not observed in ***E. tereticornis.*** Occasionally multiple cankers were found on the trunk which became confluent to form long cankerous areas (Figs. 36 d, e). Usually the cankers developed above the ground level and occasionally at the base (Figs. 36 f, 8). Mortality of trees was observed in ***E. grandis,*** ***E. citriodora*** and ***E. degluptu.***

Etiology

The identity of the pathogen, ***Cryphonectria cubensis*** (Bruner) Hodges, was confirmed by the Commonwealth Mycological Institute, where a few specimens bearing anamorphs and teleomorphs (Figs. 36h and 37a, b) (IMI 254084, 261569) and cultures (IMI 274337, 274339, 274340-274342, 274344,274345,274347,28 1616, 181617) have been deposited.

Cryphonectria cubensis was readily isolated on potato dextrose agar from the infected bark and grew satisfactorily at 25 $\pm$ 2^{o}C.

Pathogenicity test

For testing pathogenicity of the isolate, 3-year-old healthy trees of ***E. grandis,*** growing in a plantation where the disease incidence was very low, were used. The bark at the site of inoculation was cleaned with absolute alcohol and sterile water. For wound inoculation an inverted 'V' shaped deep cut was made in the bark with a sharp sterile chisel (2.5 cm wide). The cut flap was pulled open gently and an agar disc bearing mycelium and pycnidia from a culture of *C. cubensis* inserted between the bark flap and sap wood, and the flap closed. A sterile moist cotton swab was placed over the wound. To ensure high humidity around the inoculated site a

polythene cover, the under side of which was sprayed with sterile water, was tied on the stem with a swab of moist cotton placed on one side. The upper edge of the polythene was sealed with a mixture of paraffin and bees wax to protect the inoculated site from rain water. Inoculation without injury was done by keeping the agar disc of inoculum over the bark. Control inoculations were made in the same way using discs of potato dextrose agar without test fungi. A total of 20 trees (ten each with and without injury) were inoculated. Observations were recorded at monthly intervals.

All wound-inoculated trees became infected but only 20 per cent of non-wounded ones did. Infection of the bark was seen in the form of a prominent depression around the inoculated site, which spread more longitudinally than horizontally. Three months after inoculation large number of pycnidia were noticed with long yellowish orange tendrils, from which a pure culture of ***C. cubensis*** was obtained. Subsequently perithecia also developed on the cankered area. No infection occurred in the controls.

Susceptibility of various ***Eucalyptus*** provenances

Seeds of various provenances belonging to different species of ***Eucalyptus*** were obtained from the Division of Forest Research, Commonwealth Scientific Industrial Research Organization, Australia. Seedlings were raised in the nursery of the Pathology Division of the Institute. Two-year-old plants of ***E. grandis*** (Nos. 12970, 13022), ***E. tereticornis*** (Nos. 13319, 13398, 13418), ***E. camaldulensis*** Dehnh. (No. 12013). ***E. deglupta*** Bl. (No. 12322) were inoculated in May 1983 as described under the pathogenicity test. Observations were frequently recorded on the development of fructifications and cankers and death of plants.

The inoculated stems of all the eucalypt species produced a canker and numerous pycnidia within three months. Plants started to die after five months. The provenances did not differ in their response to infection. Abundant perithecia developed on the dead stems during the following monsoon (June-August 1984). During this period the disease also spread to nearby uninoculated plants of ***E. saligna*** Sm. (No. 13027), ***E. brassianu*** S. T. Blake (Nos. 13410, 13415), *E.* ***camaldulensis*** (No. 12181), *E.* ***pellita*** F. Muell. (No. 13165) and *E.* ***cloeziana*** F. Muell. (No. 12945) and caused mortality.

Influence of canker disease on coppicing

Observations on coppicing by stumps of trees of ***E. grandis*** (planted 1975) at Mavinhalla felled during April 10 - 17, 1984 were recorded 5 months later in a randomly selected plot of 10 X 50 stumps. All the stumps showing gummosis due to canker infection were scored for extent of gummosis, number of clumps of coppice shoots and their height. Similar observations were also made for healthy stumps

with no gummosis except that it was not possible to count the clumps of coppice shoots and, therefore, only number of shoots was recorded.

Of the 464 stumps present in the plot only 97 (20.9 per cent) showed gummosis. However, the incidence of the disease must have been higher because gummosis was often not produced upto the base of the trunk in above ground cankers. The per cent stumps sprouted decreased with increasing gummosis (Table 37) and when the stumps were completely gummosed the sprouts developed from its basal part instead of from the cut edge as they normally do. On diseased stumps, fewer sprouted clumps developed multiple coppice shoots which varied from a few to as many as 34 as compared to 6-15 on healthy ones. Because of the large number of shoots per clump in gummosed stumps, the shoots remained stunted and weak compared with those on healthy stumps. About 4 per cent of the sprouts on gummosed stumps dried up while ca. 4 per cent of healthy stumps did not sprout mostly because of faulty harvesting operations but occasionally for unknown reasons.

Parameter	Stumps without gummosis (Mean of *50* stumps)	Stumps with gummosis*				Mean
		Extent of gummosis (circumference of stump)–				
		$\frac{1}{4}$	$\frac{1}{2}$	$\frac{3}{4}$	Full	
% Sprouting	92	86.4	59.4	50.0	31.2	56.7
Number of sprout clumps, shoots per stump	10.1 (shoots)	3.0[a]	2.6[b]	2.7[a]		3.0 (clumps)
Average height of shoots (cm)	97.4	—	—	—	—	26.5

[a]Sprouts dead on one stump

[b]Sprouts dead on two stumps

*Total Number, 97

Discussion

C. cubensis was first reported from Cuba (Bruner, 1917) where it caused stem canker of various eucalypts. It was not reported again until 1970 when it was reported as *Endothia havanensis* Bruner by Boerboom and Maas from Surinam. Subsequently, Hodges and Reis (1974) also recorded it under the same name from Brazil. It was only later that Hodges et al. (1976) showed that what was described as *E. havanensis* from Brazil and Surinam was *Diaporthe cubensis* Bruner and recently Hodges (1980) transferred it to genus *Cryphonectria.*

This is the first time that ***C. cubensis*** has been recorded on *Eucalyptus* in India. Previous records from Cuba, Florida, Hawaii, Puerto Rico, Brazil (Hodges and Reis, 1974; Hodges *et al.,* 1979), Surinam (Boerboom and Maas, 1970), Western Australia (E. M. Davison, personal communication) and Hong Kong, Cameroons and Venezuela (D. W. Minter, personal communication) indicate that it is distributed within 30°N and S of the equator. The distribution is probably determined by the tropical humid climate needed for growth and spread of the pathogen. Thus earlier reports from Brazil and Surinam indicated that the disease was favoured by high rainfall (2000-2400 mm p.a.) and temperature above 23°C, the optimum temperature for the growth of *C. cubensis* being between 20°C and 32°C (Boerboom and Maas, 1970; Hodges *et al.*. 1976). The spatial distribution and varied severity of the pathogen in eucalypt plantations in Kerala also appears to be related to the climatic conditions. High rainfall areas of Kerala like Wynad, Munnar, Pamba (Figs. 3 a,b), where the average temperature is 25°C or more appear to be suitable for the disease.

Both the natural occurrence of the disease and results from inoculation trials in Brazil show that there is considerable inter- and intra-specific variation in susceptibility to the fungus (Ferreira *et al.,* 1977; Ferreira *et al.,* 1978). The low disease incidence and mortality of ***E. grandis*** in Kerala (2.5 per cent) as compared to Brazil (30 per cent) may reflect differences in provenances of this species which vary considerably in their relative susceptibility (Hodges *et al.,* 1976; Hodges *et al.,* 1979) or the low virulence of the pathogen. Differences in the levels of infection in plantations of ***E. grandis*** and *E. **tereticornis*** suggests that the latter species is more resistant than the former. This variation does not appear to be due to climatic differences in planting areas of these two species as in a few coppiced (second rotation) ***E. tereticornis*** plantations in Wynad, which were restocked with ***E. grandis,*** the canker disease is prevalent in the latter but not in the former species. Further evidence in this regard is available from the same-aged plantations of these two species at Mullachal. In Brazil, Ferreira *et al.* (1978) also rated provenanccs of ***E. grandis*** (+ 45) and ***E. tereticornis*** (10914) respectively as moderately susceptible and resistant. However, Hodges *et al.* (1976) have graded both the species in the same category of moderately susceptible eucalypts. Similarly ***E. citriodora, E. torelliana*** and ***E. deglupta,*** found to be highly rcsistant in Brazil, are moderately susceptible under Kerala conditions. Although the disease in Kerala was identified only during 1980 it probably occurred in an adjacent 4-year-old plantation of ***E. grandis*** (planted 1975) in 1979 where gummosis was quite prevalent. However, at the time it was considered to be a physiological disorder on the basis of symptoms described by Bakshi (1972) and May (1973) which were strikingly similar to the disease in Wynad plantations.

The basal cankers are known to reduce the sprouting of stumps by about 10-20 per cent in Brazil (Hodges and Reis, 1974; Hodges *et al.*, 1976). In Kerala, even though

the frequency of basal cankers was less, about 35% of the stumps affected with the disease (indicated by gummosis) failed to produce coppice shoots. If they coppiced at all, shoots developed mostly near ground level. It seems that excessive gummosis kills the tissues of the outer bark as do the cankers and thus bring about sprouting failure. In such plantations, eventhough the mortality was only about 3%, the impact of the disease was greater on the coppice crop of the second rotation. There is a possibility that the loss of additional trees through lack of sprouting may reduce the stocking for succeeding rotations below an acceptable economic level.

There are some striking differences between the epidemiology of the disease in Brazil and in Kerala. In Brazil the pathogen attacks trees of susceptible species (***E. saligna*** Sm., ***E. maculata*** Hook) as young as five months old (Hodges *et al*,, 1976) whereas in Kerala the earliest recorded symptoms on ***E. grandis*** have been on 2- to 3-year-old trees. In Brazil, the principal symptoms are basal cankers whereas in Kerala most of the cankers were found above ground, upto several meters high. More investigations are needed to determine whether these differences are related to the eucalypt species grown in Kerala, to different strains of the pathogen, or to the influence of soil-micro-climatic conditions.

Although at present the level of canker is low, with a maximum mortality of about ***3%,*** the anticipated increase in inoculum over the years in the conducive climate of Kerala could pose a serious threat to eucalypts, as in Brazil. The long term control of disease in a forestry crop is possible only either by field selection or by breeding for resistance. In Brazil stable resistance to Cryphonectria stem canker has already been obtained by intensive field selection followed by vegetative propagation. As a first step in this direction more than 40 eucalypts (various provenances belonging to different species) are being screened against ***C. cubensis*** as well as ***Corticium salmonicolor*** and ***Cylindrocladium*** spp. in Kerala. However, as an immediate measure to check the further spread of canker, chemical control need to be attempted, although it may not be economical for a forestry crop like ***Eucalyptus*** with very low returns.

6. CRYPHONECTRIA GYROSA STEM CANKER

Occurrence and severity

This stem canker disease was first recorded at Thrissillery in a 4-year-old plantation of ***E. grandis*** during 1980. Later it was observed on ***E. grandis*** at Mavinhalla and Mullachal and other species such as ***E tereticornis, E. torelliana, E. deglupta*** and ***E. alba*** at Kottappara and Vazhachal (Vazhachal Div.). Of the 11 plantations of eucalypts surveyed this disease occurred only in 4 plantations (Table 38). The disease caused twig, branch and main stem cankers. Death of twigs and branches was common but seldom it was observed for a tree, except in ***E. torelliana,*** which appeared to be more susceptible than other species.

The disease, initially recorded at Thrissillery during 1980, progressed slowy and spread to other trees increasing severity from low to medium (Table 38). In other plantations (Mavinhalla, Pezhad and Mullachal) the disease could be observed only during 1981 in very low incidence. Though the disease severity increased slightly it had only low rating.

Table 38. **Incidence and severity of stem canker caused by *Cryphonectria gyrosa* in eucalypt plantations at different localities in Kerala during 1980-1982**

Eucalyptus species Locality	1980			1981			1982		
	% incidence	DSI[a]	DSR[b]	%incidence	DSI	DSR	% incidence	DSI	DSR
E. grandis									
Thrissillery	0*	0.0	Nil	6.76	0.87	L	55.02	1.01	M
Mavinhalla	0*	0.0	,,	2.46	0.25	L	9.15	0.76	L
Noolpuzha	0	0.0	,,	0	0.0	Nil	0	0 0	Nil
Papathishola	0	0.0	,,	3.71	0.01	L	16.04	0.19	L
E. tereticornis									
Varavoor	0	0.0	,,	0	0 0	Nil	0	0.0	Nil
Kottappara	0	0.0	,,	0	0.0	,,	0	0.0	,,
Pezhad	0	0.0	,,	0*	0.0	,,	6.66	0.05	L
Anakulam	0	0.0	,,	0	0.0	,,	0	0.0	Nil
Mullachal	0	0.0	,,	0	0.0	,,	0	0.0	,,
E. globulus									
Silent Valley	0	0.0	,,	0	0.0	,,	0	0.0	,,

[a]DSI Disease Severity lndex
[b]DSR Disease Severity Rating
*Disease of low severity

Symptoms

The infection occurred on the lower half of stem, often near the ground during the monsoon and by September/October depressed areas, 30-45 cm long on the bark became visible. The tissues beneath the canker turned necrotic and got killed. During the dry period these cankers developed some splitting on the bark (Fig. 38 a). The pycnidia orange red in colour, developed over the bark arranged in vertical linear rows (Figs. 38 d, e) or scattered during the wet period (June-September). The pycnidia produced long orange yellow tendrils of spores during monsoon (Figs. 38 b, c). Characteristic ascomata with long beaks developed during the following dry periods (December-April) on the infected bark (Figs. 39a-e) either separately or interspersed with pycnidia. Death of ***E. torelliana*** trees occurred when

the cankers girdled them completely. No gummosis was noticed on the cankers as with ***Cryphonectria cubensis.***

Etiology

Cryphonectria gyrosa (Berk. & Br.) Sacc. (Syn. ***Endothia havanensis)*** (Bruner) Baur. (IMI 261575, 274336, 274343, 274346, 281618).

Pathogenicity

The pathogenicity of *C.* ***gyrosa*** was tested on healthy 4-year-old ***E. grandis*** at Thrissillery during August 1980. For inoculation same procedure as described under ***C. cubensis*** was followed (Fig. 39f).

Within a month of inoculation the bark around the inoculated site showed depression measuring 15-20 cm long and 8-10 cm wide. In the following months bright orange coloured pycnidia developed over the infected bark (Fig. 39 g). Beneath the bark, the tissues showed browning, though no effect was noticed on the sapwood. During the dry periods the cankers became visible due to depression which enlarged by this time upto 30-35 cm in length with vertical splitting of bark. During the dry period the growth of the cankers appeared to be slow or inhibited and some callus was seen developing at the margins, to check the spread of infection.

Susceptibility of various ***Eucalyptus*** provenances

Following the procedure used for testing the pathogenicity (see page 147) 2-year-old container seedlings of *E.* ***tereticornis*** (Seed lot nos. ex CSIRO Australia 13319, 13398, 13418), ***E. grandis*** (12970, 13022), *E.* ***pellita*** (11947, 12013), ***E. brassiana*** (13410, 13415), ***E. saligna*** (13027), ***E. camaldulensis*** (12181) and ***E. microcorys*** (12795) were inoculated during May 1984. The inoculated seedlings were observed regularly for the development of the disease and mortality, if any.

Only *E.* ***grandis*** (13022), ***E. tereticornis*** (13398), ***E. urophylla*** (12896), ***E. pellita*** (11947) and ***E. saligna*** (13027) got infected and produced cankers. During August/ September numerous pycnidia were produced with characteristic conidial ooze as tendrils. All the infected seedlings died; perithecia developed on the dead stem during November-February.

Growth of ***C. gyrosa*** on various media

Eight media viz. glucose tyrosine agar (GTA), yeast malt agar (YMA), V-8 juice agar (V-8), lima bean agar (LBA), potato dextrose agar (PDA), malt extract agar (MEA), corn meal agar (CMA) and Czapek's dox agar (CDA) were selected for this study. A disc, 5 mm in diam, punched from the margin of an actively growing colony of *C.* ***gyrosa*** was transferred at the centre of the petri dish containing the test

medium. There were three replicate plates for each medium. The plates were incubated at 25 ± 2°C and observations on diameter growth recorded on 4, 6 and 8 days after inoculation.

Initially on the fourth day LBA, PDA and MEA found to be good media showing vigorous growth (Table 39). However, on eighth day the growth in LBA slowed down and CMA, where it was initially slow, showed considerable improvement. Hence, on the eighth day PDA. MEA and CMA supported the best growth of C. ***gyrosa.*** Curiously no growth occurred on CDA.

Table 39. **Diameter growth of C. *gyrosa* on various media at 25 ± 2°C (Mean of 9 observations and SD)**

Sl. No.	Growth medium	Mean diameter growth (mm) at different days of incubation		
		4th	6th	8th
1.	Glucose tyrosine agar (CTA)	25.25(2.13)	46.16(1.19)	61.41(2.60)
2.	Yeast malt agar (YMA)	30.58(1.24)	56 33(2 42)	74.16(2.40)
3.	V-8 juice agar (V-8)	24.75(1.48)	43.58(1.08)	53.91(2 15)
4.	Lima bean agar (LBA)	36.41(1.08)	51.91(3.60)	70.12(7.47)
5.	Potato dextrose agar (PDA)	37.91(3.82)	65.75(5.54)	80.33(1.37)
6.	Malt extract agar (MEA)	35.83(3.12)	63.50(4.05)	80.66(2.05)
7.	Corn meal agar (CMA)	30.33(3.11)	58.16(2.85)	79.50(1.56)
8.	Czapek's dox agar (CDA)	0.0	0.0	0.0

Evaluation of fungicides against C. ***gyrosa***

A total of 15 fungicides viz. Benlate, Bavistin, Bayleton, Difolatan, Demosan, Daconil, Dithane M-45, Dithane 2-78. Furmatamid, Fytolan, Hexacap (Capfan). Polyram Combi, Saprol, Syllit and Tecto were evaluated at 0.1, 0.25 and 0.5% (a. i.) concentration for their efficacy against ***C. gyrosa*** following poison-bait technique. Each concentration was replicated in three petri plates.

The results presented in Table 40 indicate that the most effective fungicides at the lowest concentration were Benlate, Bayleton, Bavistin, Furmatamid, Saprol and Tecto which inhibited the growth of the test fungus completely.

Discussion

Cryphonectria gyrosa was first described as ***Endothia havanensis*** from Cuba (Bruner, 1917) on various eucalypts (*E.* ***occidentalis*** End1 **,** ***E.botryoides*** Sm., *E.* ***rostrata*** Schlecht., *B.* ***microphylla*** Willd., ***E. robusta*** Sm.), ***Peraea gratissima*** Geertn. f,

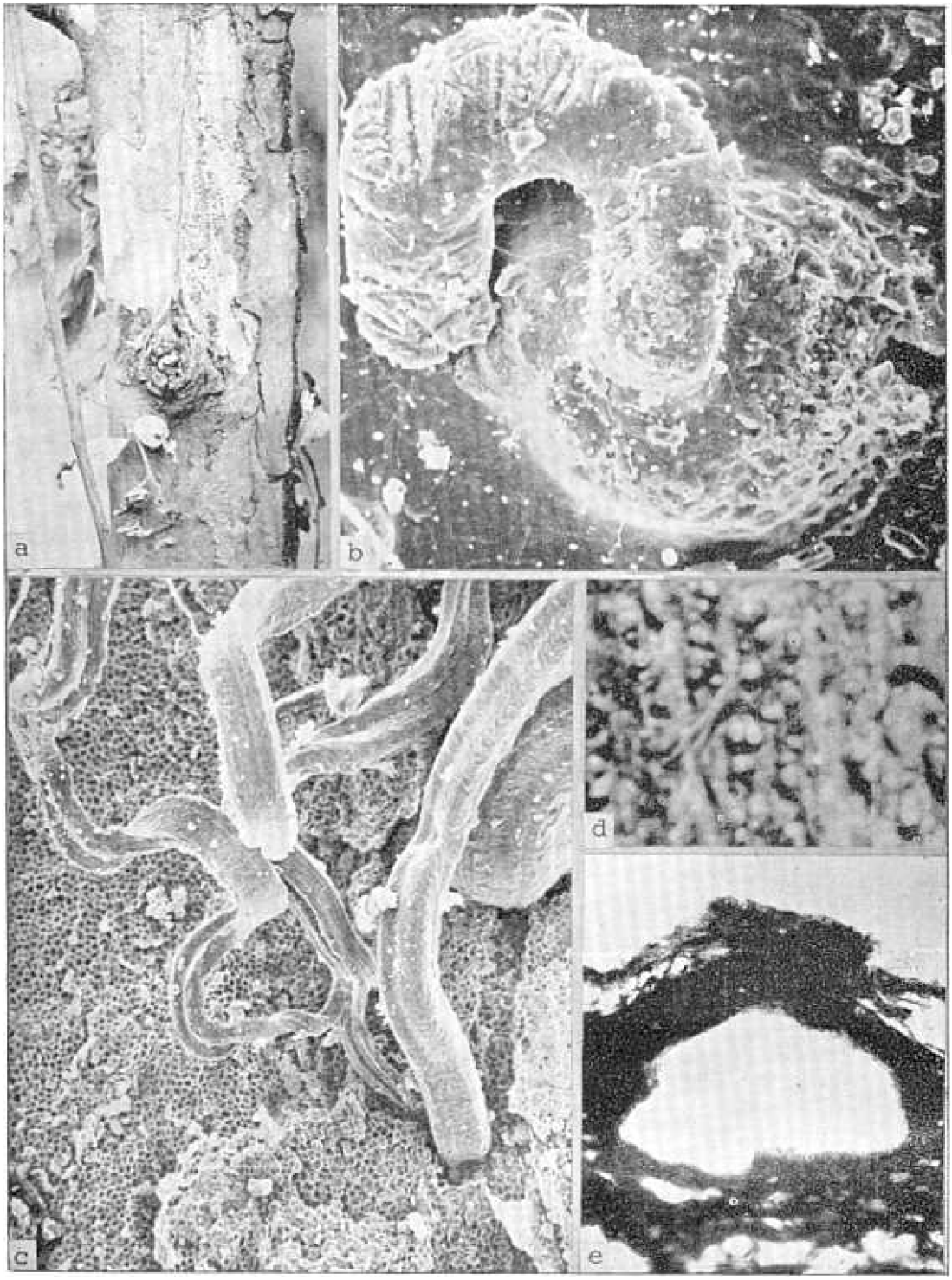


Fig. 38. Stem canker of *Eucalyptus* caused by *Cryphonectria gyrosa*. a, Stem canker on *E. torelliana*. Note the fructifications on the canker; b, SEM of a partially immersed pycnidium in the bark of *E. grandis* with a spore tendril (140 X); c, A long spore tendril produced by a pycnidium completely immersed in the bark of *E. grandis* (35 X); d, Pycnidia arranged in vertical rows over the bark of the canker; c, A vertical section through a pycnidium (70 X).

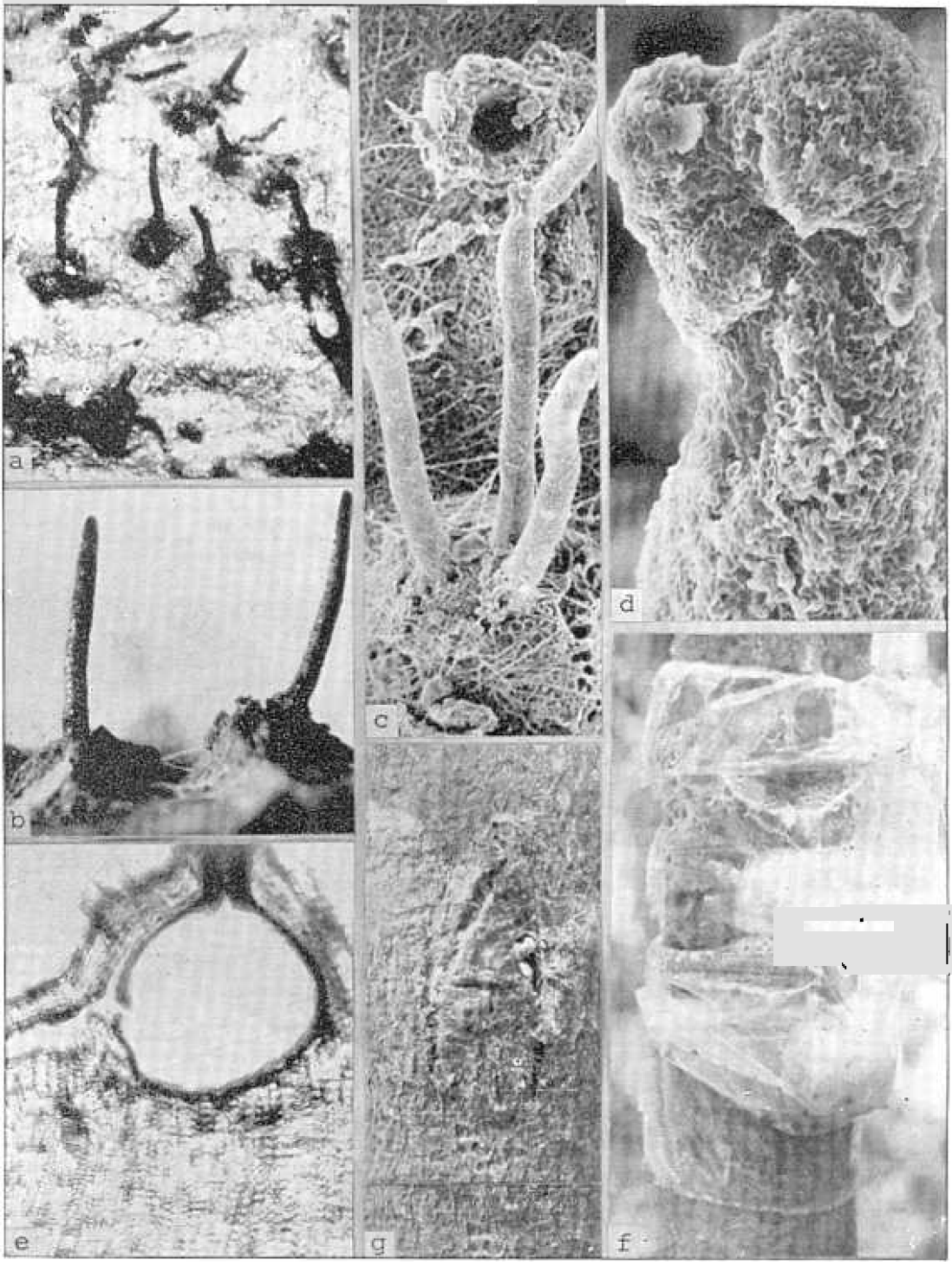


Fig. 39. Teleomorph of *Cryphonectria gyrosa*. a, b and c, Perithecia partly immersed in the bark of *E. torelliana*; c, SEM of a clusture of perithecia (40 X); d, SEM of a tip of the neck of perithecium to show the opening situated in a depression (570 X);e, A vertical section through a perithecium (70 X); f and g, Photographs showing method of artificial inoculation; g, Development of a canker at the inoculated site over which numerous pycnidia developed.

indica L., and *Spondias mombin* L. Since then it has been reported in Japan on *E. globulus* (Kobayashi and Ito, 1956), in Australia on *E. marginata* (Davison 1982, Davison and Tay, 1983). This is the first record of *C. gyrosa* on *Eucalyptus (E. grandis, E. tereticornis, E. torelliana E. deglupta, E. alba)* from India.

Table 40. Evaluation of fungicides for their efficacy against ***C. gyrosa*** following poison-bait technique (mean of 12 observations fron 3 petri dishes)

S1. No.	Fungicide	Percent inhibition in diameter growth over control on 10th day of incubation		
		0.1% (a. i.)	0.25% (a. i.)	0.5% (a. i.)
1.	Benlate	100.00	100.00	100.00
2.	Bavistin	100.90	100.00	100.00
3.	Bayleton	100.00	100.00	100.00
4.	Difolatan	50.51	51.81	59.79
5.	Demosan	75.22	84.12	100 GO
6.	Dithane M-45	19.41	78.49	100.00
7.	Dithane 2-78	18.73	63.85	78.73
8.	Daconil	23.94	44,44	86.81
9.	Furmetamid	100.00	100.00	100.00
10.	Fytolan	57.26	80.23	87.15
11.	Hexacap (Captan)	47.09	56.36	55.86
12.	Polyram Combi	26.07	77.73	100 GO
13.	Saprol	100.00	100.00	100.00
14.	Syllit	92.95	84.76	89.05
15.	Tecto	100.00	100.00	

Recently, Barr (1978) transferred *Endothia havanensis* to *Cryphonectria havanensis* keeping along with *C. parasitica* (Murr.) Barr, *C. radicalis* (Schw. ex Fr.) Barr, *C. nitschkei* (Otth) Barr, and *C. macrospora* (Kobayashi Ito) Barr in a group typified by *C. gyrosa.* Later, Hodges (1980) who found Barr's observations incorrect in considering *E. havanensis* as distinct from *C. gyrosa,* considered both as synonyms based on diamensions of ascospores.

During the artificial inoculation trials only a few provenances of *Eucalyptus,* appeared to be susceptible to *C. gyrosa.* However, the susceptible nature of other provenances giving negative results cannot be ruled out. The reason advanced for this view could be the young age of plants which due to bark characteristics failed to take infection; in plantations ***C.*** *gyrosa* was found only on > 3-year-old plants.

Though C. *gyrosa* is capable of killing branches and trees, field observations as well as artificial inoculation trials indicate that C. *gyrosa* is a weak pathogen. Often it was associated with cankers of *C. cubensis.* In older trees (> 6-year-old) C. *gyrosa* caused mild cankers without any apparent damage. The only eucalypt species, which was found to be highly susceptible to C. *gyrosa* was *E. torelliana* recording ca. 10 per cent mortality at Kottappara.

Fungicidal evaluation studies indicate that there are quite a few fungicides effective against C. *gyrosa,* which may be used in controlling the canker disease, should it spread in epidemic form. Though chemical control of this disease is not a long-term solution, till the time we have promising canker disease resistant/tolerant eucalypts, chemical control may be adopted to check its further spread.

7. VALSA STEM CANKER

Occurrence and severity

Low incidence of the disease was recorded in young (I-to-4-year-old) plantations of *E. grandis* at Thrissillery (9.29 per cent), Mullachal and *E. torelliana* at Vazhachal (Vazhachal Div.). At Meenmutti (Kerala Forest Development Corp., Idukki) the disease caused extensive juvenile twig and branch cankers resulting in die-back of > 30 per cent plants of *E. grandis.*

Symptoms

Generally, the cankers were common on twigs and branches and occasionally on main stem. Usually the infection was initiated at the base of the branch, where a canker developed. Over the dead bark numerous black fructifications were produced (Figs. 40 a-e). Often more fructifications were produced on the lower surface of a branch, away from the direct sunlight. The affected twigs and branches died due to complete girdling of phloem tissue.

Etiology

Valsa eucalypti Cook & Harkness (IMI 261564) and *Valsa eucalypticola* sp. nov. (IMI 257896, 261568).

Discussion

Valsa eucalypti and *V. eucalypticola* sp. nov. are the teleomorphs of *Cytospora eucalypti* sp. nov. and *C. eucalypticola,* respectively reported for the first time from India on *Eucalyptus* spp. Since *Valsa* spp. mostly cause cankers on lower branches, which eventually die as a result of natural pruning, it does not appear to be of serious concern. Extensive die-back of *E. grandis* at Meenmutti was due to dual infection of *V. eucalypti* and *Cytospora eucalypti.* But generally it is not common for anamorph and teleomorph stages to be present on the same affected part (Sutton, 1980).

8. MACROVALSARIA STEM CANKER

Qccusrence

The direase was observed in isolated trees of *E. tereticornis* at Elanad (Trichur Div.), Potta (Kerala Forest Development Corp., Trichur), Pezhad and *E. grandis* at Mullachal. All the affected trees were either dead or showed wilting of leaves.

Symptoms

Generally the infection occurred at the basal part of the stem, characterized by a large number of black fructifications, scattered over the bark (Figs. 41a-d.) The affected stem developed canker and the underneath tissues showed browning. When the stem was completely girdled the foliage wilted and the tree slowly died; no epicormic shoots developed.

Etiology

Macvovalsaria megalospora (Mont.) Sivan. (IMI 261566) (Figs. 41a-e).

Discussion

Since the disease was recorded in low incidence it appears to be an unimportant one. This is the first report of *M. megalospora* causing stem canker of *Eucalyptus.*

9. THYRONECTRIA STEM CANKER

Occurrence

The disease was recorded in *E. tereticornis* at Kakkavayal (Kozhikode Div.), Vazhachal (Vazhachal Div.), Anakulam and in *E. camaldulensis* and *E. torelliana* at Vazhachal (Vazhachal Div.).

Though the disease was observed to kill the trees in some plantations, it appeared to be unimportant as the incidence was very low (>1 per cent). Mortality of trees was observed in *E. tereticornis* (Anakulam; 3-year-old), in *E.*

(Vazhachal; 4-year-old) and ***E. grandis*** (Thrissillery; 4-year-old). Occasionally, branch infection was also noticed in ***E. torelliana*** which killed the shoots outright.

Symptoms

Initially the conidial state of the pathogen (anamorph) (Figs. 42 a-c) developed near the base of the stem, which soon spread upwards covering a large area. The affected tissues of the stem developed browning and when stem completely girdled it brought about wilting and consequently death of tree. The perfect stage (teleomorph) (Figs. 42 d, e) were formed in loose clusters or scattered over the dead stem. No epicormic shoots were observed on the affected trees.

Etiology

Thyroizectria pseudotricha (Schw.) Seeler (IMI 252784, 257897) (Figs. 42 a-e).

Discussion

Though ***X. pseudotricha*** is considered to be a wound parasite no injury/wounds were observed in any of the affected trees examined. The pathogen, causing branch as well as main stem cankers, is capable of killing the trees. This is the first record of *T.* ***pseudotricha*** on ***Eucalyptus.***

10. HYSTERIUM STEM CANKER

Occurrence

The disease was only recorded at Vazhachal (Vazhachal Div.) in an experimental plantation of ***E. camaldulensis*** (4 year-old). All the affected trees died due to girdling of stem by the canker.

Symptoms

The pathogen caused extensive cankers on branches and upper part of the stem. The affected tissues of the stem showed pronounced browning. On dead stem numerous characteristic black fructifications developed, aggregated or singly (Fig. 42 f).

Etiology

Hysterium angustaturn Alb. & Schwein (IMI 257895) (Figs. 42 f-h).

Discussion

H. angustaturn commonly occurs on ***Acer, Almus, Fagus, Fraxinus,*** etc., in temperate countries (Dennis, 1981). This is the first record of occurrence of ***H. angustaturn*** on ***Eucalyptus.*** Because of the localised occurrence, ***Hysterium*** stem canker may be considered as an unimportant disease of ***Eucalyptus.***

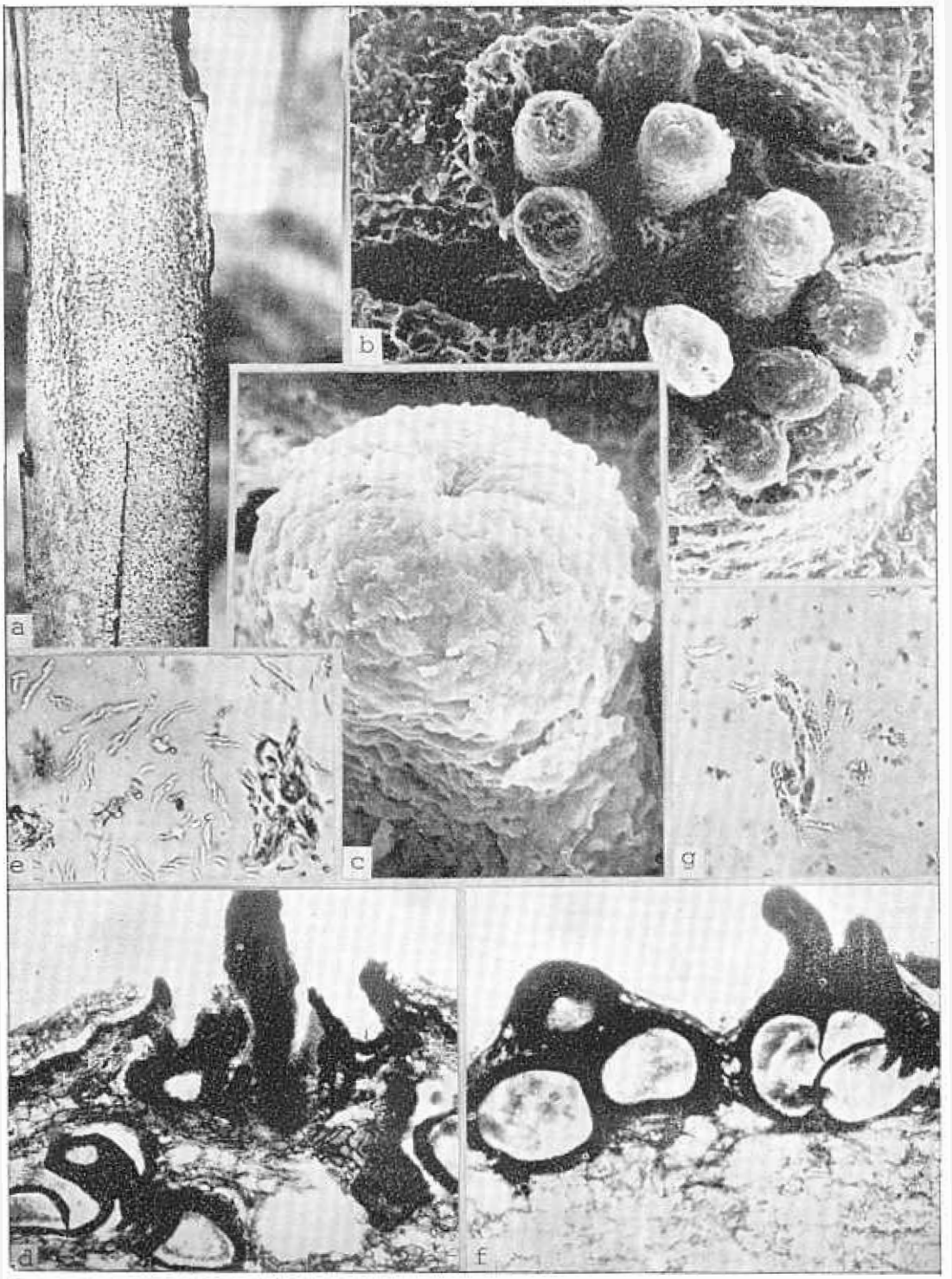


Fig. 40. Stem canker of *Eucalyptus* caused by *Valsa*. a, A branch of *E. grandis* affected with *V. eucalypti*. Note numerous dark coloured perithecia over the canker; b, SEM of ascomata deeply immersed in the pseudostromata (70 X); c, SEM of a tip of a perithecial neck to show ostiole (500 X); d, A vertical section through ascomata (60 X); e. Asci of *V. eucalypti* each containing 8, one-celled biseriate ascospores (400 X); f, A vertical section through ascomata of *V. eucalypticola* (60 X). Note that ascomata contains a few perithecia which are not deeply immersed as in *V. eucalypti*; g, Asci of *V. eucalypticola* containing 8, one-celled ascospores (400 X).

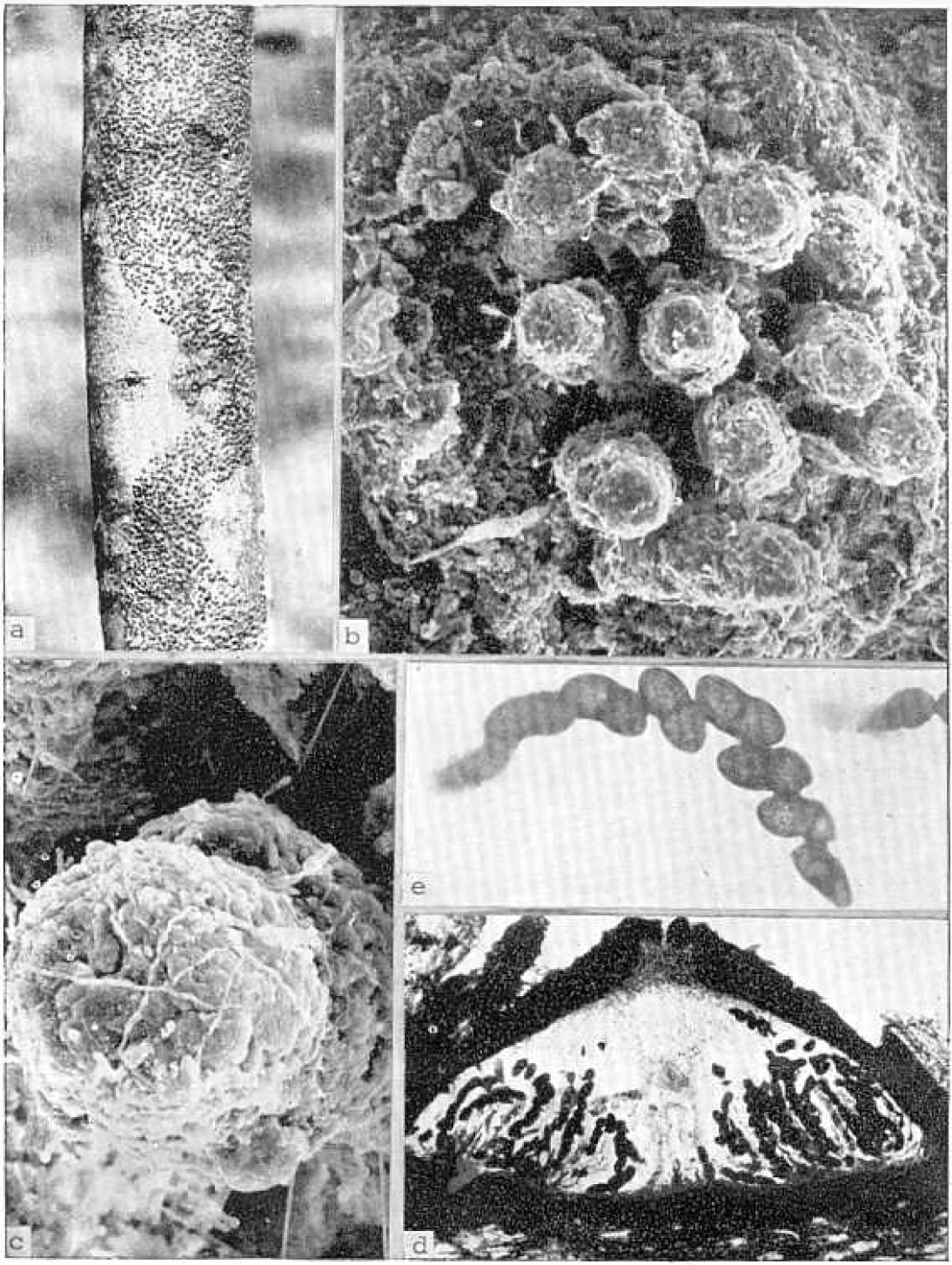


Fig. 41. Stem canker of *Eucalyptus* caused by *Macrovalsaria megalospora*. a, The main stem of *E. tereticornis* affected with canker. Note numerous ascomata over the canker; b, SEM of ascomata (100 X); c, SEM of the tip of a perithecium (430 X); d, A vertical section through the perithecium showing arrangement of asci (60 X); e, Bicelled ascospores. Note the lower cell with striations.

11. PINK DISEASE

Occurrence and severity

The pink disease was widespread in plantations of ***E. tereticornis*** throughout Kerala with incidence varying from 25 to> 75 per cent depending upon the rainfall of the area. Severe infection with high mortality of top shoots was recorded at Varavoor, Vazhachal (Vazhachal Div.), Kottappara, Pezhad, Thalakode, Neriamangalam (Kothamangalam Div.), Arippa (Kerala Forest Development Corp., Trivandrum) and Onthupacha.

In all the plantations surveyed, except at Mullachal where the severity was low throughout, it increased from low to medium (Kottappara, Pezhad and Anakulam) or remained at medium (Varavoor) during the three years of observations (Table 41).

In ***E. grandis,*** pink disease was recorded at Noolpuzha and Mullachal during 1980 and at Periya and Thrissillery during 1981. Initially only a low incidence of the disease (1 per cent or below) was observed in the form of branch cankers. In the following years an outbreak of pink disease was recorded in epidemic proportion at Noolpuzha and Periya; in other plantations the severity remained low. To monitor the progress of pink disease at Noolpuzha five plots of 400 trees (20 x 20 stakes) each were selected at random in a 1978 plantation and all the trees periodically assessed. At Periya though the pink disease was observed during 1981, plots were marked and observations recorded in 1983 only. The ***E. grandis*** plantation at Periya was originally of ***E. tereticornis*** in second rotation (coppiced 1979) where > 60 per cent stocking was that of ***E. grandis*** as casualty replacement. Yearly observations on the severity of the pink disease are given in Table 41.

Infection spread rapidly at Noolpuzha and Peryia and within 2-3 years more than 50% of ***E. grandis*** trees were found to be affected with pink disease, though the mean disease severity was recorded as low. At Noolpuzha, during 1980 and 1981 only branch cankers (shoot infection) were observed; the affected branches were killed outright. In 1982, however, typical stem cankers developed. The same pattern was observed at Periya. When the cankers ring-barked the trees, typical die-back of the main shoot occurred. As a result of infection a large number of epicormic shoots, characteristic of pink disease of ***E. tereticornis,*** produced during the same season were also found to be infected. At the site of fresh infection cob web, pustule as well as pink stages were present; necator stage developed on cankers during the dry period December-April).

Symptoms

Usually the disease was observed to attack 2-year-old and more older plants, but infection on l-year-old plants and coppice shoots was not uncommon. The fungus

Eucalyptus species	Locality	Year of planting	1980		1981		1982		1983	
			DSI[a]	DSR[b]	DSI	DSR	DSI	DSR	DSI	DSR
E. grandis	Thrissillery	1977	0.0 br	Nil	0.15br	L	0.82br	L	—	—
	Mavinhalla	1977	0.0	Nil	0.0	Nil	0.04	L	—	—
	Noolpuzha	1978	0.05br	L	0.18	L	1.07		1.17	M
	Periya (coppiced)	1979	—	—	—	ab	—	ab	0.95	L
				Nil	0.0	Nil	0.08	L	—	—
			0.14br	L	0.13br	L	0.50br	L	—	—
E. tereticornis	Varavoor	1976	1.84	M	I.25	M	1.14	M	—	—
	Kottappara	*1977*	0.56	L	0.73	L	1.06	M	—	—
	Pezhad	1978	0.81	L	1.25	M	1.77	M	-	—
	Anakulam	1977	0.4%	L	0.53	L	1.07	M	-	—
	Mullachal	1978	0.01	L	0.07	L	0.07	L	—	—
E. globulus	Silent valley	1978	0.0	Nil	0.0	Nil	0.0	Nil	--	

[a] Disease Severity Index; [b] Disease Severity Rating; ab, Disease observed in low incidence as branch cankers, but not recorded; br, Branch cankers only; —, Observations not recorded,

possibly infected the main stem or branches through the lenticels. Tissues of the inner bark, including cambium were killed and showed prominent browning. The infected area became depressed which during the dry period developed vertical splitting on the bark; no oozing of kino was noticed from the cankers. The apical shoot above the canker died when the stem was completely girdled. Numerous epicormic shoots developed from the healthy stem just below the canker. The shoots also got infected and killed following wilting and drying up. One of these shoots usually survived and became a leader, which did not escape the infection in the following season. Thus the infected trees, which appeared bushy due to repeated infections of pink disease became frail and weak. The yield and productivity of plantation was reduced considerably as the trees showed negative growth. Infection of older trees (3- to 4-year-old) usually resulted in localized cankers and trees were normally not girdled.

Etiology

Corticium salmonicolor Berk & Br.

C. salmonicolor produced four stages viz. cob web, pustule, pink encrustation (perfect state) and necator on eucalypts (Figs. 43a-f and 44 a-f).

Nomenclature: *C. salmonicolor* was first described by Berkeley and Broome (1875) from Ceylon (Sri Lanka), though according to Petch (1911). Thwaites was the first worker to observe pink disease in Ceylon about 1873. Then the causal organism was referred to as *C javanicum* Zimm. (Bernard, 1908; Petch, 1911). In 1909 Gallagher reported that *C. zimmermani* Sacc, & Syd. was associated with a similar disease of rubber trees. Cook (1913) identified the fungus causing pink disease of cacao as *C. lilacinofuscum.* Petch (1921) pointed out that this was identical fungus to that of *C. salmonicolor* described by Berkeley and Broom in 1875. Massee in (1898) had identified the pink disease fungus of coffee as *Necator decretus.* Later Rant (1911) reported that *N. decretus* was only a conidial stage of *C. salmonicolor.*

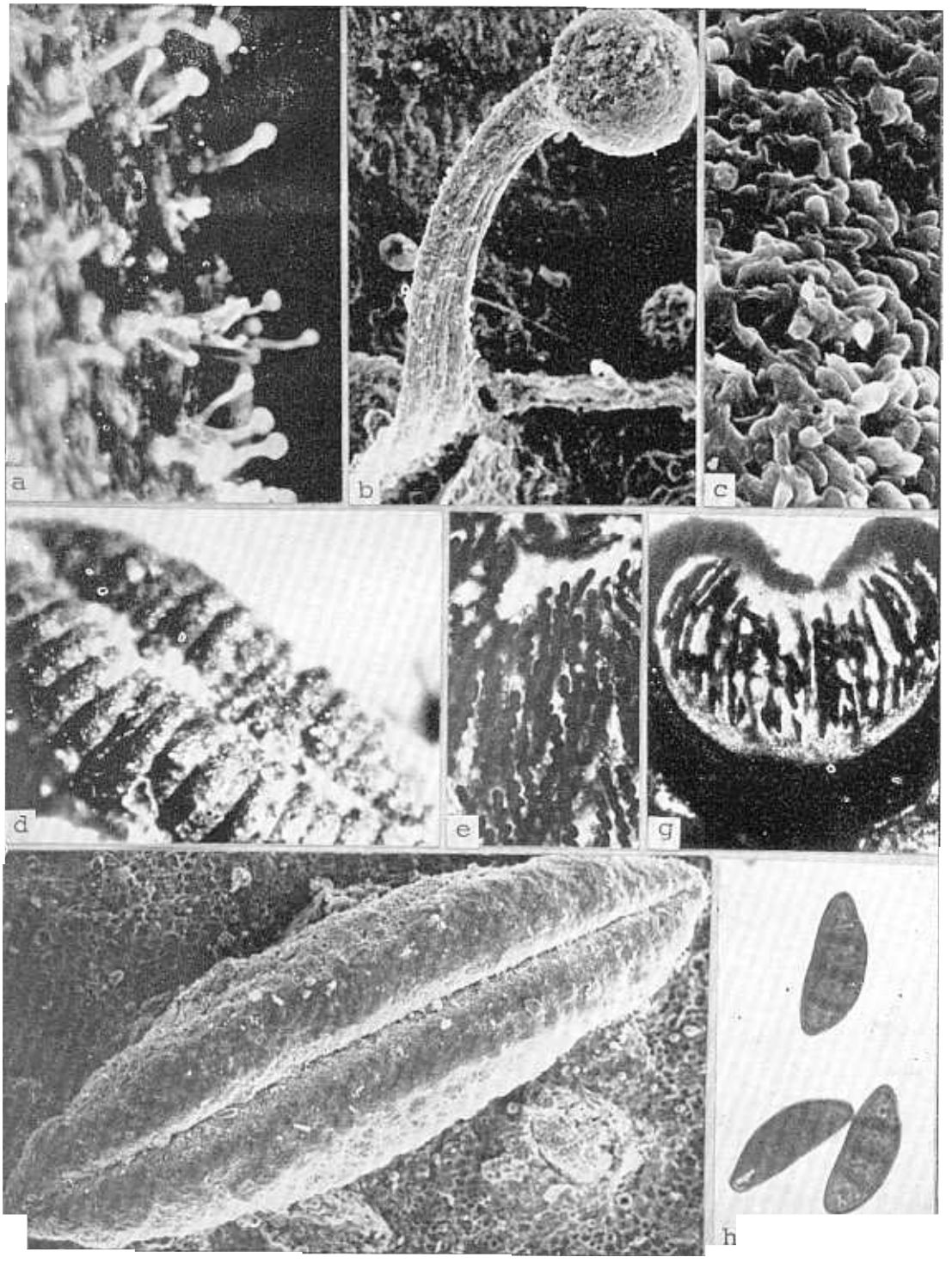
Screening of eucalypt provenances for resistance to pink disease - toxin bio-assay

As soon as the cob web stage was developed on the stem the tissues of the inner bark (phloem and cambium) became brown and got killed outright due to infection. Latcr, epicormic shoots, developed in response to this infection just below the canker region, were also killed by the rapidly spreading infection downwards. These young epicormic shoots were wilted following the development of browning to certain distance, though none of the stages of the pathogen were present in the vicinity. This observation led to the view of involvement of some chemical substance, possibly a toxin, in causing browning of the living tissues and consequently bringing about wilting of shoots.

To confirm this view, bark (outer and inner) from freshly affected stem bearing cobweb and pustule stages and from healthy stem was peeled off from the same height and diameter of stem of 2-year-old *E. tereticornis.* The bark samples were stored in a refrigerator overnight and 250 g of each bark was crushed and extracted separately in 250 ml of double distilled sterile water in a blender. The extracts were sieved through a chease cloth and centrifuged at 15,000 r.p.m. for 30 minutes. The clear supernatents, light yellowish brown in the case of diseased bark and light dull greenish of healthy bark, were filter sterilized using Gelman millipore filter (size 0.20μ m). Ten millilitre of this filter sterilized clear extract was transferred to 10 sterile culture tubes each, while in another set-up the extract was diluted (1:10) with sterile water. In each tube a surface sterilized cut shoot (excised under water) of 6-month-old *E. tereticornis* seedling, bearing three pairs of leaves was placed with cut end immersed in the liquid. All the set-ups were placed on a laboratory bench where temperature varied between 24 and 28°C. The following day all the shoots in both dilutions of the extracts of infected bark wilted while those in healthy bark extract remained unaffected. The stem of the wilted shoots had characteristic browning. These results confirmed our field observations about the possible involvement of some chemical toxic to shoots which brought about wilting. Hence, a detailed investigation was undertaken on the *in vitro* production of the toxic chemical in liquid culture and testing its efficacy in discerning level of resistance/susceptibility of various provenances of eucalypts. The details of the experiments and results obtained are described below.

Three liquid culture media viz. Richard's synthetic medium, Czepek's dox broth and Czapek's solution substituted with 3 per cent pectin (Varma and Munjal, 1980) were attempted for raising *C. salmonicolor.* Czapek's dox broth supported the best growth as compared to Richard's medium; no growth occurred on modified Czapek's solution. During the trials it was also observed that if the inoculated disc was submerged in the medium the culture grew very slowly as compared to when it floated.

Two cultures of *C. salmonicolor,* isolated from *E. tereticornis* (CS_1) and *E. grandis* (CS_1) were utilized for the production of the 'toxin'. Ten 1 l flasks, each containing 100 ml Czapek's dox broth, were inoculated with five discs, 8 mm diam, punched from 7-day-old cultures of *C. salmonicolor* on PDA. The flasks were incubated at 25 ± 2°C. One-month-old cultures were harvested, shredded in a blender and centrifuged at 15,000 r.p.m. in a Sorvall refrigerated centrifuge. The supernatant was collected and filter sterilized using Gelman millipore filters (pore size 0.20 μ m). The cell free filtrate thus collected was referred to as 'toxin'. The 'toxin' was used in bio-assay employing cut shoots from one-year-old seedlings of *Eucalyptus* belonging to 22 provenances of 11 species. The seeds of eucalypt provenances, procured from Commonwealth Scientific Industrial Research



42 tem canke of *Eucalyptus* caused by *Thyronectria* and *Hysterium*. a, Profuse growth of onidal sta of *T.pseudotricha* over the canker; b, A magnified view of conidiomata in EM (880 X Note the round conidial head and long synnamatous stalk; c, SEM of onidial he to show highly branched conidiophores bearing 1-celled oval conidia; l, Ascocar of *T. pseudotricha*; e, A section through ascocarp to show long asci ontaining septate dark coloured ascorpores; f, SEM of an ascocarp of *H. angustatum* 45 X); g, A ertical section through the ascocarp to show the arrangement of asci (60 X); (400 X).

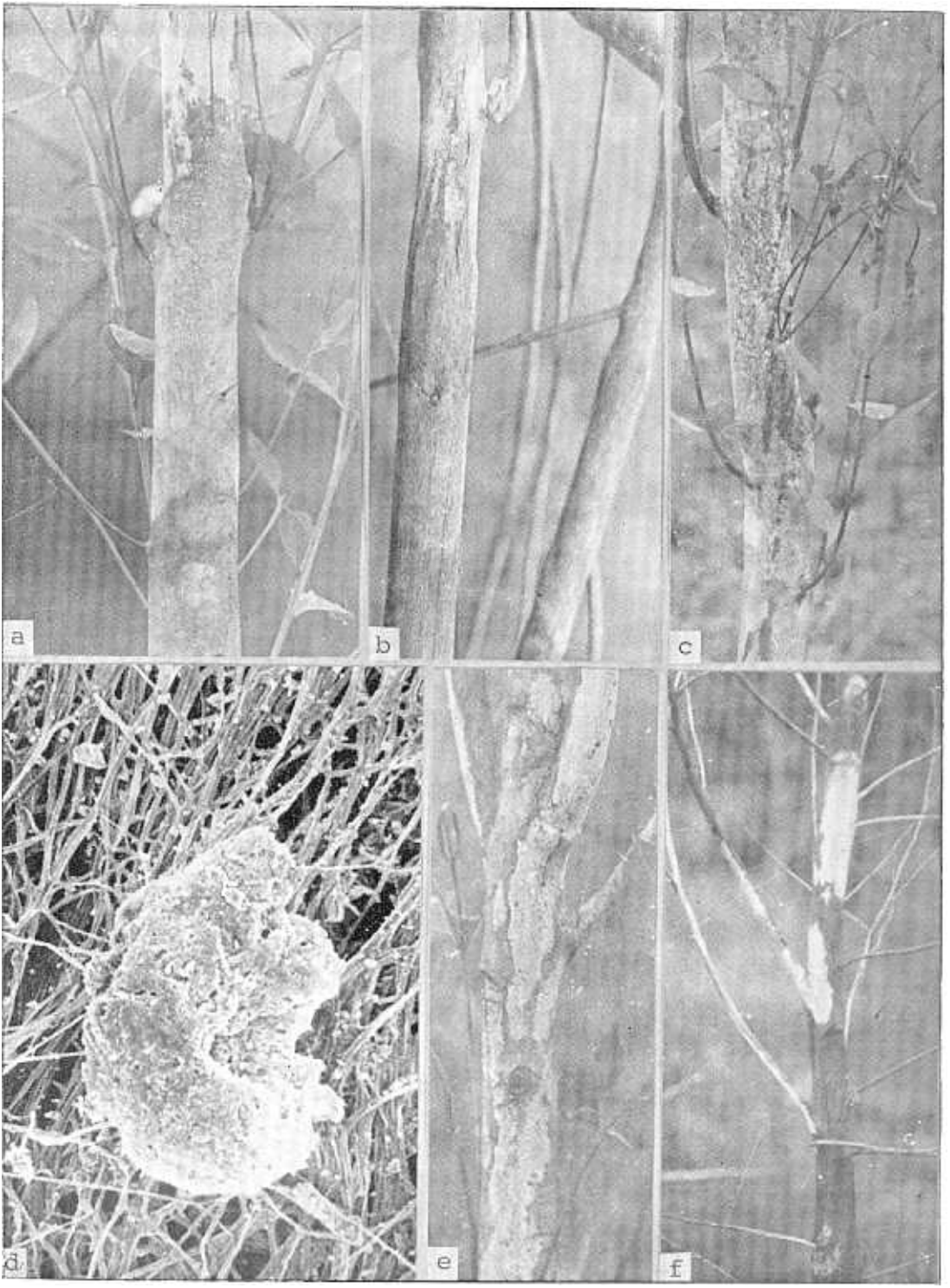


Fig. 43. Pink disease of *Eucalyptus tereticornis*. a, Stem infected with pink disease showing cob web, pustule and pink encrustation stages. Note development of epicormic shoots of which the top ones have already died; b, A branch showing a constriction at the site of the canker; c, A severely infected stem with pustule stage; d, SEM of a pustule (70 X); e, A severely infected tree covered with the pink stage; f, A dead tree with the pink stage.

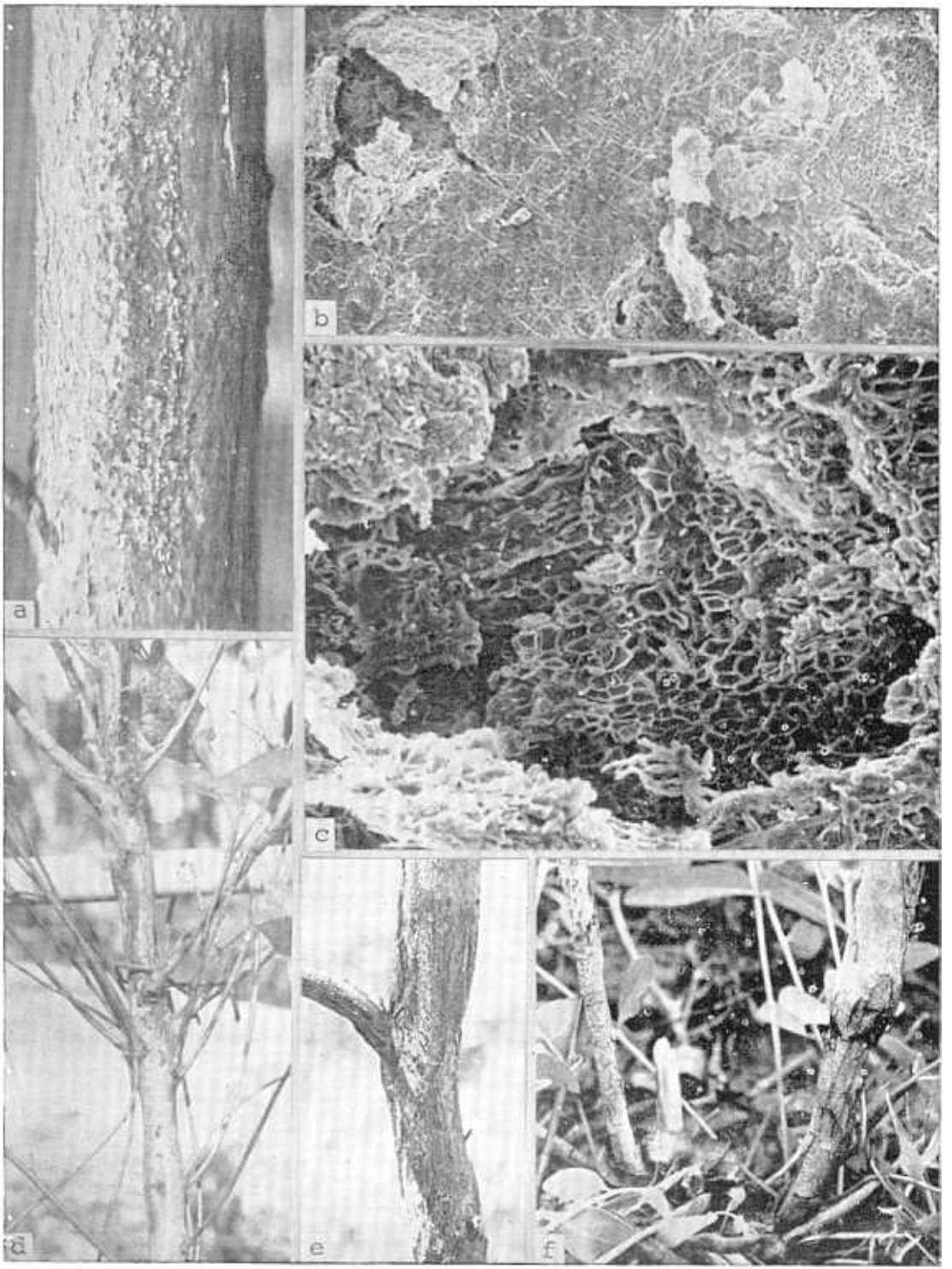


Fig. 44. Pink disease of *Eucalyptus*. a, Stem of *E. tereticornis* showing necator stage; b and c, SEM of necator stage—b (30 x), c (110 x); d, One-year-old plant of *E. grandis* infected with pink disease; e, Branch canker due to pink disease; f, Coppice shoots of *E. tereticornis* infected with pink disease.

Organization, Australia were used to raise the plants in the nursery of the Pathology Division. Young shoot tips measuring 20 cm from healthy seedlings of same height were excised with a sterile scalpel blade under water. The cut shoots were surface sterilised using 1% sodium hypochlorite and rinsed thoroughly in three changes of sterile distilled water. A measured quantity of the 'toxin' (5 ml) was transferred in 10 ml Corning tubes. The cut shoots were placed in the tubes with their basal cut end immersed in the 'toxin'. The control set contained only Czapek's dox broth. The tubes were plugged with cotton to prevent the evaporation of the toxin as well as to hold the shoots upright. Ten replicate shoots were maintained for each treatment (toxin and control). The shoots were transferred to a humidity chamber (temp. 25 ± 2°C; r. h. 100%) for 24 hrs. Later, the shoots were removed from the chamber and kept on the laboratory bench (temp. 25 - 31°C; r. h. 50-85%) and observations recorded after 18 hrs using an arbitrary objective scale based on the symptoms produced by the shoots in response to the 'toxin' as given below.

Rating index	Symptoms	Level of Susceptibility
5(4.1-5)	Twig completely wilted; leaves dried up and curled; apical shoots dried up.	Highly Susceptible (HS)
4(3.1-4)	¾ of leaves dried up, rest showing flaccidity; apical shoots showing wilting.	Susceptible (S)
3(2.1-3)	Half of the bottom leaves beginning to droop; upper leaves showing flaccidity; apical shoots drooping, wilting.	Moderately susceptible (MS)
2(1.l-2)	Leaves showing flaccidity; apical shoots drooping, wilting	Moderately resistant (MR)
I(0-1)	Leaves showing only flaccidity or unaffected	Resistant (R)

All the treated cut shoots developed different degrees of symptoms while the control ones remained healthy. Results presented in Table 42 clearly show variation in susceptibility between provenances within a species and between species of eucalypts. Browning of leaf veins was observed in provenances of ***Eucalyptus resinifera*** (13166, 13318), ***E. pellita*** (11947) and ***E. grandis*** (12409), browning of leaf midrib and veins in ***E. grandis*** (13023, 12970, Local), ***E. tereticornis*** (13398. 12744, 13277, Local), ***E. brassiana*** (13397, 13415) and ***E. tessellaris*** (12967). However, statistical analysis showed no correlation between extent of browning and susceptibility of provenances to 'toxin'.

Table 42. Susceptibility rating of various provenances of *Eucalyptus* to toxins of two isolates (CS_1, CS_2) of *C. salmonicolor*

SI. No.	*Eucalyptus* species	Seed-lot No. ex CSIRO Australia	Rating and level of susceptibility (Mean of 10 observations)	
			CS_1	CSa
1.	*E. brassiana*	13397	3.3(S)	1.7(MR)
2.	*E. brassiana*	13415	2.2(MS)	0.7(R)
3.	*E. camaldulensis*	12101	4.9(HS)	3.3(S)
4.	*E. camaldulensis*	12964	4 9(HS)	1.6(MR)
5.	*E. deglupta*	12976	3 5(S)	1.2(MR)
6.	*E. grandis*	12409	3.4(S)	2.6(MS)
7.	*E. grandis*	12970	4.9(HS)	2.3(MS)
8.	*E. grandis*	13020	3.9(S)	3.1(S)
9.	*E. grandis*	13023	4.2(HS)	3.2(S)
10.	*E. grandis*	Local	3.0(MS)	3.9(S)
11.	*E. rnicrocorys*	12795	4.9(HS)	4.0(S)
12.	*E. pellita*	11947	3.3(S)	3.8(S)
13.	*E. pellita*	12013	3.4(S)	2.2(MS)
14.	*E. resinifera*	13166	4.7(HS)	4.4(HS)
15.	*E. resinifera*	13318	1.8(MR)	3.6(S)
16.	*E. saligna*	13027	4 3(HS)	2. 9(MS)
17.	*E. tereticornis*	12944	4.9(HS)	2. 4 (MS)
18.	*E. tereticornis*	13277	3 9(S)	2 · 1(MS)
19.	*E. tereticornis*	13398	4.8(HS)	2.4(MS)
20.	*E. tereticornis*	Local	5.0(HS)	4.2(HS)
21.	*E. tessellaris*	12967	3.2(S)	2.8(MS)
22.	*E. urophylla*	12895	4.5(HS)	3.7(S)
23.	*E. urophylla*	12896	4.7(HS)	2 9(MS)

The bioassay gives positive results indicating its usefulness in discerning susceptibility of various provenances to pink disease organism, ***C. salmonicolor.*** The data show that even the provenances within a species vary in their level of susceptibility to CS_1 and CS_2 'toxins'. These differences have been found to be statistically significant in analysis of variance (Table 43) (Figs. 45 a-f). For example, of the four provenances of ***E. tereticornis*** tested against CS_2 'toxin' three (12944, 13277, 13398) gave moderately susceptible (MS) reaction while the fourth (Local) highly susceptible (HS). This trend is seen in other eucalypt species also. On the other hand provenances of some species such as ***E. camaldulensis*** (12101, 12964), ***E. urophylla*** (12895, 12896), and ***E. pellita*** (11947, 12013) exhibited similar susceptible reactions to CS_1 'toxin' (Table 44).

Table 43. Analysis of variance of data presented in Table 42

Source	SS	df	MSS	F ratio
Total	696.8728	439		
Replication	8.0091	9	0.8899	1.7755""
Provenance (P)	222.0728	21	10.5748	21.0989*
'Toxin' (T)	139.7818	1	139.7818	278.8942*
Interaction P X T	193 9909	387	0.5012	

* Significant at 0.01%
ns Nonsignificant

The other significant point emerges from this study is that the two isolates of C. ***salmonicolor*** (CS_1 and CS_2) behave like different strains (Table 44, 45). Isolate CS_2 appears to be more aggressive than CS_2. In DMRT ranking of the 22 provenances in the case of CS_1, 5 were highly susceptible (HS), 13 susceptible (S), 2 each moderately susceptible (MS) and moderately resistant (MR), and none resistant (R), while for CS_2 there were less HS and S, and more MS provenances, the break up being HS-I, S-9, MS-11, MR-I, R-I. This is also evident in reaction of individual provenances. Where the level of susceptibility of provenances to CS_2 is generally less than CS_1 except in two provenances where it was reverse; ***E. resinifera*** (13318) and ***E. grandis*** (Local) were MR and MS to CS_2 'toxin' while S to CS_2 'toxin'. E. ***brassiana*** (13415) appeared to be the only provenance resistant to both CS_1 and CS_2 'toxins', the rating being MR and R, respectively.

The 'toxin' of two isolates of C. ***salmonicolor*** from ***E. tereticornis*** (CS_2) and E. ***grandis*** (CS_2) gave HS and S reaction on their respective hosts. In this case the

interesting observation is that CS, gives MS on ***E. grandis*** (Local) while CS_2 HS on ***E. tereticornis*** (Local). This means that both CS_1 and CS_2 are more aggressive on ***E. tereticornis*** (Local) and less on ***E. grandis*** (Local). In this way in any given situation where both the strains are present ***E. tereticornis*** (Local) will be highly susceptible to pink disease while ***E. grandis*** (Local) moderately susceptible as also has been observed under field conditions.

Effect of 'toxin' on other hosts: To test the specificity of the 'toxin' surface sterilized rooted seedlings (2-week-old) of other hosts of ***C. salmonicolor (Albizia falcataria, Anacardium occidentale)*** and non-hosts ***(Capsicum annum, Lycoperscicum esculentum)*** were transferred to culture filtrates of CS_1.

Only seedlings of ***Capsicum*** did not show any wilting while all the others wilted within 24 hours. The wilted seedlings showed characteristic browning in the tissues. The results indicate that the 'toxin' produced by CS_1 isolate is non-host specific.

Effect of 'toxin' on conidial germination of certain fungi: Germination of conidia of ***Colletotrichum gloeosporioides, Botryodiplodia theobromae*** and ***Cylindrocladium quinqueseptatum*** was studied in different dilutions (1:10, 1:5, 1:1,) of CS_1 and CS_2 'toxins' following hanging drop slide culture technique. The conidial concentration was adjusted to 10-15 conidia per microscopic field of l0x objective. Proper control was kept in sterile water. The set-ups were incubated for 24 hrs. at $25 \pm 2^{o}C$ and 25 observations recorded from three replicates.

conidial inhibition of any of the test fungi was observed. Instead in some cases e. g., ***Colletotrichum*** the growth of the germ tubes as well as percent germination of conidia were promoted.

Field trials to control pink disease

To explore the possibilities of controlling the pink disease chemically, a one hectare plot was selected in a 2-year-old plantation of ***E. tereticornis*** at Thalakode (Kothamangalam Div.), where the incidence of the disease was>95 per cent with extensive shoot die-back. The affected trees were paint marked and were subject to two treatments of Calixin in latex, a brush on formulation (BASF India), during October 1981. In half of the plants either the main stem (with or without dead apical shoot) or branches or both were pruned 10 cm below the canker and 30 cm of the stem and branches treated with fungicide. In rest of the plants, cankers as well as an area 15 cm below and above the affected region were treated with fungicide. Observations on the performance of the treatment were recorded during September 1982.

Table 44. Susceptibility ranking of various provenances of *Eucalyptus* screened against 'toxin' of CS_1 strain of *C. salmonicolor*

Sl. No.	*Eucalyptus* species	Seed-lot No. ex CSIRO Australia	Rating and level of susceptibility
1.	*E. resinifera*	13318	1.8(MR) a*
2.	*E. brassiana*	13415	2.2(MS) a
3.	*E. grandis*	Local	3.0(MS) b
4.	*E. tessellaris*	12967	3.2(S) b
5.	*E. brassiana*	13397	3.3(S) bc
6.	*E. pellita*	11947	3.3(Sj bc
7.	*E. pellita*	12013	3.4(S) bc
8.	*E. grandis*	12409	3.4(S) bc
9.	*E. deglupta*	12976	3.5(S) bc
10.	*E. grandis*	13023	3.9(S) cd
11.	*E. tereticornis*	13277	3.9(S) cd
12.	*E. grandis*	13020	4.2(HS) de
13.	*E. saligna*	13027	4.3(HS) def
14.	*E. urophylla*	12896	4.5(HS) def
15.	*E. urophylla*	12895	4.7(HS) f
16.	*E. resinifera*	13166	4.7(HS) f
17.	*E. tereticornis*	13398	4.8(HS) f
18.	*E. camaldulensis*	12181	4.9(HS) f
19.	*E. microcorys*	12964	4.9(HS) f
20.	*E. grandis*	13970	4.9(HS) f
21.	*E. tereticornis*	12944	4.9(HS) f
22.	*E. tereticornis*	Local	5.0(HS) f

* Means with the same letter(s) are not significantly different based on Dunkans' multiple range test.

Table 45. Susceptibility ranking of various provenances of *Eucalyptus* screened against 'toxin' of CS_2 strain of *C. salmonicolor*

Sl. No.	*Eucalyptus* species	Seed-lot No. ex CSIRO Australia	Rating and level of susceptibility	
1.	*E. brassiana*	13415	0.7(R)	a*
2.	*E. deglupta*	12976	1.2(MR)	ab
3.	*E. brassiana*	13397	1.7(MR)	bc
4.	*E. tereticornis*	13277	2.1(MS)	cde
5.	*E. pellita*	12013	2.2(MS)	cde
6.	*E grandis*	12970	2.3(MS)	cdef
7.	*E. tereticornis*	13398	2.4(MS)	def
8.	*E. tereticornis*	12944	2.4(MS)	def
9.	*E. camaldu/ensis*	12964	2.6(MS)	defg
10.	*E. grandis*	12409	2. 6(MS)	defg
11.	*E. tessellaris*	12967	2.8(MS)	efgh
12.	*E. urophylla*	12896	2.9(MS)	fgh
13.	*E. saligna*	13027	2.9:MS)	fgh
14.	*E. grandis*	13020	3.1(S)	ghi
15.	*E. grandis*	13023	3.2(s)	ghij
16.	*E. camaldulensis*	12181	3.3(S)	hijk
17.	*E. resinifera*	13318	3.6(S)	ijkl
18.	*P. urophylla*	12895	3.7(S)	ijkl
19.	*E. pellita*	11947	3.8(S)	jklm
20.	*E. grandis*	Local	3.9(S)	klm
21.	*E. microcorys*	12795	4.0(S)	Im
22.	*E. tereticornis*	Local	4.2(HS)	lm
23.	*E. resinifera*	13166	4 . 4(HS)	m

* Means with the same letter(s) are not significantly different based on Dunkans' multiple range test.

In the first treatment the pruned trees produced epicormic shoots, of which one invariably became a leader. These shoots as well as other smaller shoots in 80 per cent of the treated plants were found to be affected with pink disease. In the other treatment also cent per cent trees developed fresh infection, though the treated canker/infected area got cured and the shoots did not die. The infection appeared either below or above the treated area. The results clearly indicate that though Calixin is highly effective in curing the disease, target application of Calixin is not sufficient to prevent fresh infection on other parts in the following year.

Effect of site and taungya crop (tapioca) on pink disease

The site of the plantation appeared to have a considerable impact on the incidence of pink disease. ***E tereticornis*** plantations situated in high rainfall area, in a valley surrounded by hills at low elevation (<500 m above msl) or near a permanent water source recorded a high incidence of pink disease, as all these conditions contributed to high humidity which favoured infection and spread of the disease.

Among the cultural practices, raising of tapioca as a taungya crop for the first two to three years of establishment also had considerable impact on the incidence of pink disease. Since the tapioca grows faster and forms a closed canopy even if the above described conditions were absent, it created conducive microclimatic conditions which facilitated pink disease infection. Under identical conditions a crop of either ginger, sesame or paddy as taungya had low incidence of pink disease.

Discussion

Pink disease caused by ***C. salmonicolor*** has worldwide distribution (Mordue and Gibson, 1976; Seth *et al.*, 1978). ***C. salmonicolor*** possesses a wide host range among woody plants numbering more than 141 species belonging to 104 genera (Sharples, 1936). In India it is indigenous and occurs on a wide range of horticultural as well as forest plants.

On ***Eucalyptus, C. salmonicolor*** has been recorded from tropical Africa and Central and South America (Bazan de Segura, 1969; Ferreira and Alfenas, 1977: Gibson, 1967) where it has assumed moderate local importance but has apparently not caused damage on the scale that has occurred in Southern India (Gibson and Armitage, 1979). While the fungus is known in Australia it has not been recorded **on** ***Eucalyptus.*** It would be erroneous, however, to at tibute any outstanding susceptibility to ***C. salmonicolor*** in exotic eucalypt plantings to this fact (Seth *et al.*, 1978).

High incidence of pink disease and its serious effects on plants are known generally in tropical regions with high rainfall (>2000 mm) (Hilton, 1958; Seth *et al.*,

1978; Ferreira and Alfenas, 1977). Under low rainfall conditions, ***C. salmonicolor*** forms primary cankers which soon occlude and callus over thus reducing the chances of secondary spread.

In Kerala, ***C. salmonicolor,*** which is widespread on indigenous trees in the low and medium altitude zones where conditions are favourable for infection, caused an epidemic in ***E. tereticornis*** plantations in the early 1970s. Losses were estimated at 55-95% due to severe infection in 5-to 11-year-old plantations (Seth *et al.*, 1978). During this disease survey even 4-month-old coppice shoots of ***E. tereticornis*** were found to be attacked with pink disease. Of the five plantations of ***E. tereticornis*** surveyed the incidence of the pink disease increased over the years and so the severity except at Mullachal where it remained quite low (Table 41).

There is no record of ***E. grandis*** being affected to the same extent by the pink disease, although it has been recorded sporadically on branches of older trees of ***E. grandis*** in the Pamba area (Gibson and Armitage, 1979). Until recently ***E. grandis*** plantations in Wynad district also were free from pink disease. However, a sudden outbreak of the disease in ***E. grandis*** though still of low severity is a matter of concern as this species was considered to be tolerant to pink disease (Anon.. 1982). Although the data presented here is only for two plots each at Noolpuzha and Periya, an extensive survey indicated that pink disease has already spread in many other plantations in Wynad district. This clearly shows that ***E. grandis*** is also vulnerable to ***C. salmonicolor.*** The susceptible nature of this species is further confirmed by our observations of infection of 6-and 12-month-old seedlings by pink disease at Periya (Fig. 44 d). The affected seedlings eventually died. This together with infection of one-year-old coppice shoots of ***E. tereticornis*** (Fig. 44 f) also at Periya, contradicts the earlier observations of Seth *et. al.* (1978) that pink disease in eucalypts occur only at the age of two to three years. The explanation given for them was the development of brown bark at this stage; the green bark of younger seedlings was considered to be resistant. Furthermore, the observations of the same authors that the development of epicormic shoots in ***E. grandis*** is rare, are found to be incorrect as the profuse development of epicormic shoots, similar to that of ***E. tereticornis*** was observed.

First shoot infection (branch cankers) (Fig. 44 e) of pink disease on ***E. grandis*** in Wynad was recorded only during 1980. The question of why ***Corticium*** infection of ***E. grandis*** did not occur much earlier as, besides the presence of other alternate hosts (cashew, coffee, mango, citrus, jackfruit), ***E. tereticornis,*** with moderately severe to severe infection, was already present in the area. It may be noted here that the earlier plantations raised in Wynad were those of only ***E. tereticornis.*** Felling after the first rotation started from 1980 onwards. Whether this outbreak is due to some

virulent strain of ***C. salmonicolor,*** the liberation and spread of inoculum as a result of disturbances caused by the felling of diseased ***E. tereticornis*** trees or the time taken to build up enough inoculum to cause infection of ***E. grandis*** need to be investigated in detail. Some support for the first view is available from our studies on growth' characteristics and 'toxin' production by ***C. salmonicolor*** isolates from ***E. grandis*** and ***E. tereticornis.*** The isolates from the former had hyaline colonies in contrast to the light pink colour of the latter. The 'mycotoxin' from the two isolates behaved differentially when tested against a number of provenances of ***E. grandis*** and ***E. tereticornis.*** However, ***C. salmonicolor*** is a pathogen which has shown no sign of host specialization or the production of specially virulent races during the last 80 years (Hilton, 1958).

In contrast to the Wynad situation, in Pamba, although occasional branch cankers on old trees of ***E. grandis*** were observed almost 10 years ago, so far the infection has not spread to main stems. The reason for this could possibly be an absence of primary inoculum as plantations were raised on grasslands, and potential alternate hosts, and the unfavourably cooler climate at high elevations. In Wynad, where the eucalypts were raised after the clear. felling of the natural forests, the primary inoculum, alternate hosts and favourable climatic conditions were already present. All of these contributed to the spread of pink disease in ***E. tereticornis*** and later to ***E. grandis*** plantations. The rate at which the pink disease infection of ***E. grandis*** is spreading in Wynad means there is an urgent need for the selection of appropriate provenancels of this species with resistance to this disease. For this provenance trials have to be conducted in various geographical areas after preliminary laboratory screening, before it is too late as happened in the case of ***E. tereticornis.***

Toxin bioassay: Though extensive work has been done on pink disease of ***Hevea brasiliensis*** Muell. Arg. (Pinching, 1973; Weir, 1976; Radhakrishnan Pillai and George, 1981) and ***Albizia falcataria*** (L.) Fosberg. (Agnihothrudu, 1962; Bakshi *et al.,* 1972; Eusebio *et al.,* 1979) not much attention has been paid on screening various cultivars to ***C. salmonicolor*** under laboratory conditions. Prior to the field trials, as a first step to eliminate relatively susceptible provenances/species of eucalypts to pink disease, a simple, rapid and efficient method was standardized based on the production of a 'toxin' by the pathogen.

Plant pathogens have long been considered to produce toxic substances that play a role in pathogenesis. Toxins change the properties of ceIlular membranes in susceptible plants and this makes possible the colonization of tissue by the toxin producing fungus. These toxins may be categorised into two: host specific and lion-host speoific (Scheffer, 1976; Rudolph, 1976; Yoder, 1981). Toxins

involved with a particular disease provide an efficient means to screen for resistance, in terms of time, space and labour. Behnke (1979, 1980) obtained reproducible results in screening plants regenerated from callus resistant to culture filtrates of ***Phytophthora infesfuns.*** However, the method of using crude culture filtrates in such screening of resistance has not been advised by Yoder (1981). Scheffer and Briggs (1981) have also pointed out that culture fluids of almost any micro-organism to cut-shoots are toxic and that such evidence can be misleading. Nevertheless, reports of such work still appear in the literature.

Regarding the cut shoot method, Yoder (1981) has stated that an inherent difficulty with the use of cut shoot assay is that the concentration of toxin to which living cells are exposed never be determined because cuttings may or may not concentrate solutions after taking them into their transpiration stream. Furthermore, a toxin solution administered to cuttings at low concentration may actually accumulate to high concentration before toxicity occurs; potency of the toxin also cannot be determined by cut-shoot assay. Even though subjected to severe criticism, the cut shoot method for toxin bio-assay, which is used in this study, still remains to be only simple method for basic bioassays (Scheffer, 1976; Yoder, 1973, 1981).

One of the criticism of using culture filtrate is that many substances, especially high molecular weight carbohydrates may also cause wilt by obstructing the xylem of cuttings and this can be misleading. In our experiment, however, only the phloem and cambium were affected shown by prominent browning of these tissues and no obstruction of xylem vessels was observed. Similar browning observed in cut shoots with crude bark extract and in epicormic shoots under field conditions, rules out the cause of wilting of cut shoots of eucalypts due to blocking of vessels. Another indication that the wilting of cut shoots was not due to obstruction of vessels, was the browning of stem which in certain cases extended even upto leaf veins. If the vessels got obstructed the culture filtrate will not go that far and the shoots will be wilted but that was not the case. The extent of browning in shoots was not found to be significant in relation to susceptibility rating of a provenance.

Bioassays are inherently variable because biological systems are complex (Roberts and Boyce, 1972). Variability due to treatment effect can sometimes be distinguished from that due to experimental error in statistical analysis (Hewitt, 1977). In our results, analysis of variance clearly showed no differences in replicates indicating that there was no experimental error. This was possibly adhieved by minimizing the variability factors in cut shoot bioassay method since (i) seedling age and height from which cut shoots of equal lengths were taken were constant, equal amount of culture filtrate was provided and (iii) the environmental conditions provided during the experiment were also constant. One draw back was that equal amount of

culture filtrate ('toxin') was used for all the species, which differed considerably in rate of transpiration due to varying size of leaves. The cut shoot method employing culture filtrate gave reproducible results as similar susceptibility rating was obtained for a particular eucalypt provenance. This clearly shows the usefulness of the toxin bioassay method in assessing the relative susceptibility of various eucalypt provenaces to pink disease.

The 'toxin' of ***C. salmonicolor*** is possibly non-host specific as even the tomato seedlings developed browning and wilted; only chilli seedlings did not wilt. Non-host specific nature of the 'toxin' of ***C. salmonicolor*** confirms the views of Mitchell (1984) that pathogens producing non-host specific toxins generally have a varying number of susceptible host plants. It is perhaps significant that non-host specific toxins have apparently a high biochemical specificity yet little specificity between plant species, presumably because the targets are enzymes that are common to most plants. This way the pathogen gains full benefit from production of such toxins in the broadening of its potential range of hosts (Mitchell, 1984).

The results of screening of different provenances indicate that none of them were resistant to both the isolates of ***C. salmonicolor.*** ***E. brassiana*** (13415) was the only provenance which gave resistant reaction to CS_2 and moderately resistant to CS_1 'toxin'. Provenances within a species showed variation in susceptibility The local ***E. grandis*** and ***E. tereticornis*** gave susceptible to moderately susceptible reactions to two 'toxins' as observed under field conditions. The other interesting point which emerges from the results that CS_1 'toxin' is more 'virulent than CS_2 thus behaving differently in their reaction on various provenances. This possibly indicates that CS_1 and CS_2 are genetically different strains.

This study thus makes possible the preliminary selection of comparatively resistant varieties of eucalypts for planting and also for breeding for disease resistance. However, to establish the reliability of this method isolation and characterization of the toxin need to be carried out.

Control of pink disease: The control of pink disease by fungicides has been an established practice in rubber plantations in India and South-East Asia (Hilton, 1958). Bordeaux mixture in paste or spray form or systemic fungicides such as Calixin have been used successfully by manual application. Laboratory screening of fungicides to control ***C. salmonicolor*** followed by small-scale field trials have shown that copper fungicides and Calixin are equally effective in controlling pink disease of eucalypts. Observations made by Seth *et al.* (1978) that Calixin was effective in preventing pink disease infection was based on artificial inoculation trials lasting for a few months only. However, a pilot experiment conducted by us clearly indicates that though

the fungicide was highly effective, to check the recurrence of the disease, chemical control is not practical and not economically feasible. Some of the constraints faced for the control of a stem disease, such as pink disease in extensive eucalypt plantations have been highlighted by Gibson and Armitage (1979).

The only possible long-term solution for controlling the pink disease appears to be through species selection and tree improvement. After the provenance or the species of eucalypts are screened in laboratory following the cut shoot bio-assay, they should be field tested for their disease resistance besides growth character, coppicing, etc., in a multilocational trials. Similarly, progenies from trees, which have escaped infection in high disease incidence areas, may be raised and screened for disease resistance. Tree improvement through hybridization, though time consuming, may also be attempted for long-term disease control.

12. CYTOSPORA STEM CANKER

Occurrence

The disease, usually causing branch cankers, was recorded in several plantations of ***E. tereticornis*** (Pezhad 2.33 per cent; Anakulam, 2.4 per cent; Mullachal, 6.71 per cent). Severe infection of main stem was observed in l-year-old second rotation coppice shoots of ***E. tereticornis*** at Begur during September 1984. About 75 per cent of the sboots were found to be infected, of which ca. 45 per cent had died. Incidence of branch canker resulting in die-back was recorded to be high (ca. 30 per cent) at Meenmutti in a ***E. grandis*** plantation; elsewhere the incidence was low.

Symptoms

In the case of branch and twig cankers infection occurred on any part of the stem. The tissues of the infected region showed pronounced browning and leaves wilted and defoliated. Numerous, black conidiomata (pycnidia) developed scattered over the entire infected region (Fig. 46 a-d). Complete girdling of the phloem and cambium usually resulted in death of the branches.

Infection on the main stem of coppice shoots was initially observed at the base near the stump and later it gradually spread towards the apex. Girdling due to the canker at the base resulted in wilting of leaves and death of shoot. Numerous conidiomata were developed on the dead stem. In severe cases the infection even spread to roots killing the stump. Generally all the shoots of a stump got infected and died.

Etiology

Cytospora eucalypticola van der Westhuizen (IMI 284046) causing main stem canker and ***C. eucalypti*** sp. nov. (IMI 261564) causing twig or branch cankers.

Discussion

Cytospora eucalypticola and ***C. eucalypti*** are new pathogen records for ***Eucalyptus*** in India. ***C. australis*** Speg., ***C. eucalyptina*** Speg. and ***C. eucalypticola*** have been reported to cause cankers on ***E. ficifolia, E. globulus*** and ***E. marginata*** in Australia (Gibson, 1975; Davison and Tay, 1983). ***C. australis*** has also been found on E. globulus in Portugal (Azevedo, 1971). ***C. eucalyptina*** and ***C. australis*** form cankers on twigs and branches from which they extend to main stem. Trees of all ages are affected but young trees may be killed outright (Kristic, 1964). Among the three species described earlier, ***C. eucalypticola*** (recorded from Begur in Kerala), reported first from West Africa (van der Westhuizen, 1965 a, b) is considered to be the most damaging one (Gibson, 1975). Species other than E. ***saligna*** have been found to be susceptible to ***C. eucalypticola,*** and while trees upto 3-year-old were only attacked in South Africa, older trees have been infected in Central and East Africa. The disease appears to be favoured by stress conditions for the host. It is now known to occur in Uganda, Malawi, Kenya, Pakistan, Burma and Western Australia (Gibson, 1975). Another species ***C. chrysosperma*** (Pers.) Fr. has been reported to cause branch cankers in Chili (Garces, 1964) on ***Eucalyptus*** spp.

Cytospora, the anamorph of Valsa-Fr., sometimes occur with their ascomatal state, such as ***Valsa eucalypti*** and ***C. eucalypti*** at Meenmutti, although they are more frequently found alone.

Detailed investigation on the epidemiology, especially the infection process and control measures are needed so that appropriate steps may be taken, should the disease spread in epidemic form.

13. NATTRASSA STEM CANKER

Occurrence

The disease was recorded in 2- to 3-year-old ***E. tereticornis*** plantations at Potta, Elnad (Trichur Div.) and Tamarassery (Kozhikode Div.). The incidence of the disease was < 1 per cent.

Symptoms

Generally the infection appeared on the stem near the ground and later spread upwards covering a large part of the stem. The infected area got differentiated in a depression, which later turned into a canker. Occasionally, the roots also got infected and complete girdling of stem resulted in death of plants; the leaves gradually wilted and defoliated. On the canker numerous minute fructifications, pycnidia, developed arranged in vertical broken lines (Figs. 47 a-c). The tissue of the affected-area showed greyish-black discolouration in which dark brown, septate, thick-walled, inter and intra-cellular mycelia were found in abundance.

Etiology

Nattrassa toruloidea (Nattrass) Dyko & Sutton (= ***Hendersonula toruloidea*** Nattrass) (IMI 261565) and its conidial state ***Scytalidium*** (IMI 267017).

Discussion

Sommer (1955) regarded ***N. toruloidea*** as a weak parasite causing branch wilt of walnut in California following sun scorch. It is known to kill the cambial region of wide range of hosts, often following injury. It has been reported from Ghana on ***Melia azadiracta,*** in Pakistan on ***Morus*** alba and in the U. S. A. on ***Fuglans regia*** and ***Populus*** spp. (Browne, 1968) and from Iraq on almonds, peach, plum, ***Casuarina*** sp., ***Eucalyptus*** sp. and persimmon (Al-Zarari *et al.,* 1979). It has been reported that usually the initial infection occurs in the crown and later it spreads downwards. However, in ***Eucalyptus*** the infection was always recorded on the stem near the ground.

This is the first record of ***N. toruloidea*** on ***E. tereticornis.***

14. STEM DECAY

Occurrence

Decay of standing trees in a 6-year-old trial plantation of ***E. citriodora*** was observed at Kottappara. The incidence of the disease was very low as only a few trees were found to be affected. Considering the extent of decay in trees the infection appeared to be at least 3-4 years old. The fructifications of the decay fungus were collected during September.

Etiology and symptoms

(i) ***Microporus xanthopus*** (Fries) O. Kuntze

Large number of fructifications were observed on a partially dead trunk on one side from base to 60 to 75 cm above ground (Fig. 47 e). The tissues in the affected area became soft and pulpy and roots also got partially decayed.

(ii) ***Lentinus squarrosulus*** Mont.

The stem near the ground was affected with decay (Fig. 47 d) where numerous fructifications developed in groups. Extensive fan shaped mycelium was observed on the decayed and the surrounding healthy tissues.

Discussion

This is the first occurrence of ***M. xanthopus*** and ***L. squarrosulus*** on ***E. citriodora,*** Decay in living trees is usually facilitated by wound or injury; fire injury plays a major role in the establishment of decay fungi. The pathogen enters initially through the injured dead tissue and later when established attacks nearby healthy tissues. Since the incidence of stem decay was very low the disease appears to be unimportant.

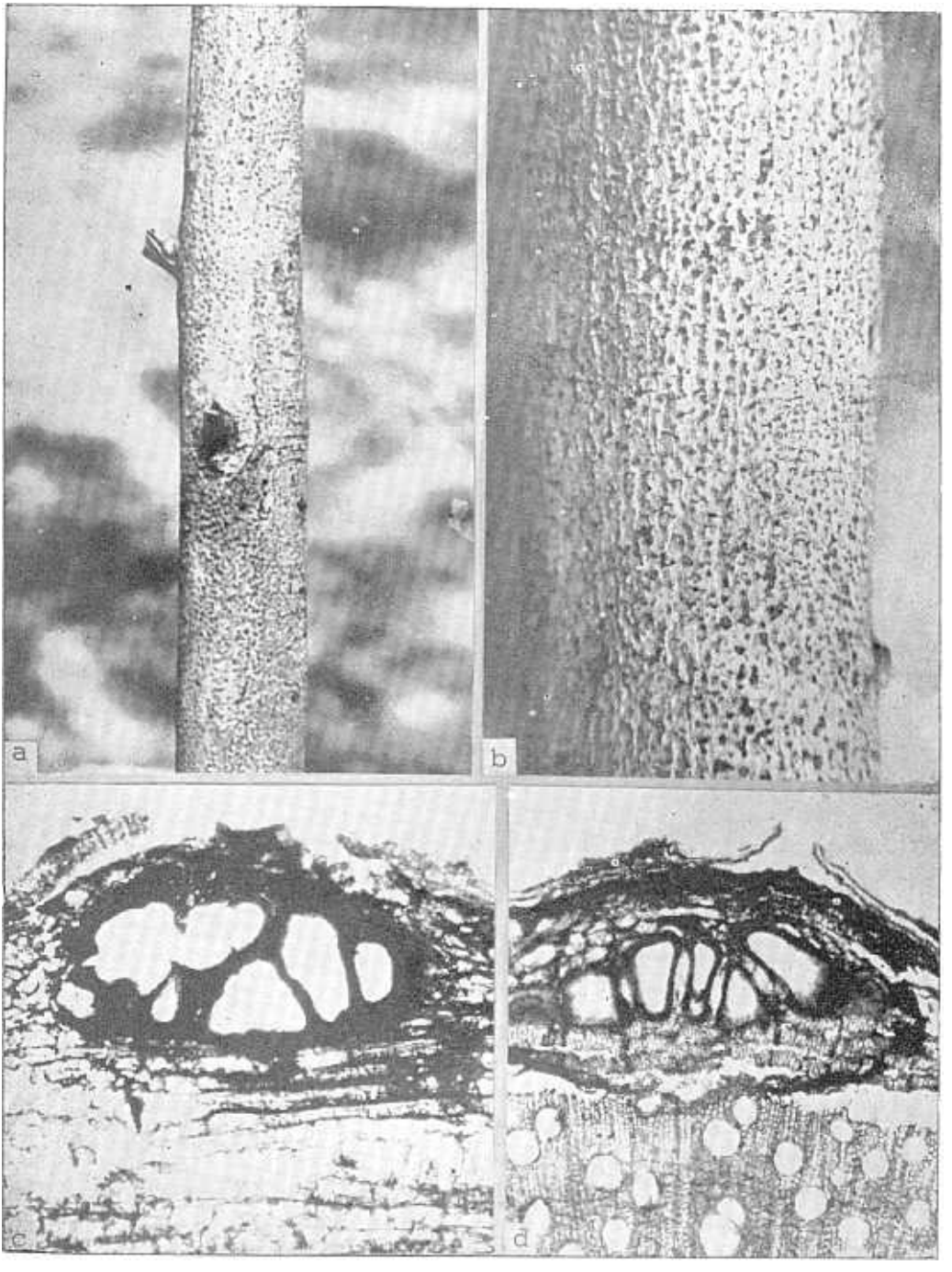


Fig. 46. Cytospora stem canker of *Eucalyptus*. a and b, A severely infected stem of *E. tereticornis* with *C. eucalypticola*; c, A vertical section through the pycnidial conidiomata of *C. cucalypticola*; d, A vertical section through pycnidial conidiomata of *C. eucalypti*.

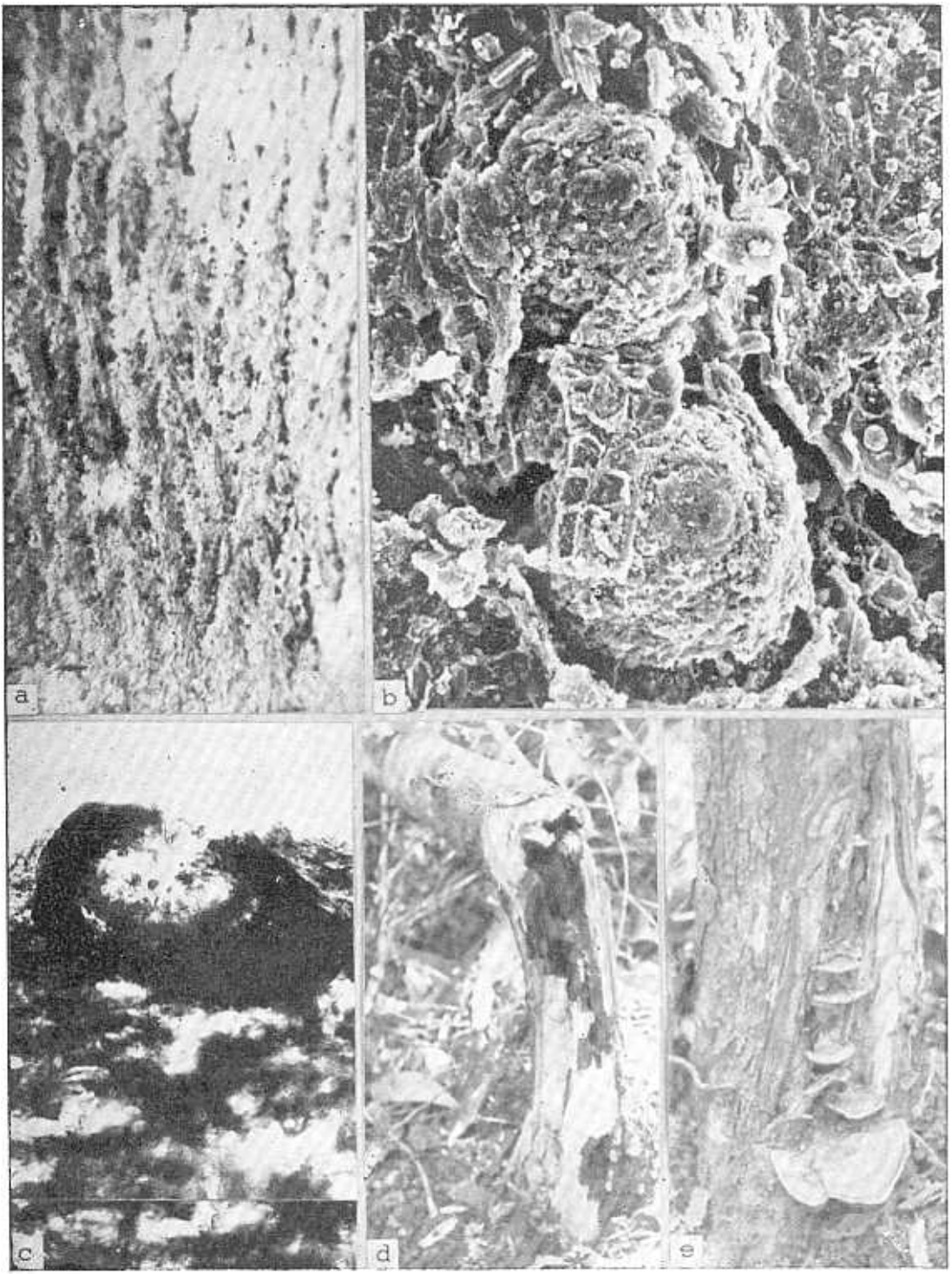


Fig. 47. Stem diseases of *Eucalyptus*. a, Stem of *E. tereticornis* infected with *Nattrassa toruloidea*. Note vertical rows of perithecia in the bark; b, SEM of pycnidia partially immersed in the bark (210 X); c, A vertical section through pycnidium; d, Stem of *E. citriodora* decayed due to *Lentinus squarrosulus*; e, Fructifications of *Microporus xanthopus* on the stem of *E. citriodora*.

15. CYLINDROCLADIUM SHOOT INFECTION

Occurrence and severity

Shoot infection, affecting leaves and stem, of ***Eucalyptus*** was widespread during and immediately after the monsoon (June - September), irrespective of age of plants and species. Stem infection, which was more common in young plants (1-to 2-year-old) and coppice shoots, was observed only during the monsoon (Figs. 3a,b), while those of leaves was prevalent round the year, especially in humid tracts and high elevation areas such as Wynad plateau, Munnar, Idukki and Pamba.

Severity of ***Cylindrocladium*** infection generally depended upon the microclimatic conditions, especially rainfall and age of plants. Medium to severe infection was recorded in younger plantations (I-to 3-year-old) situated in high rainfall areas (Fig. 3) irrespective of species of ***Eucalyptus,*** whereas in older plantations (4- to 8-year-old) the severity was usually low, and occasionally medium due to high rainfall (Table 46) (Fig. 3). Severe infection of leaves, which caused blight, resulted in extensive premature defoliation as observed in ***E. tereticornis*** at Thalakode (Kothamangalam Div.), Kottappara (coppice shoots) (Malayattoor Div.), Vazhachal (Vazhachal Div.). Tamarassery (Kozhikode Div.), in ***E. grandis*** at Chandanathode, Periya (Wynad Div.), Thariode (coppice shoots) (Kozhikode Div.), Vazhachal (Vazhachal Div.), Idukki (KFDC, Idukki), Pamba (KFDC, Pamba) during the peak of monsoon when the rainfall was heavy and incessant for many days. Extensive die-back of young shoots due to stem infection was recorded in ***E. tereticornis*** at Chandanathode (Wynad Div.) and Tamarassery (Kozhikode Div.) and in ***E. grandis*** at Idukki (KFDC, Idukki), Pamba and Pachakanam (KFDC, Pamba).

Symptoms

Stem infection: The stem infection, observed in coppice shoots and branches of young trees, appeared somewhere on the branch and caused a canker characterised by a dull brown depression on the stem. During high humid periods ***Cylindrocladium*** was seen to produce profuse mycelium and conidial mass. The portion of the branch above the canker was killed outright when completely girdled. During the rainy season; numerous fructifications (teleomorphs) developed on the dead stem. Stem infection coupled with severe leaf blight caused by ***Cylindrocladium*** resulted in die-back of shoots. Striking damage due to ***Cylindrocladium*** was recorded in a small area (0.4 ha) of a failed plantation of ***E. tereticornis*** at Chandanathode, Wynad in 1979. Severe infection not only caused premature defoliation but also extensive die-back of young shoots. New epicormic shoots, developed as a result of die-back, also got infected in the subsequent monsoon. Thus, repeated attacks of ***Cylindrocladium*** had left 4-year-old trees with a frail stem and attenuated appearance (Figs. 48 a,b).

Table 46. Incidence and severity of shoot infection by ***Cylindrocladium*** in different eucalypt plantations surveyed during 1980-1982

Sl. No.	Locality	1980			1981			1982		
		% incidence	DSI[a]	DSR[b]	% incidence	DSI	DSR	% incidence	DSI	DSR
	E.grandis									
1.	Thrissillery	68.35	0.92	L	96.05	1.23	M	41.53	0.48	L
2.	Mavinhalla	22.41	0.26	L	50.90	0.58	L	26.55	0.25	L
3.	Noolpuzha	22.29	0.27	L	63.32	0.66	L	45.20	0.32	L
4.	Pappathishola	0	0.0	Nil	58.21	0.67	L	—	—	—
5.	Mullachal	38.04	0.38	L	61.88		L	3.35	0.03	L
	E.tereticornis									
6.	Varavoor	61 86	0.74	L	92.80	0.96		73 44	0.51	L
	Kottappara	0	0.0	Nil	38.83			—		—
8.	Pezhad	40.14	0.47	L	94.26			53.74	0.62	L
9.	Mullachal	64.00	0.68	L	33 33	0.38	L	26.15	0.41	L
10.	Anakulam	33.60	0.47	L	65.18	0.74	L	68.75	0.65	L
	E. globulus									
11.	Silent Valley	0	0.0	Nil	38.07	0.45	L	—	0.0	Nil

aDSI, Disease Severity Index
bDSR, Disease Severity Rating
—. Observations not recorded

Leaf infection: Leaves of a11 maturities from young and old plants, epicormic and coppice shoots were found to be equally susceptible to ***Cylindrocladium*** infection. The symptom expression, mainly the colour, size and spread of the lesions varied depending upon the leaf maturity and micro- and macro-climatic conditions and eucalypt species. Symptoms of leaf infection in respect of various species of eucalypts are described separately below.

E. grandis- On young leaves the infection appeared in the form of minute greyish-black spots which coalesced to form large necrotic area. Under high humid conditions the initial spots were usually large greyish black patches, which spread

further at times to cover the entire leaf lamina. In mature leaves occasionally the infection initiated either from the leaf tip and spread downwards (Fig. 48 c) or from the margins and gradually spread towards the mid rib. During the dry period the lesions became dull pale brown. Extensive leaf infection caused blight which resulted in premature defoliation.

E. tereticornis — Initially the symptoms were similar to ***E. grandis*** but later the colour of the lesions became different. Since the foliage of this species exhibited some degree of polymorphism the symptom of leaf infection also varied greatly. The infection took place at any place on the leaf lamina, but usually at the tip or margins and produced irregular greyish black spots, which coalesced to form large necrotic areas (Fig. 48 f). These later turned light to dark brown during the dry period. Severe infection of leaves caused premature defoliation.

E. urophylla — Infection usually occurred either at the margins or the tip in the form of large greyish black lesions with regular margins (Fig. 48 e). In young leaves the lesions spread rapidly and covered a large part of lamina. During the dry period these spots turned light pale brown in colour giving a blotchy appearance to leaf.

E. torelliana — Usually infection was common at the leaf margins and tip. Initially the infection appeared as minute dark greyish-black circular spots, which became as reddish brown spots lined with dark brown margin (Fig. 48 d). These spots along the margins and at the tip coalesced to give rise large irregular necrotic areas.

E. citriodora — The symptoms on this species produced as small greyish black flecks which later turned as yellowish brown to reddish brown necrotic areas. Usually the individual lesions remained as small spots without further spread except at the tip or along the margins, where they coalesced to form large irregular reddish brown necrotic area.

E. deglupta — The infection appeared as minute purplish flecks which turned into yellowish brown spots. Generally, these spots did not spread and coalesce, and remained as individual spots.

E. globulus — The infection was confined either at the tip or along the margins of leaves as dull yellowish brown areas.

Etiology

A total of five species of ***Cylindrocladium*** were found to be associated with shoot infection of eucalypts; the symptom sproduced by them were not distinguishable.

(i) ***Calonectria quinqueseptata*** Figueiredo & Namekata and its anamorph ***Clyindrocladium quinqueseptatum*** Boedijn & Reitsma causing leaf spots and/or leaf blight of

E. citriodora, E. camaldulensis, E. deglupta, E. grandis, E. globulus, E. tereticornis and ***E. torelliana*** and die-back of E*., tereticornis* and ***E. grandis.***

(ii) ***Calonectria ilicicola*** Boedijn & Reitsma and its anamorph ***Cylindrocladium ilicicola*** (Hawley) Boedijn & Reitsma causing leaf spots and leaf blight of ***E. grandis*** and ***E. tereticornis.***

(iii) ***Calonectria theae*** Loos and its anamorph ***Cylindrocladium theae*** (Petch) Alf. &. Sob. (IMI 280734-37, 280739-41) causing leaf blight of ***E. grandis.***

(iv) ***Cylindrocladium clavatum*** Hodges & May (IMI 270185) causing leaf spot and blight of E*. grandis* and ***E. tereticornis.***

(v) ***Cylindrocladium scoparium*** Morg. causing leaf spots and blight of ***E. grandis*** and ***E. tereticornis.***

Pathogenicity

The pathogenicity of all the species was confirmed in detached leaf culture as well as seedlings (5-month-old) of ***E. grandis*** and ***E. tereticornis,*** kept in a humidity chamber maintained at > 95 per cent r. h.

Influence of tapioca cultivation on Cylindrocladium infection

Tapioca as a taungya crop in young eucalypt plantations had a considerable impact on the incidence and severity of ***Cylindrocladium*** infection. The tall variety of tapioca almost covered the eucalypt and formed a closed canopy, thus providing conducive microclimatic conditions for the manifestation of the disease. Extensive leaf blight resulting in premature defoliation and die-back of shoots occurred in such plantations. In 2- to 3-year-old plantations, the plant parts under the tapioca only suffered and the top above the canopy remained healthy. However, in severe cases the infection gradually moved upwards, thus infecting the entire foliage of trees. Defoliated branches produced new flush within two to three weeks.

Discussion

Shoot infection caused by ***Cylindrocladium*** spp. is the most serious and widespread disease of eucalypts in Kerala. A total of five species viz. ***Cylindrocladium guinqueseptatum*** and its teleomorph. ***C. ilicicola*** and its teleomorph, ***C. theae*** and its teleoinorph, ***C. clavatum*** and ***C. scoparium*** were recorded in Kerala. Earlier, Bakshi (1975) has recorded only ***C. quinqueseptatum*** and ***C, scoparium;*** the rest of the three species are new records for Kerala and India. ***C. theae*** and Its anamorph are new pathogens for eucalypt. ***C. scoparium*** has been reported as the most serious pathogen causing various diseases in Brazil (Batista, 1951; Magnani, 1964) and Goa, India (Bakshi, 1975). However, during the present survey in contrast to the reports from Brazil and Costa Rica, ***C. scoparium*** was very rarely found with the foliage infection

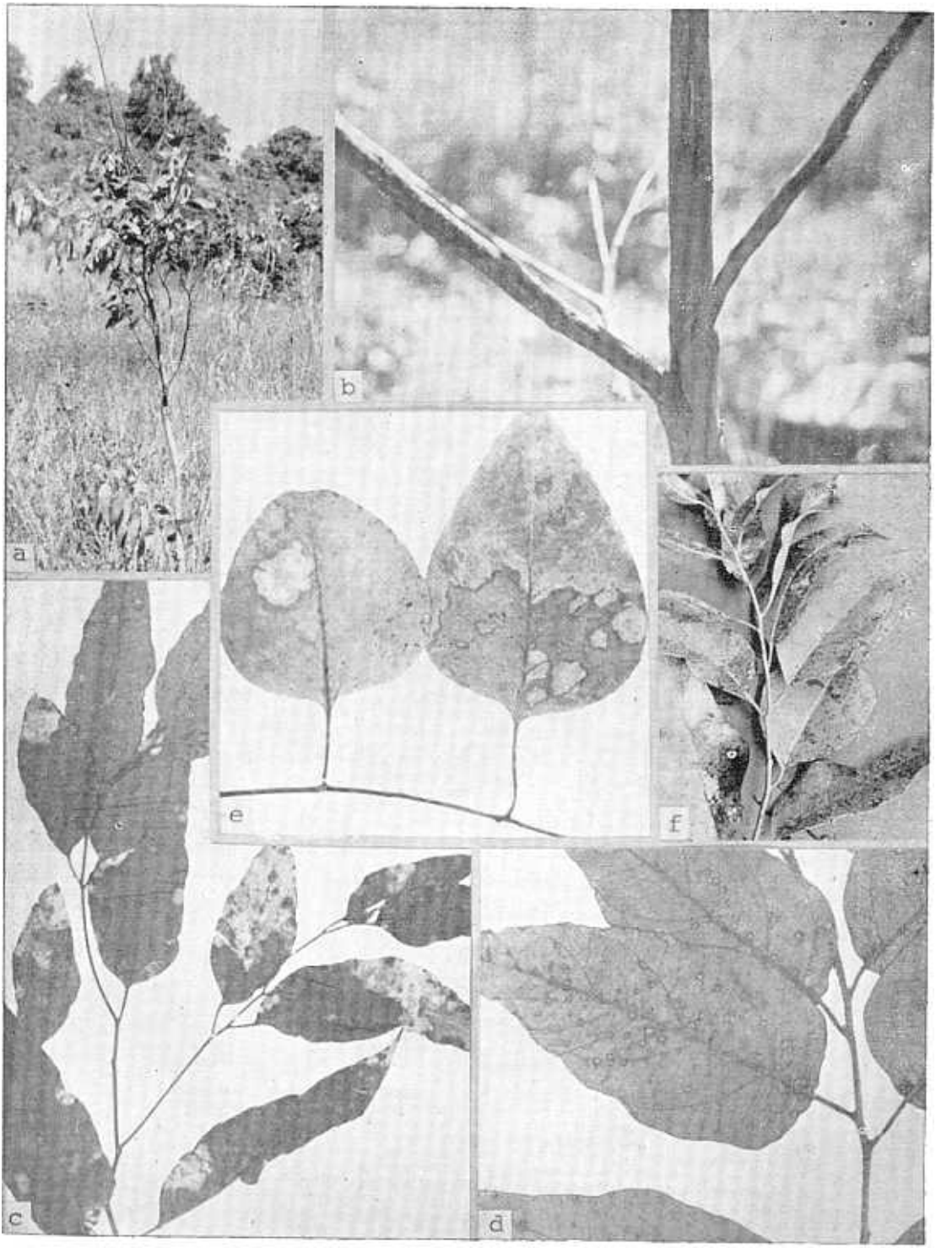
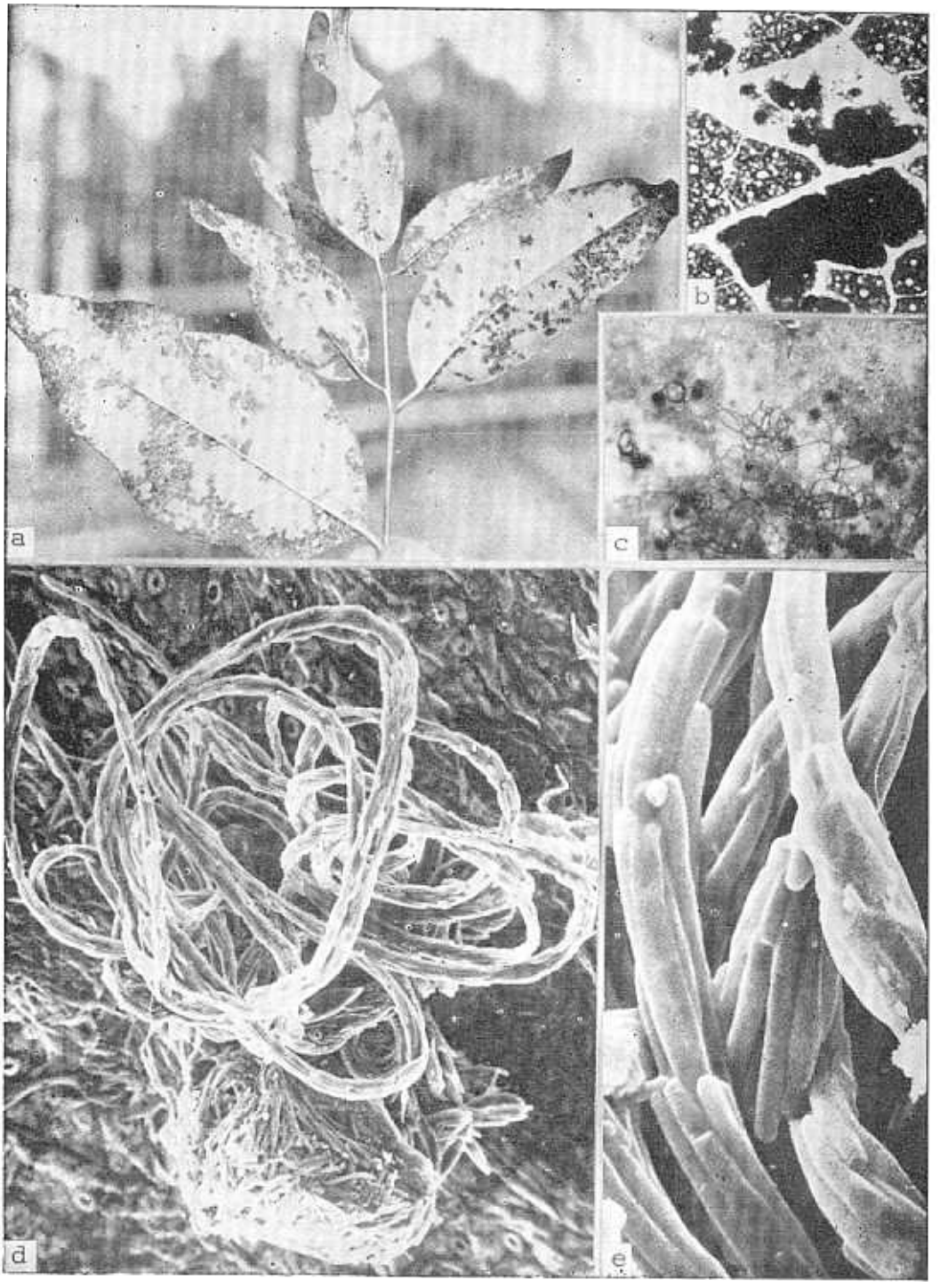


Fig. 48. Shoot diseases of *Eucalyptus* caused by *Cylindrocladium*. a, A 4-year-old plant of *E. tereticornis* at Chandanathode showing extensive die-back of shoots due to repeated infections. Some healthy looking shoots in the middle are epicormic branches; b, cankers on main stem and branches; c-f, Leaf spots of *E. grandis* (c), *E. torelliana*. (d), *E. urophylla* (e) and *E. tereticornis* (f).



leaf spots

of eucalypts. ***C. quinqueseptatum,*** the most common species occurring in plains as well as high elevations of Kerala, was recorded for the first time on ***E. camaldulensis, E. citriodora, E. globulus*** and ***E. torelliana,*** in addition to ***E. grandis*** and ***E. tereticornis.*** It was followed by ***C. theae*** and ***C. ilicicola*** which were found only in high elevations and were responsible for severe leaf blights in Wynad, Idukki and Pamba areas. ***C. ilicicola*** has earlier been reported causing die-back in Karnataka on ***E. globulus*** Linn. (Reddy, 1973), die-back (Figueiredo and Cruz, 1963) and leaf spots (Alfenas *et al.*, 1979) on various species of eucalypts in Brazil.

Severity of ***Cylindrocladium*** infection depended upon the rainfall and severe infection was observed during monsoon (June-September) (Fig. 3) as the microclimatic conditions were conducive for the infection and spread of the disease through water dispersed conidia. The infection first appeared on the foliage of lower branches and proceeded upwards indicating the soil-borne nature of the pathogen. Mortality was observed in the case of I-year-old plants and young coppice shoots of ***E. grandis*** and ***E. tereticornis*** in high rainfall areas only. In older plants, infection only resulted in premature defoliation and die-back of shoots. During the survey we recorded only low to medium infection as the observations were recorded after September when the severely infected leaves had already defoliated prematurely and only a few infected leaves remained on the plants.

Raising tall variety of tapioca was observed to be an important factor responsible for high incidence and severity of ***Cylindrocladium*** infection in 1-to 3-year-old plantations and young coppice shoots. The closed canopy of tapioca created conducive micro-climatic conditions for the manifestation of the disease.

Control of ***Cylindrocladium*** infection in plantations is not economically feasible. However, the incidence and severity of infection can be brought down to low level either by replacing tapioca with some other crop like ginger, paddy, sesame or growing only a dwarf variety of tapioca. Mortality in 1-year-old plantations due to ***Cylindrocladium*** infection can be avoided by planting only healthy seedlings of appropriate age after the onset of pre-monsoon showers or during the first week of the arrival of the monsoon. Any delay in the planting programme will facilitate ***Cylindrocladium*** infection. During the period of heavy showers seedlings will not be able to establish properly after the transplanting shock and succumb easily to ***Cylindrocladium*** infection.

16. GUIGNARDIA LEAF SPOT

Occurrence

This leaf spot disease was observed in ***E. grandis*** plantations in Wynad plateau and Munnar Div. The incidence of the disease varied from low to medium.

Symptoms

Initially the symptoms developed as minute purplish spots on the upper leaf surface. Later they enlarged and became purplish brown necrotic areas, 4-5 mm across, spherical to irregular, often lined by the veins.

Etiology

Guignardia citricarpa Kiely (IMI 287783).

Discussion

Guignardia citricarpa is of wide geographical distribution recorded on a number of hosts. It is known to cause various types of lesions in fruits and leaf spots of ***Citrus*** spp. (Kiely, 1949). It is interesting to note that the disease was recorded only from high elevation areas where ***Citrus*** spp. are grown on large-scale. ***G. citricarpa*** has earlier been reported to cause diffuse leaf necrosis of ***E. grandis*** in South Africa and Malayasia (Gibson, 1975). This is the first report of Guignardia leaf spot on ***Eucalyptus*** from India.

17. PHAEOSEPTORIA LEAF SPOT

Occurrence and severity

Phaeoseptoria leaf spot, common to both nurseries and plantations, was recorded throughout Kerala on ***E. tereticornis, E. grandis*** and ***E. globulus.*** Though the disease was prevalent in dry season (December to May) yet it was observed even during peak of monsoon period (July/August). Severe infection caused extensive premature defoliation. In nurseries raised in dry areas, container seedlings as well as seedlings left in the motherbeds were found to be seriously affected with this leaf spot.

In eucalypt plantations surveyed, medium infection was recorded in ***E. tereticornis*** at Varavoor during I980 and in ***E. globulus*** during 1980 and 1982. In rest of the plantations. the severity was low (Table 47). Generally, as the plants matured the disease severity showed a decreasing trend in most of the plantations, with the exception of ***E. tereticornis*** at Anakulam and Kottappara

Symptoms

The infection first appeared on mature leaves as purple to brownish purple amphigenous spots (Fig. 49 a) which were characteristically angular and marked by veins (Fig. 49 b), especially on ***E. tereticornis*** and ***E. grandis.*** The leaf spots gradually progressed upwards and late in the season, they were frequently noticed on younger leaves. By this time generally, all the mature leaves had defoliated prematurely

due to heavy infection. When the spots turned necrotic, minute black fruiting bodies (pycnidia), generally more on the abaxial surface, developed embedded in the leaf tissue. Pycnidia produced long greyish-black tendrils which appear as brownish-black wooly mass on both the leaf surfaces (Figs. 49 c-e). Due to rain or dew the conidia got dispersed from the tendrils and formed a black layer over the leaf surface.

Etiology

Phaeoseptoria eucalypti (Hansf.) Walker (IMI 246483).

Table 47. Severity of ***Phaeoseptoria*** leaf spot in various eucalypt plantations in Kerala surveyed during 1980-1982

Sl. No.	Locality	% incidence	DSI[a]	DSR[b]	% incidence	DSI	DSR	% incidence	DSI	DSR
	E. grandis									
1.	Thrissillery	6.15	0.12	L	7.32	0.07	L	***4.09***	0.09	L
2.	Mavinhalla	20.64	0.20	L	***0***	0.0	Nil	***0***	0.0	Nil
3.	Noolpuzha	65.20	0.85	L	3.46	0.24	L	4.25	0.03	L
4.	Pappathishola	32.65	0.40	L	66.77	0.67	L	—	—	—
5.	Mullachal	61.59	0.58	L	29.56	0.18	L	22.67	0. 11	L
	E. tereticornis									
6.	Varavoor	27.78	1.07	M	36.33	0.92	L	35.21	0.31	L
7.	Kottappara	69.11	0.95	L	37.37	0 48	L	—	—	—
8.	Pezhad	16.54	0.17	L	21.14	0.10	L	47.32	0.23	L
9.	Anakulam	4.6	0.05	L	40.28	0. 42	L	61.25	0.67	L
10.	Mullachal	41.33	0 47	L	21.33	**0.22**	**L**	18.33	0.09	L
	E.globulus									
11.	Silent valley	87.55	**1.20**	**M**	**93.08**	1.05	M	—	—	—

[a]DSI. Disease Severity Index
[b]DSR, Disease Severity Rating
–, Observations not recorded

Induction of sporulation

Since under normal conditions the culture of ***P. eucalypti*** failed to sporulate, different media such as eucalypt leaf extract agar, malt extract agar and yeast extract agar were tried with and without amendments. The inoculated plates were incubated under near ultra violet, ordinary red, blue, green fluorescent lamps and dark under 15 hr light period at 25 ± 2°C, but even after three months no pycnidia developed. In certain cases mycelial bodies were formed but they failed to form pycnidia. Various combinations of media tried are given in Tables 48 and 49.

Table 48. Different media prepared by amending malt extract agar medium to induce sporulation in ***P. eucalypti*** (agar ***2.5%;*** all ingredients (g) to prepare 1 l of medium)

Ingredients	Different amended media					
	i	ii	iii	iv	v	vi
Malt extract	20	20	20	20	—	20
Dextrose	20	—	20	20	20	—
Peptone	1	—	1	—	—	1
Yeast extract	—	2	2	2	2	2

Yeast extract	4	—	—	—	—	—	—
Malt extract	10	10	10	10	10	10	10
Dextrose	4	4	—	4	—	—	—
Vegemite	—	4	4	8	8	12	2

Control measures

A total of 13 fungicides were evaluated for their efficacy against ***P. eucalypti*** following poison bait technique. The results are summarised in Table 50.

Benlate, Bavistin and Tecto were the most effective fungicides as they inhibited the growth completely even at 0.1 per cent (a.i.). Other fungicides viz. Demosan, Furmetamid and Saprol inhibited the growth only at higher concentrations.

Table 50. Efficacy of various fungicides in inhibiting growth of *Phaeoseptoria eucalypti* (mean of 12 observations recorded from 3 replicates)

Sl. No.	Fungicide	% Concentration (a. i.)	Radial growth (mm) and SD
1.	Benlate	0.1	0
		0.25	0
		0.5	0
2.	Bavistin	0.1	0
		0.25	0
		0.5	0
3.	Bayleton	0.1	7.75 (0.95)
		0 25	4.91 (1.16)
		0.5	3.0 (0.96)
4.	Demosan	0.1	17 0 (0.95)
		0 25	8.12 (0.35)
		0.5	0.0
5.	Difolatan	0.1	17.62(2.62)
		0.25	17.75 (2.92)
		0.5	21.5 (0.52)
6.	Daconil	0.1	22.83(0.93)
		0.25	20.37 (1.50)
		0 5	9.16(1.33)
7.	Dithane M-45	0.1	13.83(1.89)
		0.25	8.66(1.37)
		0.5	6.75(0.46)
8.	Furmetamid	0.1	3.0
		0.25	2.04(0.75)
		0.5	0.0
9.	Fytolan	0.1	9.75 (0.88)
		0.25	7.5 (0.90)
		0.5	5.66 (0.77)
10.	Polyram combi	0.1	28.33(0.61)
		0.25	13 75 (0.93)
		0.5	2.75(0.50)
11.	Saprol	0.1	11.83(0.83)
		0.25	0.0
		0.5	0.0
12.	Syllit	0.1	24.58 (0.56)
		0.25	17.0
		0.5	9.91 (1.44)
13.	Tecto	0.1	0
		0.25	0
		0.5	0
14.	Control	—	65.58(2.84)

In another experiment where Bavistin and Benlate were evaluated at 0.01, 0.02 and 0.05 per cent (a.i.) growth of ***P. eucalypti*** was inhibited completely at the latter two concentrations. Efficacy of Bavistin (0.02 per cent a. i.) was confirmed in nursery trials where the disease was effectively controlled by two applications given at weekly intervals.

Discussion

Phaeoseptoria eucalypti was first reported by Hansford (1957) from Sydney, Australia on leaves of ***E. grandis.*** Later, Heather (1965) who studied the epidemiology of the disease on ***E. bicostata*** Maiden *et. al.* found that in natural forests, eucalypts belonging to subgenus Macranthera were only affected. However in nurseries and glasshouses, seedlings of other eucalypt sub-genera were attacked as well.

The disease has not been reported from any other country where eucalypts have been introduced, except India, though it has been noticed in Japan (J. K. Sharma, personal observation). There is a possibility that the disease got introduced through seeds or other plant material long time back when eucalypts were brought to India. In India, ***Phaeoseptoria*** leaf spot has earlier been reported from Karnataka on ***E. globulus*** (Padaganur and Hiremath, 1973). ***E. tereticornis*** and ***E. grandis*** are new host records for ***P. eucalypti*** in India. Another species, ***P. luzonensis,*** causing black powdery spots on ***Eucalyptus*** app. has been reported from Philippines (Kobayashi, 1978).

All attempts failed to induce sporulation on various media, including malt agar medium on which Heather (1965) had reported sporulation.

Chemical control trials indicate that the disease can be controlled effectively by Bavistin. It is recommended to treat the seedling in nurseries as soon as the disease is noticed. If the remedial measures are delayed, two applications of Bavistin may not be sufficient because of build up of high inoculum.

18. CONIELLA LEAF SPOT

Occurrence

Coniella leaf spot was first recorded at Periya (Wynad Div.) affecting ***E. grandis*** during monsoon of 1981. The disease caused extensive premature defoliation of the lower branches due to severe infection. During that year the rains were very heavy and incessant for a long period. However, during 1982 when the rainfall was comparatively less and intermittant, the incidence of the leaf spot in the same plantation was quite low, suggesting that the incidence and severity of the disease was greatly favoured by high humidity and moisture. This leaf spot was later

observed in ***E. tereticornis*** at Pezhad (0.33 per cent), Thalakode, Neriamangalam (Kothamangalam Div.), Anakulam (0.2 per cent), Mullachal (2.92 per cent) and in ***E. grandis*** at Noolpuzha (34.45—87.4 per cent), Mavinhalla (76.51 per cent) and Thrissillery (28.50 per cent). Though the disease may pose some problem during heavy monsoon in some areas, it was not of common occurrence.

A similar leaf spot caused by a different species of ***Coniella*** was observed on various provenances of ***E. grandis*** raised at peechi and at Chandanathode (Wynad Div.).

In plantations it was recorded at Pamba (Kerala Forest Development Corp.), Attappara (Ranni Div.) during monsoon of 1982. This spot disease affecting leaves of any maturity was not as serious as the first one.

Etiology and symptoms

(i) ***Coniella fragariae*** (Oudem.) Sutton (Syn. ***Coniella pulchella*** Hohnel) (IMI 262984, 278252).

The spots, usually along the leaf margins, appeared during the rainy season as more or less circular areas with regular margins (Figs. 50 a, b). Initially the spots were greyish-black in the centre gradually becoming lighter towards the periphery. During the dry period the spots turned light to pale brown. Leaves which had large area covered by the spots blighted and defoliated. Numerous light to dark brown coloured pycnidia (Figs. 50 c-e), arranged more or less in concentric rings developed even in a very small necrotic spot. As the spots enlarged new rings of pycnidia were added. During the wet period pycnidia produced off white to light pale coloured conidial ooze which was easily dispersed by rain drops, thus spreading the infection to other healthy leaves.

(ii) ***Coniella castaneicola*** (Ell. & Ev.) B. Sutton (IMI 278251).

The infection usually restricted to margins and the spots extended towards the midrib (Figs. 50 f, g). Initially the spots were greyish, irregular, later became dark reddish brown during the dry period, the necrotic area being brittle. The pycnidia, developed over the necrotic spot, were irregularly distributed, dark brown and minute.

Pathogenicity

Pathogenicity of ***C. fragariae*** and ***C. castaneicola*** was tested on 3-month-old container seedlings. The leaves of the test seedlings were washed thoroughly with sterile water mixed with Tween 20 (two drops in one litre of water). The seedlings were transferred to a humidity chamber maintained at > 95 per cent r. h. and

temperature and sprayed with sterile distilled water. The plants were inoculated after 24 hrs with a spore suspension (concentration of 100 to 150 spores/drop of a Pasteur pipette), prepared from a 15-day-old culture to which added a drop of Tween 20 to increase the adhesion with the leaf surface. Small drops of this spore suspension were placed on the adaxial surface of leaves of different maturities.

A small greyish black spot measuring about 2-3 mm developed at the site of inoculation on the younger leaves on the second day of inoculation. In mature leaves it took three to four days. These spots grew rapidly in size, measuring about 5-8 mm across within a week when pycnidia also started to develop. Koch postulates were confirmed by reisolating ***Coniella*** spp. in culture from these spots.

Discussion

So far on eucalypts three species of ***Coniella*** namely, ***C. castaneicola*** (E. ***viminalis, E. grandis, E. saligna, E. citriodora, E. tereticornis*** from various countries), ***C. minima*** Sutton & Thaung apud Sutton (***E. camaldulensis*** from Burma) and C. ***australiensis*** Petrnak ***(E. deglupta*** from Australia) have been recorded (Sutton, 1980). Of these only (***C. castaneicola*** is known to have a wide host range. ***C. fragariae*** recorded during the present survey is a new pathogen record for ***Eucalyptus; C. castaneicola*** and ***C. fragariae*** are reported for the first time from India.

19. PESTALOTIOPSIS LEAF SPOT

Occurrence

Leaf spots caused by ***Pestalotiopsis*** were recorded throughout Kerala on E. ***grandis, E. tereticornis*** and ***E. globulus,*** being more prevalent on the latter species. The leaf spots though observed throughout the year were prevalent during September to April.

Symptoms

Varied kind of symptoms were produced depending upon the species of ***Pestalotiopsis, Eucalyptus*** species and maturity of leaf (Figs. 51, 52). Usually the amphigenous spots were irregular pale brown to dark brown with clearly defined border along the leaf margins or on the lamina as light grey to pale scorchy spots. Occasionally black dot like fructifications developed either on both the surfaces of the necrotic spot or only on the (abaxial) lower surface. Detailed symptoms are described separately in respect of each species of ***Pestalotiopsis.***

Etiology and symptoms

P. disseminata (Thum.) Steyaert (1MI 268328)—On E. ***grandis*** at Noolpuzha and Pappathishola, on ***E. tereticornis*** at Kottappara; severity—low to medium.

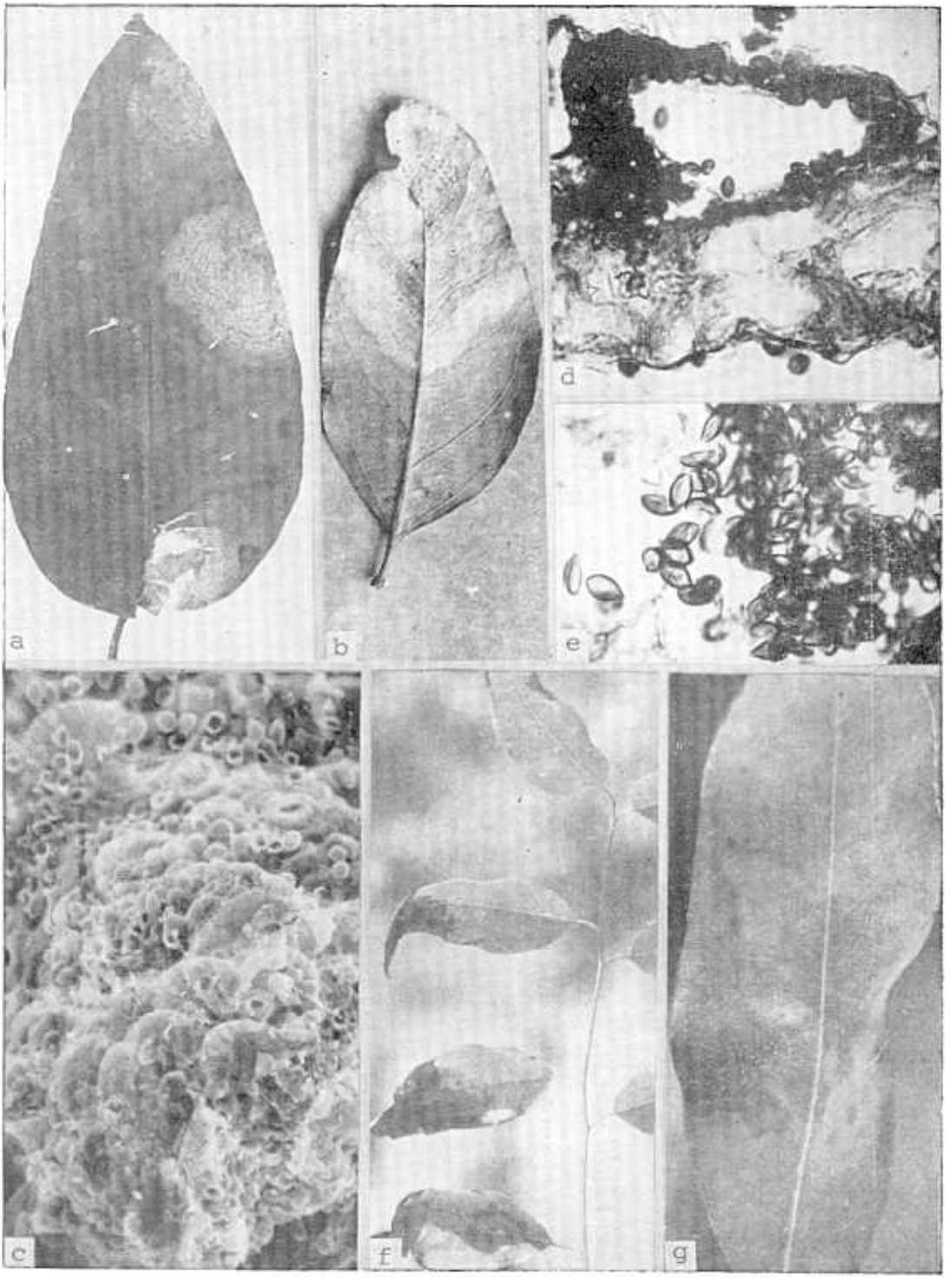
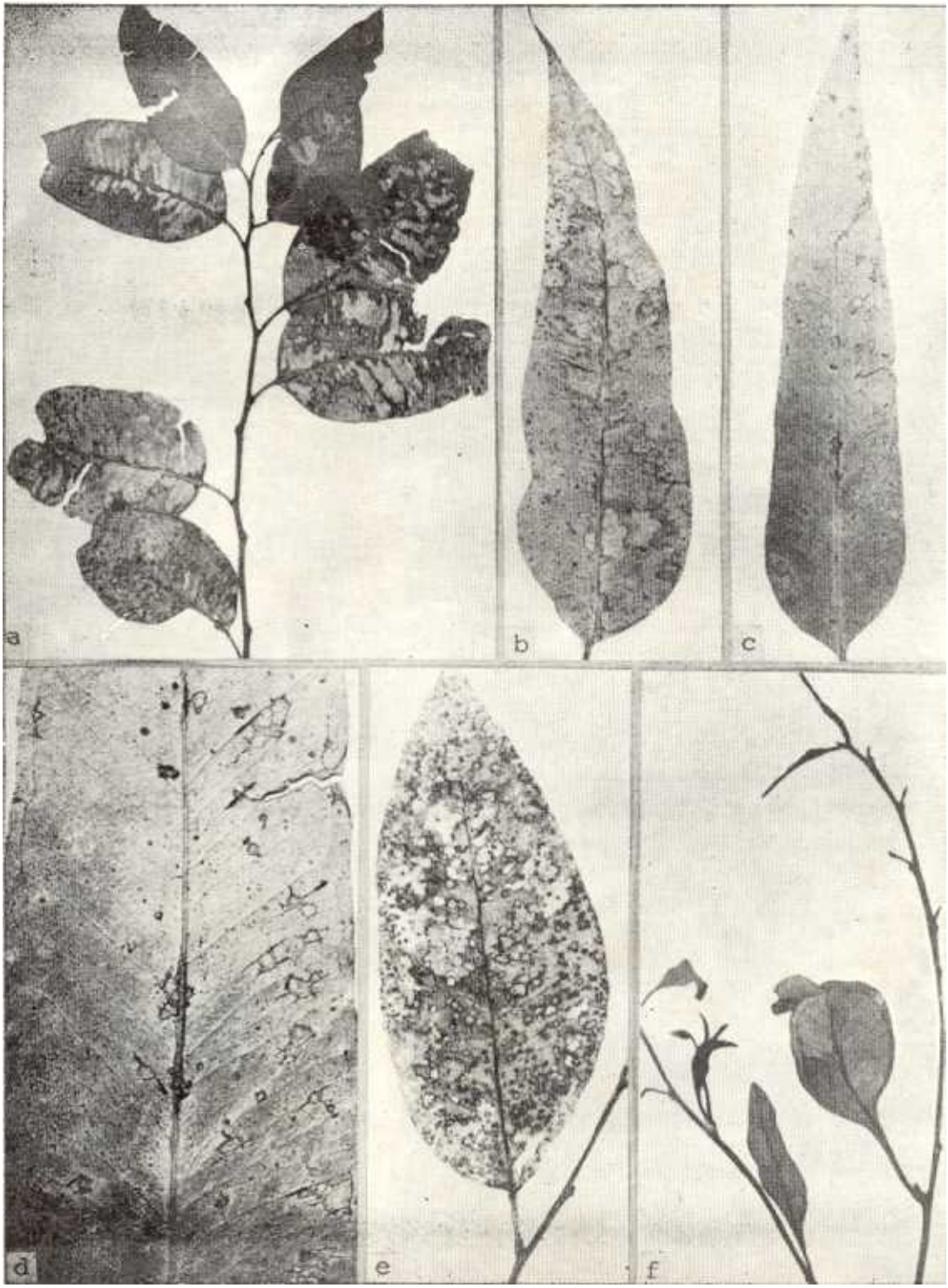


Fig. 50. Leaf spot of *Eucalyptus* caused by *Coniella* spp. a and b, Leaf of *E. grandis* and *E. tereticornis* respectively showing typical symptoms caused by *C. fragariae*. Note the concentric arrangement of pycnidia; c, SEM of surface of pycnidia with some conidia (250 X); d, A vertical section through a pycnidium (350 X); e, Conidia of *C. fragariae* (400) X); f and g, Leaves of *E. grandis* showing spots caused by *C. castaneicola*. Note that pycnidia are distributed irregulary over the lesion (g).



Fig

ised

Initial spots amphigenous pale brown with dark brown margins, elongated to oval on both sides of mid rib, more or less lined with secondary veins (Figs. 51 a,b). Later they coalesced to give rise larger irregular spots, similar in colour to smaller ones. Numerous acervuli were developed on the under surface of the spots.

Discussion

This is the first report of *P. disseminata* causing leaf spot of *E. grandis.*

(ii) *P. guepinii* (Desm.) Steyaert (IMI 268324, 268327, 268336) - on *E. grandis* at Noolpuzha, on *E. tereticornis* at Anakulam, Kottappara; severity- low.

Foliar spots amphigenous, circular, oval to irregular, light pale brown with a raised border, 1-2 mm wide, camel brown in colour, often marked by veins; occasionally along the veins a light brown streak was seen (Figs. 51 c,d).

Discussion

P. guepinii is considered to be a true parasitic species usually causing leaf spots. This is the first report of its occurrence on *E. grandis* and ***E. tereticornis.***

(iii) *P. neglecta* (Thum.) Steyaert (IMI 268329, 268331, 268333) - On *E. grandis* at Pachakanam and Uppupara (Peermade Div.), on *E. globulus* at Silent Valley; severity- low on *E. grandis* and medium to severe on *E. globulus.* It caused tip blight as well as leaf spots.

Initially spots minute, 1-2 mm diam., circular, purple on upper surface and greyish on the lower surface. These spots enlarged and became circular, oval to elliptical, dull brown with diffused to marked outline. The spots coalesced to form large necrotic areas causing leaf blight (Figs. 51e, 52a).

Tip blight, observed in *E. grandis,* first caused wilting of the apical shoot, 3-5 cm in length. Later the leaves, stem and the apical bud of the affected shoot turned greyish black in colour (Fig. 51 f).

Discussion

Eucalyptus grandis and *E. globulus* are new host record for *P. neglecta.*

(iv) *P. mangiferae* (P. Henn.) Steyaert (IMI 268326, 268330, 268334, 38) - On *E. grandis* at Noolpuzha, Thrissillery, on *E. tereticornis* at Periya, Thariode (Wynad Div.); severity- low to to medium.

The initial spots were circular, 2-3 mm in diam., pale yellowish-brown on the upper surface delimited by a sharp thin dark border, and light brown on the lower

surface. The spots later coalesced and became irregular (Fig. 52b); fructifications were seen only on the lower surface of the spots.

Discussion

P. mangiferae is a facultative parasite affecting a large number of host plants. This is the first report of its occurrence on ***E. grandis*** and ***E. tereticornis.***

(v) ***P. versicolor*** (Speg.) Steyaert (IMI 268332) - on coppice shoots of ***E. tereticornis*** at Onthupachha (Punalur Div.): severity - low.

Infection occurred usually along the margins and tip of leaf in the form of dark brown discolouration (Fig. 52 c). Later, the necrotic area extended towards the midrib appearing as diffused light brown patches.

Discussion

This is the first report of ***P. versicolor*** on ***E. tereticornis.***

20. PESTALOSPHAERIA LEAF SPOT

Occurrence

The disease was recorded only from Kottappara on ***E. grandis*** and ***E. tereticornis.*** The incidence of the disease was very low.

Symptoms

E. grandis : Initially the spots light brown, amphigenous, appeared at the leaf tip. Later, they coalesced to give rise large dull brown necrotic spots covering a large area, at times three-fourth of the lamina. Individual scattered small spots were also present on the leaf as irregular dull brown areas (Fig. 52 d).

E. tereticornis : Leaf spots were amphigenous, scattered, 1-2 mm diam. and more common along the leaf margins (Fig. 52 e). These spots coalesced to form irregular light brown areas with well demarcated slightly raised margins.

Etiology

Pestalosphaeria elaedis (Booth & Robertson) van der Aa (IMI 268335).

Discussion

This is the first report of ***Pestalosphaeria elaedis*** causing leaf spots of ***Eucalyptus*** spp. This fungus is also being recorded for the first time from India.

21. ALTERNARIA LEAF SPOT

Occurrence

This leaf spot disease was commonly observed in low incidence in young plants or coppice shoots of ***E. grandis*** and ***E. tereticornis.*** The disease usually appeared during the dry period i.e., December-April.

Symptoms

Initially minute greyish brown spots developed near the tip and along the margin of leaf (Fig. **53** a). These coalesced to form large dull brown to pale brown irregular necrotic areas with diffused margins. The necrotic area along the leaf margins often showed splitting due to wind.

Etiology

Alternaria alternata (Fr.) Kiessler (IMI 246476).

Discussion

Alternaria alternata is a common facultative parasite found to cause foliage diseases of many plants. Earlier leaf spots caused by ***A. alternata*** have been recorded on ***E. tereticornis*** from Uttar Pradesh (Bakshi *et al.*, 1972). ***E. grandis*** is a new host record for ***A. alternata.***

22. LITTLE LEAF DISEASE

Occurrence and severity

A little leaf disease of ***Eucalyptus tereticornis, E. grandis*** and ***E. globulus*** hitherto unreported, was recorded at several places. The incidence of little leaf disease was assessed in representative plantations as described earlier. In all the observation plots total number of affected plants were counted. In nurseries the total number of seedlings showing little leaf symptoms was counted in a standard bed (12 x 1.2 m) and the per cent incidence estimated by the seedling density in the bed. The latter was calculated from three 30 cm^2 areas in the bed.

The survey indicated that though the disease was widespread in the State, the incidence was below one per cent in the plantations (Table 51). The disease incidence, recorded in 1- to 5-year-old plantations, was higher in ***E. tereticornis*** than in ***E. grandis*** or ***E. globulus.*** Unlike in the plantations the incidence of little leaf disease in nurseries was relatively low (Table 52) possibly because of high density of seedlings in seedbeds. Further, the disease was observed more frequently in ***E. grandis*** than in ***E. tereticornis,*** except in a nursery at Thenoor where the incidence was much higher than in any of the nurseries of ***E. tereticornis*** surveyed. There was also an indication that different provenance of the same eucalypt species may vary in their level of susceptibility to little leaf disease. ***E. tereticornis*** beds raised from seeds collected from a nearby stand grown from pure Australian seeds recorded very low incidence of the disease when compared to the ***E. tereticornis*** (so called ***Eucalyptus*** 'hybrid') seeds obtained from Tamil Nadu Forest Department.

Table 51. Incidence of little leaf disease recorded in various *Eucalyptus* plantations of Kerala

Eucalyptus species	Year of planting	Locality	Forest Range	Forest Division	% incidence
E. tereticornis					
	1979	Kakkavayal	Tamarassery	Kozhikode	0.4
	1976	Nellikkara	Tamarassery	Nilambur	0.25
	1979	Cheenkannipally	Tamarassery	Nilambur	0.12
	1976	Varavoor	Wadakanchery	Trichur	0.05
	1979	Kottappara	Kodanad	Kothamangalam	0.17
	1976	Anakulam	—	Punalur (KFDC)[a]	0.08
	1979	Mullachal	Peringamala	Trivandrum (KFDC)	0.42
E. grandis					
	1978	Noolpuzha	Sultan's Battery	Kozhikode	0.05
	1980	Mavinhalla	Sultan's Battery	Kozhikode	0.0
	[b]	Thalayar	—	Munnar	0.05
	1978	Pappathishola	Devicolum	Munnar	0.58
E. globulus	1979	Silent valley	—	Munnar (KFDC)	0.35

[a]Kerala Forest Development Corporation

[b]Not known

Symptoms

The affected plants showed prominent stunting and produced much smaller leaves when compared to healthy ones. The new leaves showed considerable reduction in size and became thin, pale, scaly with narrow lamina. The apices of such leaves often showed browning. The internodes became stunted and all axillary buds got sprouted resulting in bushy shoots with abnormal minute leaves (Figs. 54 a-e). The root system of the diseased plants remained apparently unaffected as no abnormality was observed. Affected ***E. tereticornis*** trees became weak due to reduction in stem diameter and height growth; however, in ***E. grandis*** apparently no such symptoms were observed, except for the compact bushy appearance of shoots, which snapped easily on bending.

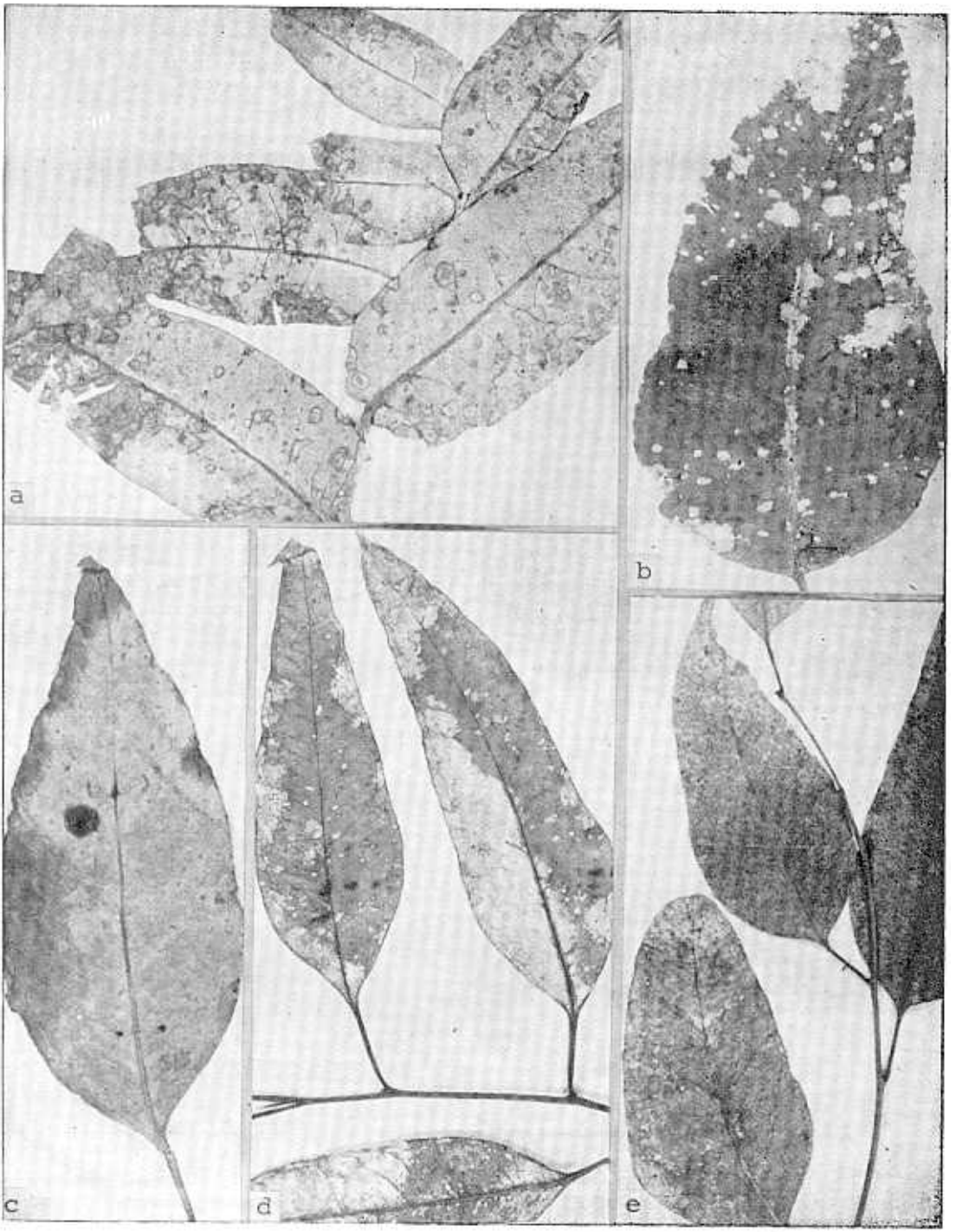


Fig. 52. Leaf spots of *Eucalyptus* caused by *Pestalotiopsis* and *Pestalosphaeria*. a, *P. neglecta* on *E. globulus*; b, *P. mangiferae* on *E. grandis*; c, *P. versicolor* on *E. tereticornis*; d and e, *Pestalosphaeria elaedis* on *E. tereticornis* (d) and *E. grandis* (e).

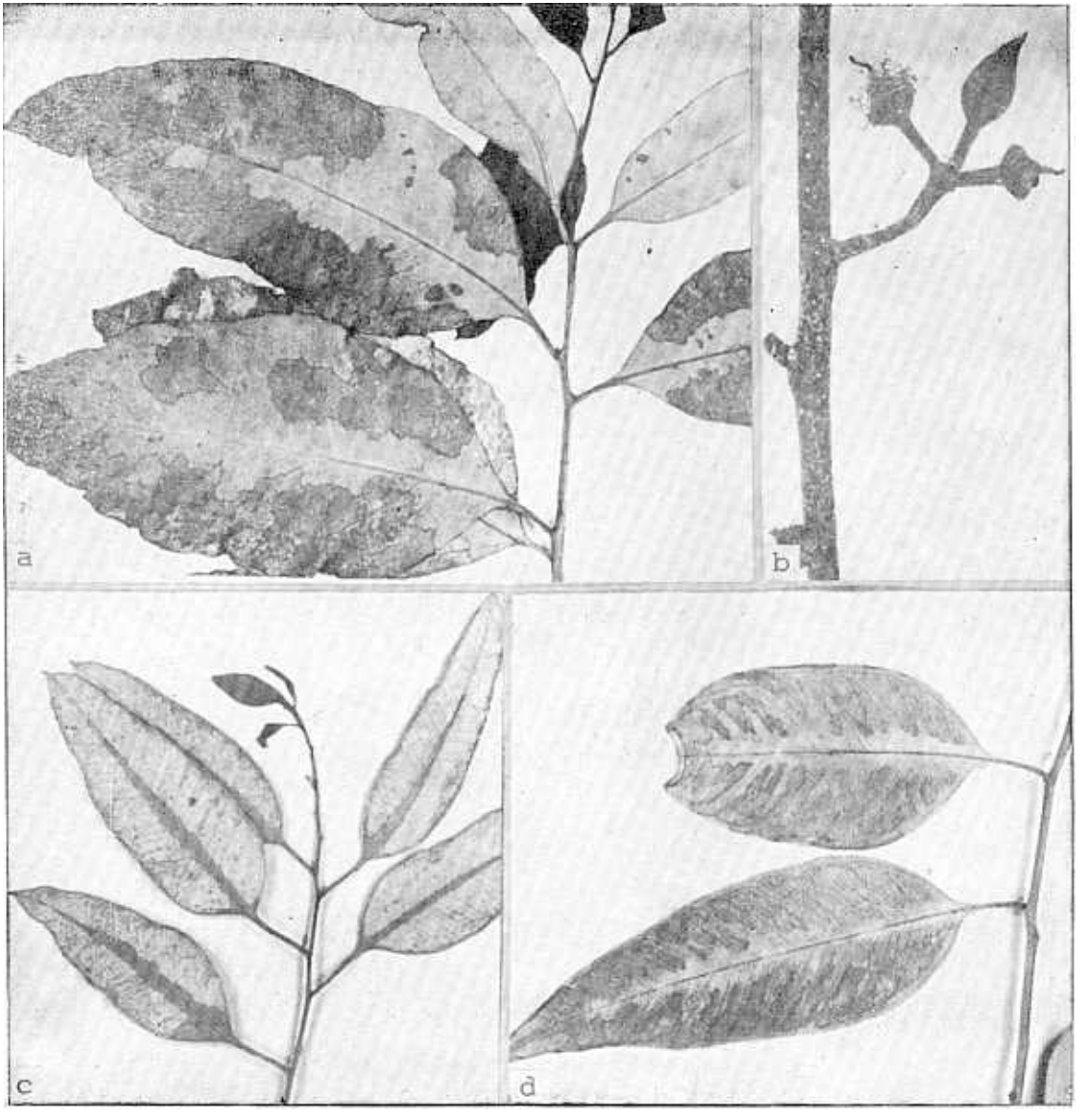
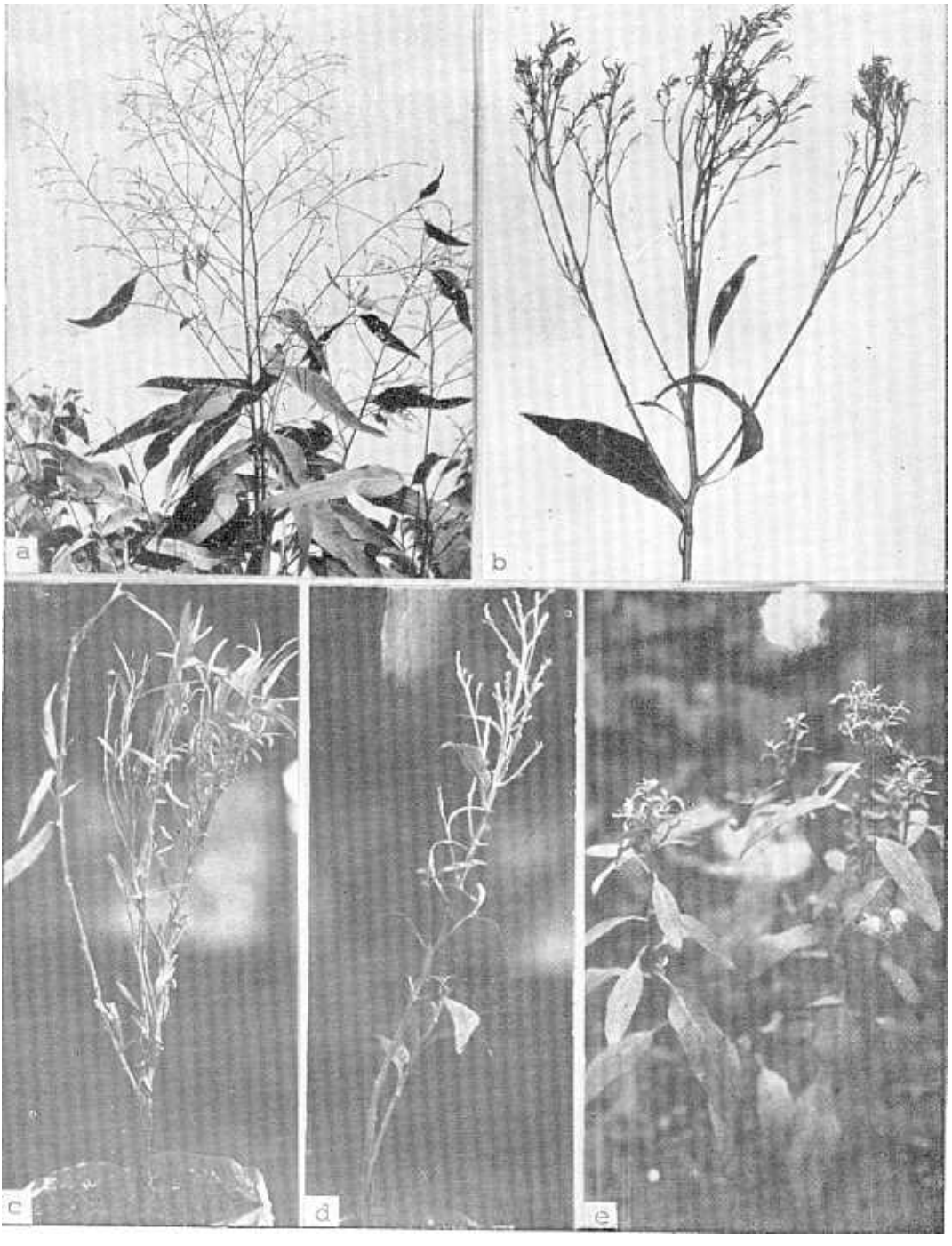


Fig. 53. Miscellaneous diseases of *Eucalyptus*. a, Leaf spots caused by *Alternaria alternata*; b, Capsule infection of *E. camaldulensis* caused by *Colletotrichum gloeosporioides* and *Torula* sp. c, Injury caused by fluorine on leaves of *E. tereticornis*; d, Injury caused by sulphur dioxide on leaves of *E. tereticornis*.


a
b
c
d
e

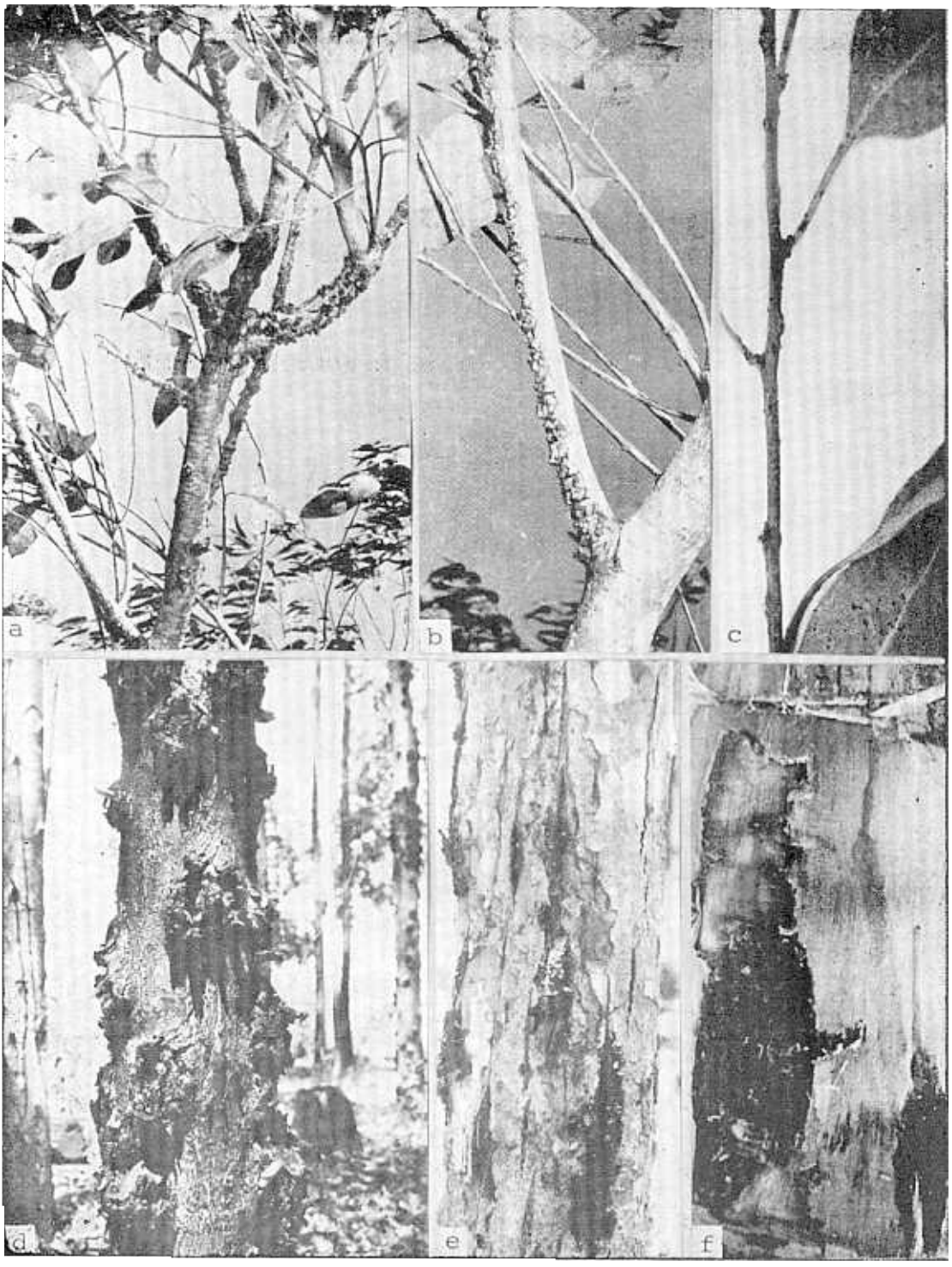


Fig. 55. Diseases of unknown etiology of *Eucalyptus*. a, A 3-year-old tree of *E. tereticornis* infected with Cankerous stem gall disease; b, Development of galls on the under surface of the branch; c, Initial infection in the form of minute tumour like outgrowths on stem, petiole and leaf; d, e and f, Seven-year-old tree of *E. grandis* infected with gummosis. Note oozing of kino from the canker area (e) and an exposed kino pocket in the stem (f).

Table 52. Incidence of little leaf disease of ***Eucalyptus*** recorded in various nurseries situated in different locations during 1980-1982

Eucalyptus species	Year (nursery raised)	Total No. of seedbeds 12x1.2m	Locality	Forest range	Forest Divn.	% incidence
E. grandis	1980	30	Potta	Wadakkanchery	Trichur, KFDC[b]	0.011
E. teretieornis	1980	52	Potta	Wadakkanchery	Trichur, KFDC[b]	0.00
E. grandis	1981	40	Pongali	Sultan's Battery	Kozhikode	0.01
E. grandis	1981	100[a]	Chandanathode	Kannoth	Wynad	0.032
E. tereticornis	1981	100[a]	Chandanathode	Kannoth	Wynad	0.005
E. grandis	1982	45	Chandanathode	Kannoth	Wynad	0.074
E. tereticornis	1982	45	Chandanathode	Kannoth	Wynad	0.003
E. grandis	1982	7[a]	Peechi	—	KFRI Campus	0.096
E. tereticornis	1982	120	Uppukuzhi	Kothamangalam	Kothamangalam	0.008
E. tereticornis	1982	8	Thenoor	Peringamala (Palode)	Trivandrum (KFDC)	0.002
E. tereticornis (commonly called ***Eucalyptus*** 'hybrid')	1982	42	Thenoor	Peringamala (Palode)	Trivandrum (KFDC)	0.254

[a] bed size 3 x1 m; ***E. tereticornis*** and *E. **grandis*** were sown separately in half area of the bed

[b] Kerala Forest Development Corporation

Etiology

Occurrence of isolated eucalypt tree(s) in plantations and variation in disease incidence in different provenance of the same species of eucalypt at Thenoor, rule out the possibility of association of little leaf symptoms with any nutritional deficiency in the soil. Since no fungal or bacterial isolations could be made from the plant parts (leaves and roots) of the affected trees, infection due to a viral or mycoplasma-like-organisms (MLO) was suspected. Graft and sap transmission studies for both the little leaf diseases from ***E. tereticornis*** and ***E. grandis*** were conducted employing six-month-old plants of ***E. tereticornis,*** which even after one-year did not yield any positive results.

In histopathological studies presence of mycoplasma like organisms was confirmed using Dienes' stain technique (Deeley ***et al.,*** 1979).

Discussion

The little leaf disease is suspected to be seed- borne as it is seen appearing even in very young seedlings. The reason for observing the disease more frequently in ***E. grandis*** than in ***E. tereticornis*** in the nursery as compared to the plantation may reflect late expression of symptoms in the latter species, which may be related to the seed source and the climatic conditions. This was later confirmed in the nurseries situated at Chandanathode (Wynad Div.) and Pattikkad (Trichur Div.) by making observations at regular intervals from the day of emergence till the saplings were 10 months old. Typical symptoms of abnormal leaves appeared in ***E. grandis*** as early as one month following emergence, whereas in ***E. tereticornis*** it took normally 3-5 months for expression of the symptoms. In both the eucalypts prior to the appearance of the disease all the leaf pairs produced on the seedlings were apparently healthy and of normal size (Fig. 54 a, e).

Earlier Sastry *et al*. (1971) have reported a graft transmissible little leaf disease in four or five years old trees of ***E. citriodora*** which was stated to be caused by a virus. As most of the little leaf diseases reported so far are caused by MLO, graft transmission studies on plants of different ages of both the species were repeated following standard procedures but even after one year of grafting, no symptoms appeared. Since the symptoms of little leaf disease are expressed earlier in ***E. grandis*** than in ***E. tereticornis*** it is suggested to attempt transmission of the causal organism on ***E. grandis.***

The positive fluorescence observed in phloem tissues with Dienes stain is an indicaition of possible association of MLO with the little leaf disease. Recently transmission electron microscopic studies of the diseased tissues and tetracycline, therapy of little leaf affected trees by Ghosh *et al.* (1985) have proved that little leaf disease of ***Eucalyptus*** is caused by MLO.

23. CAPSULE INFECTION

Occurrence

Infection of capsules was observed in a 5-year-old *E. **camaldulensis*** plantation at Vazhachal during 1980. The infection was widespread only on this eucalypt species though others were also in flower.

Symptoms

The infection was initiated on the operculum of the capsule, which became leathery and then dried up (Fig. 53 b). Due to infection the operculum were not

shed and remained attched. The stamens inside showed curling and browning. Later the infection also spread to the capsule where numerous fructifications of the causal organisms were noticed. Occasionally the infection was also observed on the pedicel of the capsule.

Etiology

i. ***Colletotrichum gloeosporioides*** and ii. ***Torula*** sp.

Discussion

Due to widespread infection of capsules the seeding in ***E. camaldulensis*** was reduced considerably. Flowers required for hybridization work by the Genetics Division of KFRI could not be obtained due to the infection of capsules. ***C. gloeosporioides*** is known to be pathogenic but the role of ***Torula*** sp. in causing the infection is doubtful. This is the first report of infection of capsules of ***Eucalyptus.***

DISEASES OF UNKNOWN ETIOLOGY

24. CANKEROUS STEM GALL

Occurrence

The disease was recorded only in ***E. tereticornis*** plantations at Varavoor, Elanad (Trichur Div.), Potta (Kerala Forest Development Corp., Trichur), Kottappara, Thalakode (Kothamangalam Div.), Anakulam and Mullachal. Almost in all the plantations of ***E. tereticornis*** a few trees were always found affected with cankerous stem gall. The incidence was high ca. 10 per cent in a 1977 plantation at Anakulam but none of the affected trees died.

Symptoms

Initially minute tumour like outgrowths (galls) developed on the underside of the lower branches near the main stem. The small galls enlarged in size and got fused with the neighbouring ones to give rise large round to irregular galls. Later these galls extended and covered the entire area of the branch (Figs. 55 a-c). The bark over the galls and branch as a whole became dark brown and showed splitting. In severe cases all the branches and even the petioles developed the galls. The infection resulted in stunted growth of plants.

Etiology

Possibly ***Agrobacterium tumefaciens*** (E. F. Sm.) Conn.

All the efforts to isolate the bacterium on various bacteriological culture media failed. An indirect method of isolation of bacterium was tried employing different known hosts of ***A. tumefaciens.*** For the purpose, 2-week-old test seedlings of ***Petunia***

sp., *Viciafaba, Vigna sinensis, Phaseolus vulgaris, Lycopersicum esculentum* were grafted with a V-shaped diseased tissue (taken from a small gall) in V-shaped wedge cut out on the stem of the test plant. The inoculated plants were transferred to a humidity chamber and observed regularly for the development of the disease. Minute gall like structures were observed only in *Phaaseolus vulgaris* and *Vigna sinensis* but no bacterium could be isolated from them.

Discussion

A gall disease known as crown gall caused by *Agrobacterium tumefaciens* has been reported on *E. citriodora* in Brazil (Arruda, 1943), *E. camaldulensis* in Argentina (Fernandez Valida *et. al,*, 1954), Chile (Autter, 1964), on *Eucalyptus* spp. in Madagaskar (Gibson, 1967), Spain (Benito, 1957), Argentina and Columbia (Reis and Hodges, 1975), and on *E. maculata* in Taiwan (Hsieh, 1980). Serious losses due to crown gall disease have only been reported from Brazil.

The disease recorded in Kerala resembles the crown gali reported elsewhere, but the identity of the pathogen could not be established in our studies. Since an incidence of ca. 10 per cent was recorded at Anakulam, it may become a serious problem in future plantings as in Brazil. Detailed investigations are required to confirm the etiology and epidemiology of this gall disease.

25. GUMMOSIS

Occurrence

Gummosis was recorded both in *E. tereticornis* and *E. grandis,* more common in the latter species. At least a few plants in each plantation developed gummosis symptoms. Plants from 2-year-old onward were found to be affected. Incidence of the disease varied from <1 per cent to 22.37 per cent in various plantations, the latter beeing at Mavinhalla, at Thrissillery it was 5.07 per cent. Occasionally the younger plants (Zyear-old) died due to gummosis as recorded at Peermade (UPASI *E. grandis* plantation). However, gummosis in old plants did not cause any mortality.

symptoms

In *E. tereticornis* usually the bark near the base of the stem first showed irregular vertical splitting through which kino oozed out in the from of redish brown streak. Gradually, cracks also appeared upwards and kino oozing was noticed. The stem of the affected tree showed some abnormalities in the form of swelling.

In *E. grandis,* initially the .symptoms were different. The bark of the affected stem developed prominent tumour like swellings followed by extensive splitting of bark, which accompanied with profuse exudation of kino (Figs. 55 d, e). When the bark was cut and the sap wood exposed, thick kino oozed out from the kino venis or

pockets (Fig. 55 f). The stem of the affected tree appeared dark in colour with abnormal outgrowth spread over its entire surface. A branch showed die-back when gummosis reached its base.

Etiology

All attempts to isolate the causal organism failed.

Pathological anatomy: Bakshi and Sujan Singh (1964) have described in detail the development of kino veins in eucalypts. According to them a mild injury causes the cambium to develop undifferentiated parenchymatous tissue instead of normal cells. The medullary rays become laterally displaced and the ray cells become markedly broadened at the region of the false ring. Within this tissue, kino pockets develop lysigenously or schizo-lysigenously. In the next growing season, cambial activity may be renewed resulting in formation of normal wood and bark in which the parenchyma tissue gets embedded as an incomplete or a false ring.

Discussion

Gummosis of eucalypts has earlier been recorded from Mauritius (Anon., 1958 a), Sudan (Scharif, 1964), Congo (Gibson, 1967) and South America (Anon., 1958 b). In KeraIa as the disease occurs in low incidence it is not considered as a serious problem. Often it was difficult to ascribe any cause, such as injury, soil or water stress for the gummosis especially when the growth of the trees was fairly good. It is not known that some injury to roots could also initiate the gummosis. The disease needs to be investigated in detail to understand the etiology and the factors responsible for gummosis.

26. MOSAIC

Occurrence

A mosaic disease of *E. **tereticornis*** was recorded in nurseries and plantations. In nurseries the disease occurred rarely, however, in plantations invariably a few trees were always found to be affected with mosaic disease. The incidence of mosaic disease was generally very low (<0.1 per cent) except at Naduvanoor Pacha (Thenmala Div.) where it was ca. 0.5 per cent.

Symptoms

All the leaves, young as well as mature, of the affected plants had mosaic symptoms i.e., light pale to white, irregular patches. Even the new leaves showed these symptoms (Figs. 56 a,b). The diseased plants did not show any effect on growth, except that the affected leaves became leathery and thick.

Etiology

Suspecting that the disease could be viral in nature, standard methods of sap and graft transmission were attempted on 1-year-old container seedlings of ***E. tereticornis*** but negative results obtained.

Discussion

Sastry *et al*. (1971) have reported a mosaic disease of ***E. citriodora*** caused by tobacco mosaic virus (TMV) from India, which was sap transmissible. The mosaic disease recorded in Kerala differs considerably in symptomatology and also in that it could not be transmitted. Though the disease is not economically important some studies are warranted to establish the etiology.

27. LEAF CURL

Occurrence

A leaf curl disease of ***E. tereticornis*** was observed in a 2-year-old plantation at Kottappara and of ***E. grandis*** at Thrissillery. The incidence of the disease was very low.

Symptoms

In affected plants all the leaves, young as well as mature showed upward or downward curling of the lamina with the margins becoming wavy and curved inward (Fig. 56 c); some puckering and vein bending was also observed. The internodes of the diseased plants showed elongation.

Etiology

The etiology of the disease could not be established as it could not be transmitted either through sap or graft methods.

Discussion

A leaf crinkle of ***E. citriodora*** has been reported earlier by Sastry *et al*. (1971) which was graft transmissible. Since the leaf curl disease was recorded in very low frequency it appears to be unimportant.

28.

Occurrence

A disease similar to ring spot virus disease was observed in ***E. tereticornis*** at Pezhad and Anakulam. The incidence of the disease was very low.

Symptoms

All the affected leaves, including young ones had typical symptoms of ring spot. One to two pale green concentric rings of varying sizes were observed all over

the lamina (Fig. 56 d). The disease did not cause any stunting or malformation, except the affected leaves became thick and leathery. Occasionally ring spot symptoms were observed only in leaves of a few branches of the trees.

Etiology

Since no studies could be carried out the etiology of the disease is unknown.

Discussion

In eucalypts there is no earlier record of ring spot virus. Though the symptoms resembled to a ring spot virus disease in absence of transmission studies the etiology could not be established. The disease appears to be unimportant as it occurred in very low incidence.

NON-INFECTIOUS DISEASES

Fluorine and sulphur dioxide injuries

Occurrence

Foliar injury was observed in ***E. tereticornis,*** planted as a 12 ha green-belt around Indian Aluminium Co. (IAC), Kalamassery due to gases emitted from the factory of IAC and nearby factories, especially Fertilizers and Chemicals Travancore Ltd. (FACT) and Hindustan Insecticides Ltd. (HIL). Two different types of injuries recognized are described below.

Fluorine (F) injury

The incidence of this injury was very high in saplings planted in the vicinity of the factory and low in the belt planting.

The symptoms were expressed as decolourization of leaf lamina from tip downwards, covering half to three-fourth of the area (Fig. 53 c). In severe cases the whole leaf got affected and dried up.

Sulphur dioxide (SO_2) iujury

The incidence of the injury was common on plants in the green belt planting, which was close to FACT and fell in the region of easterly winds from the FACT side, which brought SO_2 to this area.

The leaves showed very characteristic reddish brown spots obliquely placed on both sides of midrib along the margin (Fig. 53 d). The affected leaves soon dried up.

Control

Though it is possible to prevent the pollution injuries by application of certain chemicals, it is not economical. Since ***E. tereticornis*** appears to be susceptible to F and SO_2 injuries it is recommended to grow other plant species such as ***Ficus***

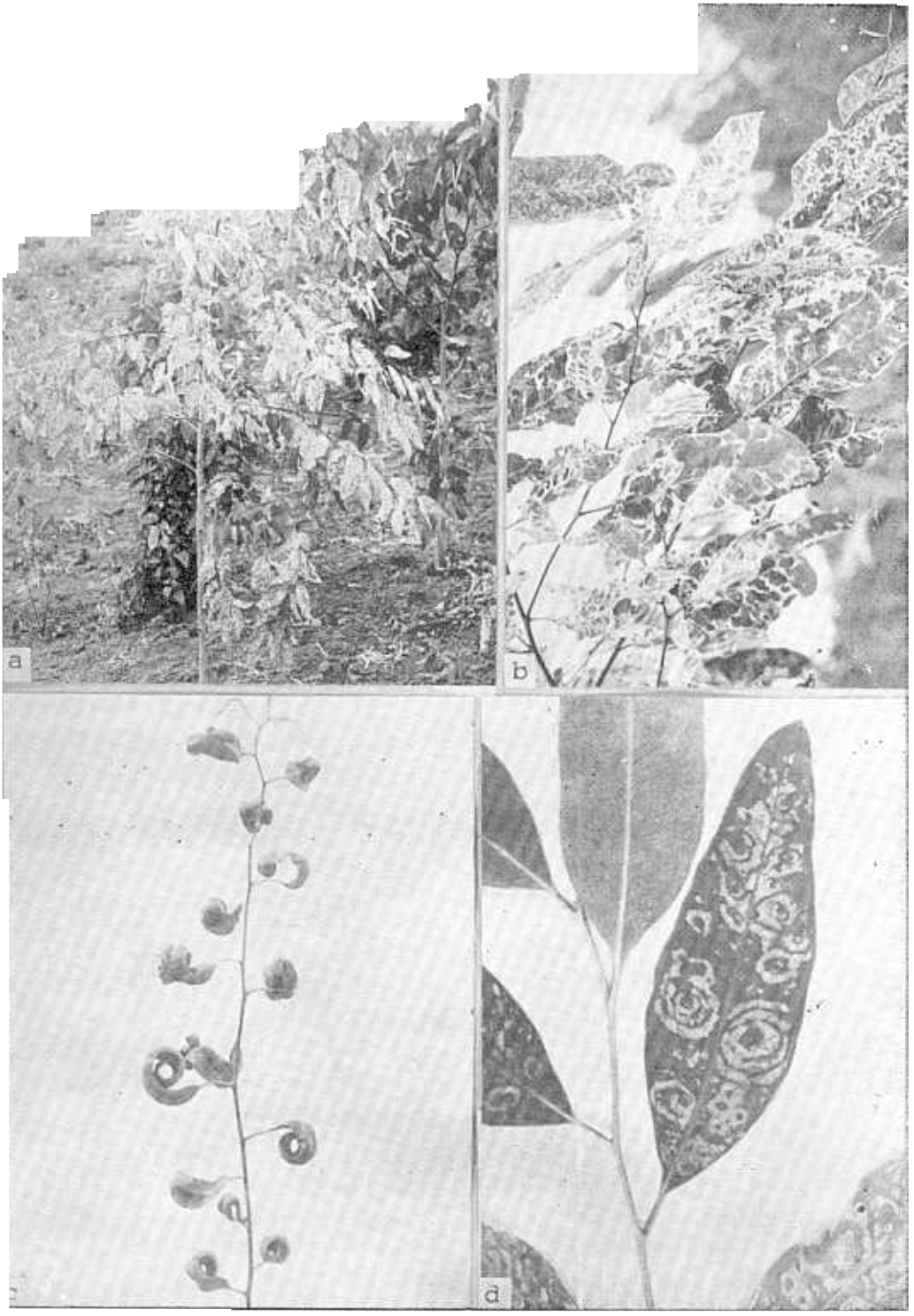
elastica, Ficus spp. and Fan Palm (for Fluorine) and mulberry (for Sulphur dioxide) which are known to be resistant to these gaseous injuries.

GENERAL DISCUSSION

During the survey a total of 46 fungal organisms were recorded on *Eucalyptus* of which 15 caused various diseases of seedlings in nursery and 35 in plantations; six pathogens i.e., *Cylindrocladium quinqueseptatum, C. ilicicola, C. scoparium, C. floridanum, Fusarium oxysporum* and *Phaeoseptoria eucalypti* were common to both nursery and plantations. As many as 31 pathogens *(Pythium deliense*, P. myriotylum*, P. spinosum*, Cylindrocladium parvum, C. camelliae*, C. curvatum, Calonectria ilicicola, C. theae* and its imperfect state *Cylindrocladium theae, Coniella granati*, C. fragariae, C. castaneicola", Cylindrocarpon lucidum*, Sclerotium rolfsii*, Botryodiplodia theobromae*, Valsa eucalypticola* sp. nov., *Cytospora eucalypti* sp. nov., *Macrovalsaria megalospora, Thyronectria pseudotricha*, Hysterium angustatum, Nattrassa toruloidea** and conidial state *Scytalidium, Pestalotiopsis disseminata*, P. guepinii*, P. neglecta*, P. mangiferae*, P. versicolor*, Pestalosphaeria elaedis, Microporus xanthopus, Lentinus squarrosulus*, Colletotrichum gloeosporioides** and *Torula** sp.) were recorded for the first time on *Eucalyptus.* Of these 19 pathogens (marked with*) are already known to occur in India on various other hosts, while the remaining ones together with *Cylindrocladium floridanum* and its perfect state, *Calonectria floridana, Cylindrocladium clavatum, C. curvatum, Cryphonectria cubensis, C. gyrosa, Valsa eucalypti, Cytospora eucalypticola, Scytalidum* state of *N. toruloidea* are new reports from India.

Though the number of pathogens in nursery was far less than in plantations, they caused disproportionately large number of diseases in high incidence within a short period of six months (Table 53). Gibson and Jones (1977) have also pointed out that in nurseries, due to increased proximity between host units, which makes microclimatic condition conducive for infection, generally high incidence of diverse diseases occur. Further evidence that these nursery diseases are largely favoured by crowded condition and age of plants is provided by the fact that many of them were much reduced or disappeared altogether after the stock was planted in the field. This appears as true of diseases of leaves and stem as it is for less specialized root pathogens.

Some disease problems either of minor or major importance were always observed in eucalypt nurseries surveyed. The most prevalent nursery diseases were damping-off, web blight and seedling blight in seedbeds and stem canker, leaf and shoot blights and Phaeoseptoria leaf spot in container plants. *Cylindrocladium* spp., *Rhizoctonia solani* and *Pythium* spp. were the main serious pathogens. Various diseases found in seedbed and container seedlings appeared to be related to the age of



diseases of unknown etiology

the seedlings, and to prevailing micro and macro-climatic conditions. Eucalypt seedlings remained in seedbeds for about two to three months (when the season was warm and dry) and in containers for about four to five months (when initially the climatic conditions were also warm and dry but later changed to wet-humid following the onset of the monsoon). A few of the diseases were associated only with a particular growth stage of seedlings while others appeared just after the emergence of seedlings and continued to affect even the container plants. Thus a trend could be seen in the succession of diseases in eucalypt nurseries. Often absence or appearance of only a particular disease in eucalypt seedlings of a specific age was also recorded. This appeared to be related to the particular nursery practices, such as sowing time, density of seedlings, shade, quantity and frequency of watering, etc., followed in raising seedlings.

Table 53. Checklist of nursery diseases of ***Eucalyptus*** recorded in Kerela

SI. No.	Disease	Pathogen (s)	New pathogen record for ***Eucalyptus***	First record of pathogen from India
1.	Pre-emergence damping-off	***Rhizoctonia solani*** Kuhn.	—	—
2.	Post-emergence damping-off	(i) ***R. solani*** Kuhn. state of ***Thanatephorus cucumeris*** (Frank.) Donk	—	—
		(ii) ***Pythium de/iense*** Meurs	+	+
		(iii) ***P. myriotylom*** Drechsler	+	—
		(iv) ***P. spinosum*** Saw	+	—
		(v) ***Cylindrocladium quinqueseptatum*** Boedijn & Reitsrna	—	—
		(vi) ***C. ilicicola*** (Hawley) Boedijn & Reitsrna	—	—
		(vii) ***C. floridanum*** Sobers & Seymore	—	+
		(viii) ***C. parvum*** Anderson	+	+
		(ix) ***Fusarium oxysporum*** Schlecht.	—	—
3.	Cylindrocladium cotyledon spot	***C. quinqueseptatum***	—	—
4.	Web blight	***R. solani*** state of ***Thanatephorus cucumeris***	—	—
5.	Seedling blight	(i) ***R. so/ani***	—	—
		(ii) ***Cylindrocladium quinqueseptatum***	—	—
		(iii) ***C. ilicicola***	—	—
		(iv) ***C. parvum***	+	+
		(v) ***C. clavatum*** Hodges & May	—	+
		(vi) ***C. camelliae*** Venkataramani & Venlcataram	+	—
		(vii) ***C. scopar/um*** Morg.		—
		(viii) ***Coniella granati*** (Sacc.) Petrak & Syd.	+	—

Table 53. Contd.

6.	**Seedling wilt**		***Sclerotium rolfsii*** Sacc. state of ***Corticium rolfsii*** Curzi	+	—
7.	**Cylindrocladium leaf spot**	**(i)**	C. ***quinqueseptatum***	—	—
		(ii)	***C. ilicicola***	—	—
		(iii)	***C. clavatum***	—	+
		(iv)	***C. camelliae***	+	—
8.	**Seedling stem infection**	**(i)**	***Cylindrocladium quinqueseptatum***	—	—
		(ii)	***C. ilicicola***	—	—
		(iii)	***C. clavatum***	—	+
9.	**Shoot blight**	**(i)**	***Cylindrocladium quinqueseptatum***	—	—
		(ii)	***C. ilicicola***	—	—
		(ili)	**C.** ***clavatum***	—	+
10.	**Pbaeoseptoria leaf spot**		***Phaeoseptoria eucalypti*** (Hansf.) Walker	—	—
11.	**Root rot**	**(i)**	***Cylindrocladium curvaturn*** Boedijn & Reitsma	+	+
		(ii)	***Sclerotium rolfsii***	+	—
		(iii)	***Rhizoctonia solani***	—	—
12.	**Collar rot**	**(i)**	***R. solani***	—	—
13.	**Little leaf**		Mycoplasma like organisms	+	+

In plantations, diseases of foliage (18 pathogens) dominated, followed by those of stem (14 pathogens) (Table 54). There were only three root diseases i. e., root rot caused by ***Calonectria floridana*** and ***Cylindrocarpon lucidum*** and a wilt by ***Fusarium oxysporum.*** In plantations, association of a large number of pathogens with various diseases would be expected due to changing morphology and physiology of plants with age, micro-climatic conditions, season and also because plants are exposed for a long duration.

During the early sixties, when large-scale planting of eucalypts began in Kerala, infection by ***C. quinqueseptatum*** was a serious problem in raising healthy nurseries, A clear picture of various seedling diseases occurring in eucalypt nurseries has emerged from this survey. What was earlier considered to be due to a single species of ***Cylindrocladium,*** has turned out to be due to as many as eight species viz. ***C. quin queseptatum, C. scoparium, C. ilicicola, C. clavatum, C. curvatum, C. floridanum. C. camelliae, C. parvum,*** the latter six being recorded for the first time on ***Eucalyptus*** from India. ***Cylindrocladium*** spp. together with ***Rhizoctonia solani*** and ***Pythium*** **spp.** ***(P. spinosum, P. myriotylum*** and ***P. deliense)*** cause a disease complex (damping-off seedling blight, leaf spot. stem infection, leaf and shoot blights) at different growth stages of seedlings. As these diseases relate to a particular growth phase of plants, they appear almost in a succession and cause mortality in various degrees at every

Table 54. Checklist of plantation diseases of *Eucalyptus* recorded in Kerala

Sl. No.	Disease	Pathogen (s)	New host record for pathogen	First record of pathogen from India
1.	Root rot	(i) *Calonectria floridana* Sobers *Cylindrocladium floridanum* Sobers & Seymore	—	+
		(ii) *Cylindrocarpon lucidum* Booth	+	—
2.	Wilt	*Fusarium oxysporum* Schlecht.	—	—
3.	Botryodiplodia Stem (root collar) canker	*Botryodiplodia theobromae* Pat.	+	—
4.	Stem canker	(i) *Cryphonectria cubensis* (Bruner) Hodges	—	+
		(ii) *C. gyrose* (Berk. & Br.) Sacc.	—	+
		(iii) *Hysterium angustatum* Alb. & Schwein	+	+
		(iv)' *Valsa eucalypti* Cook & Harkness	—	+
		Valse eucalypticola sp. nov.	+	+
		(v) *Macrovalsaria megalospora* (Mont.) Sivan.	+	+
		(vi) *Thyronectria pseudotricha* (Schw.) Seeler	+	—
		(vii) *Corticium salmonicolor* Berk. & Br.	—	—
		(viii) *Cytospora aucalypticola* van der Westhuizen	—	+
		Cytospora eucalypti sp. nov.	+	+
		(ix) *Nattrassa toruloidea* (Nattrass) Dyke & Sutton	+	—
		Scytalidium state of *N. toruloidea*	+	+
5.	Stem decay	(i) *Microporus xanthopus* (Fries) O. Kunze	+	+
		(ii) *Lentinus squarrosulus* Mont.	+	—
6.	Cylindrocladium stem canker	(i) *Cylindrocladium quinqueseptatum*	—	—
		(ii) *C. ilicicola*	—	—
		(iii) *C. theae*	—	+
7.	Phaeoseptoria leaf spot	*Phaeoseptoria eucalypti* (Hansf.) Walker	—	—
8.	Cylindrocladium leaf spot; leaf blight	(i) *Cylindrocladium quinqueseptatum* Boedijn & Reitsma	—	—
		(ii) *Calonectria ilicicola* Boedijn & Reitsma *Cyl. ilicicola* (Hawley.) Boed. & Reit.	—	—
		(iii) *Calonectria floridana* Sobers *Cyl. floridanum* Sobers & Seymore	—	+
		(iv) *Calonectria theae* Loos *Cyl. theae* (Petch) Alf. & Sob.	+	+
		(v) *Cyl. clavetum* Hodges & May	—	+
		(vi) *Cyl.scoparium* Morg.	—	—
9.	Coniella leaf spot	(i) *Coniella fragariae* (Oudem.) Sutton	+	+
		(ii) *Coniella castaneicola* (Ell. Ev.) B. Sutton	—	—

Contd.

10.	Pestalotiopsis leaf spot	(i) ***Pestelotiopsis disseminata*** (Thum.) Steyaert	+	
		(ii) ***P. guepinii*** (Desm.) Steyaert	+	—
		(iii) ***P. neglecta*** (Thum.) Steyaert	+	—
		(iv) ***P. mangiferae*** (P. Henn.) Steyaert	+	—
		(v) ***P. versicolor*** (Speg.) Steyaert	+	—
11.	Pestalosphaeria leaf spot	***Pestalosphaeria elaedis*** (Booth & Robertson) van der Aa	+	+
12.	Alternaria leaf spot	***Alternaria alternata*** (Fr.) Kiessler	—	—
13.	Guignardia leaf spot	***Guignardia citricarpa*** Kiety	—	—
14.	Capsule infection	(i) ***Colletotrichum gloeosporioides***	+	—
		(ii) ***Torula*** sp.	+	—
15.	Little leaf	Mycoplasma like organisms	+	+
16.	Cankerous stem gall	Unknown etiology	—	—
17.	Gummosis	"	—	—
18.	Mosaic	„	—	—
19.	Leaf curl	"	—	—
Non-infectious diseases				
	(i) Flourine injury		—	—
	(ii) Sulphur dioxide injury		—	—

stage. The extent of damage caused by these diseases depended upon the climatic conditions and the nursery practices.

Cylindrocladium spp. (*C*. quinqueseptatum, *C. ilicicola, C. theae, C*. *clavatum, C*.floridanum) also caused severe defoliation, branch die-back and mortality of 1- to 2-year-old plants in plantations situated in high rainfall areas of the State. The problem was more severe in plants where tapioca was grown as a taungya crop. Apparently the plants do not suffer much damage from the defoliation as they give rise to new flush after the monsoon. However, infection of branches and main stem, which often killed the plants upto 2-year-old and coppice shoots, was damaging. Such severe infection is facilitated by the prolonged high humidity due to incessant rains for 2 to 3 months. In this way Cylindrocladium which is a minor pathogen of eucalypts in Australia poses a serious problem in nurseries and plantations in Kerala.

Among other foliar pathogens, ***Phaeoseptoria eucalypti, Coniella*** spp. and ***Pestalotiopsis*** spp., are important which may cause severe defoliation during the monsoon and continue even afterwards.

Among the stem diseases, pink disease caused by ***Corticium salmonicolor*** and stem canker by ***Cryphonectria cubensis*** are the most serious ones. The pink disease affects 25 to 90 per cent plants in 2- to 3-year-old plantations of ***E. tereticornis,*** thus reducing the productivity to a considerable extent. The recent outbreak of pink disease in Wynad in ***E. grandis*** is also a matter of concern since this species was considered to be tolerant to ***Corticium salmonicolor.*** Contrary to earlier reports (Anon., 1982), the pink disease in ***E. grandis*** appears to be as destructive as in the case of ***E. tereticornis.***

Our pilot scale trial to control the pink disease was unsuccessful which clearly indicates that chemical control of this disease is not a viable solution. The only possible way to contain the disease within limits is raising disease resistant provenance(s) or species after laboratory screening and extensive field trials. With the toxin bio-assay standardized by us, it should be easier to select promising provenances of eucalypts because preliminary selection can be made in the laboratory itself.

Cryphonectria cubensis, a serious canker pathogen on ***E. saligna*** and ***E. camaldulensis:*** in Brazil, Surinam, Hawaii and Cuba has been recorded during the survey chiefly affecting ***E. grandis,*** though it was also found on some other species. The survey indicates that though the disease is not widespread, it has developed upto the stage of pink disease in the early seventies, possibly indicating the recent origin of this disease. The severity of infection varies greatly (0 to ca. 30 per cent) with the locality, host species, age of trees and also elevation. In high incidence areas like Wynad though the mortality of the trees is low (average 3.2%), possibly because of resistance in ***E. grandis,*** the disease situation needs to be monitored carefully so that if it develops on an epedemic scale, appropriate control measures can be taken up. Another species causing stem canker, ***Cryphonectria gyrosa,*** was also recorded in Kerala but its incidence was fairly low and it does not appear to pose a serious problem to the eucalypt species grown on large-scale.

On the whole though many pathogens have been found to cause diseases of ***Eucalyptus*** in Kerala, pathogens which cause serious diseases are only a few, while a few others appear to have a potential to become serious in due course of time. The serious pathogens are ***Rhizoctonia solani, Cylindrocludium*** spp **,** ***Corticium salmonicolor, Phaeoseptoria eucalypti, Cytospora euculypticola*** and ***Cryphonectriu cubensis.*** Of these the former three pathogens have wide host range. ***Cryphonectria cubensis*** which may become a problem as in Brazil is possibly indigenous with a wide geographical

distribution, as reported from several countries situated within 30°N and S of equator. *P. eucalypti,* known to occur in Australia and not recorded so far from any eucalypt growing country, may be an introduced pathogen from Australia. This is so because no alternate hosts of this pathogen are known in India, which inakes difficult its survival on hosts other than eucalypts.

It becomes clear that the indigenous pathogens with wide host range, which have adopted the introduced *Eucalyptus* in Kerala, have attained a status of serious pathogens. Besides the susceptibility of eucalypts to these pathogens in the new environment, the warm-humid climatic condition of Kerala has also played an important role in various kinds of fungi adopting eucalypts. In Australia also almost a similar situation exists where in spite of many fungi only a few of them viz. *Phytophthora cinnamomi* Rands and *Armillariella luteobubalina* Watling and Kile have emerged as serious pathogens, which have an extremely wide host range and Podger, 1972; Raabe, 1962).

DISCUSSION AND CONCLUSIONS

The present survey on the occurrence of diseases in *Tectona grandis, Bombax ceiba, Ailanthus triphysa, Gmelina arborea, Dalbergia latifolia, Eucalyptus* spp., and *Ochroma pyramidale* in Kerala was conducted systematically in representative plantations at regular intervals and in several nurseries at different stages of growth of each tree species. Additionally, disease problems not recorded during the survey but referred to the Institute by the Forest Department or observed by us in unscheduled visits to plantations were also investigated. This facilitated a comprehensive coverage of as many diseases as possible, whether of major or minor significance. Such knowledge is of great importance in the successful establishment of plantations.

Since the survey was carried out intensively, a number of diseases, majority of them of minor significance, which possibly would have gone unnoticed otherwise, were recorded. This resulted in the record of a total of 65 pathogenic and 13 other diseases (unknown etiology, non-infectious and angiospermic parasites) from all the tree species, maximum being 30 on *Eucalyptus* (Table 62). Altogether 88 pathogens were found to be associated with these diseases of which *Eucalyptus* had the maximum number of 46. Of the total pathogens recorded 64 are new host records. including seven new species viz. *Pseudoepicoccum tectonae* and *Phomopsis variosporum* on *T. grandis, Meliola ailanthi* on *A. iriphysa, Griphosphaeria gmelinae G. arborea, Physaiospora dalbergiae* on *D. latifolia* and *Cytospora eucalypti* and *Valsa eucalypticola* on *Eucalyptus* spp. While 29 are first record from India, majority of the pathogens are indigenous and already established on a number of other hosts. In this category the exceptions are a few pathogens of *Eucalyptus* such as *Cytospora eucalypticola* (recorded from South Africa and Australia), *Valsa eucalypti* (from Australia), *Cryphonectria cubensis* (from Brazil, Hawaii (USA), Puerto Rico, Surinam, Hong kong, Australia), *C. gyrosa* (from Cuba, Brazil, Australia), *Phaeoseptoria eucalypti* (Australia), and *Cylindrocladium floridaum* (USA) which have been recorded earlier from other countries. Among these *P. eucalypti* reported exclusively from Australia and observed in Japan (J. K. Sharma, unpublished observation) could be an introduced pathogen. For the others possibly it is a matter of chance that they are being recorded only now.

During its short rotation period of 8-10 years exotic *Eucalyptus* suffers from a large number of diseases showing its vulnerability to indigenous pathogens to which it has never been exposed to in its natural habitat. However, balsa, another exotic, there were only two diseases. This could be due to a number of factors such as its

general resistance to common pathogens, and its limited cultivation. As compared to ***Eucalyptus*** fewer diseases were recorded in indigenous tree species viz. ***T. grandis, B. ceiba, A. triphysa, G. arborea*** and ***D. latifolia*** with the exception of teak where of the 15 diseases, most of them were of foliage.

Table 62. Total number of diseases and their causal organisms recorded on various plantation trees during the survey in Kerala

Sl. No.	Tree species	Number of diseases (Total pathogens)				New pathogen record for the host	First record of pathogen from India
		Total	Nursery	Plantation	Common to nursery & plantation		
1.	*Tectona grandis*	15 (10)	2 (2)	14 (10)	1 (2)	6	4
2.	*Bombax ceiba*	8 (8)	4 (4)	6 (6)	2 (2)	4	—
3.	*Ailanthus triphysa*	9 (8)	8 (7)	4 (3)	3 (3)	8	2
4.	*Gmelina arborea*	10 (10)	3 (4)	8 (8)	1 (1)	10	3
5.	*Dalbergia latifolia*	4 (4)	3 (3)	4 (4)	3 (3)	4	1
6.	*Ochroma pyramidale*	2 (2)	0	2 (2)	—	2	1
7.	*Eucalyptus* spp.	30 (46)	13 (17)	21 (36)	4 (7)	30	18
	Total	78 (88)	33 (47)	59 (69)	13 (18)	64	29

High incidence of diseases was recorded in nursery due to increased proximity between host units and improper nursery practices which provide conducive microclimatic conditions, This is evident from the fact that disease incidence declines or disappears altogether after the stock is transplanted to the field. In nurseries facultative parasites such as ***Rhizoctonia solani*** and ***Sclerotium rolfsii*** have emerged as the main serious pathogens. Considering the serious mortality of seedlings of ***Bombax ceiba, Ailanthus triphysa*** and ***Eucalyptus*** caused by either one or both of these parasites, these ubiquitous pathogens cannot be neglected. Besides, ***Colletotrichum*** on ***Ailanthus*** and ***Gmelina*** and ***Cylindrocladium*** on ***Eucalyptus*** were also pathogens of serious concern as they caused considerable mortality of seedlings. In teak and

rosewood none of the diseases were found to be serious whereas in balsa no seedling disease was recorded.

Diseases in plantations are economically important as they affect wood production, both qualitatively and quantitatively. While outplanting of nursery stock to the field may be expected to be accompanied by a decline in the variety and the impact of diseases. the converse may also occur as age-related changes in host morphology and physiology make it susceptible to fresh parasites. In the plantations effect of monoculture is usually more pronounced in causing serious diseases of stem, especially canker diseases, than of foliage (Gibson and Jones, 1977). The present survey confirms these observations as the stem diseases like pink disease of ***Eucalyptus,*** die-back of teak, ***Gmelina*** and balsa were the most serious ones as they either killed the affected trees or retarded the growth considerably, Outbreak of pink disease in ***E. grandis*** is important as this species was earlier considered to be resistant . Other potentially serious diseases of eucalypts are stem canker caused by ***Cryphonectria*** and ***Cytospora.*** These diseases were already known to be of major significance in Brazil and South Africa, respectively Though the mortality caused by ***Cryphonectria*** canker in Wynad is only ca. 3% and much less in Mullachal, its occurrence in Kerala has to be taken seriously as it may spread in epidemic proportion as in Brazil after the building up of inoculum. Although ***E. grandis*** grown in Kerala does not appear to be as susceptible as ***E. saligna*** in Brazil, the disease needs to be monitored so that necessary steps may be taken should it pose threat to eucalypts. The other significant disease, ***Cytospora*** stem canker (caused by ***C. eucalypticola)*** which has caused heavy mortality in South Africa, was recorded in Wynad killing ca. 65 per cent of the coppiced shoots of ***E. tereticornis*** This species has not been found to cause mortality in plains, where ***E. tereticornis*** is the major species. Since planting of this eucalypt has been stopped in high elevations it is anticipated that due to unfavourable climate at low elevation ***C. eucalypticola*** will not spread in epidemic form. Nevertheless, considering its potential in killing trees outright this disease also needs surveillance. Botryodiplodia stem canker, a potentially serious disease in young plantations is mostly related to management practices. If the cultural practices are improved occurrence of this disease can be avoided considerably.

Beside eucalypts, pink disease was also recorded in teak, ***Bombax, Ailanthus*** and ***Gmelina.*** Except teak and ***Ailanthus*** where either the whole affected tree (latter species) or the top half of the main shoot (former) was killed outright, the other two hosts appeared to be resistant as no mortality was observed and the incidence of the disease was also insignificant. In teak a die-back disease caused by insect-fungus complex is a serious disease. Though it is localized in certain areas, it is capable of killing 20 to 30-year-old trees. Die-back of ***Gmelina*** caused by ***Griphosphaeria*** and of

balsa by two pathogens, ***Calonectria rigidiuscula*** and ***Fusarium moniliforme*** are also diseases of serious concern as they have caused large-scale mortality in certain plantations. In ***Ailanthus*** there was no serious disease as such but Botryodiplodia stem canker, also found to attack seedlings, is a potentially serious disease. Though it has been recorded only at a few places, it weakens and disfigures the form of the stem due to canker and epicormic shoots or kills the trees outright. This way both the growth of the tree and the productivity of the plantation may be greatly affected. Next in significance are foliar diseases such as caused by ***Cylindrocladium, Phaeoseptoria*** and ***Coniella*** in ***Eucalyptus, Phomopsis*** and ***Pseudoepicoccum*** in teak, ***Myrothecium*** in ***Bombax*** and ***Colletotrichum*** in ***Ailanthus*** which cause premature defoliation and hence retarding growth of plants.

From the above account it becomes clear that the maximum pressure of diseases is on ***Eucalyptus*** in nurseries as well as in plantations, which may account for their low productivity in Kerala. Planting of Eucalyptus in monoculture may not be alone responsible for the occurrence of these diseases, some of which have already caused epiphytotics. In this context the role of the tropical-humid climate of Kerala with high rainfall (Figs. 3 a, b) cannot be overlooked. As we know from the basic concepts of plant pathology for a disease to develop successfully it requires the combined effort of three factors i.e., susceptible host, virulent pathogen and favourable environment and we should not find fault with one of them separately for the present disease situation in eucalypts. This gets further support from the fact that no such disease problems of eucalypts as in Kerala have been reported from other states in India with camparatively low rainfall, even though the same pathogens are present there also and the same species of eucalypts are raised on large-scale. The possibility that these diseases remain undetected may be completely ruled out as diseases of such serious consequences cannot go unnoticed for long. In other plantation species such as teak, ***Ailanthus, Bombax, Gmelina,*** balsa and rosewood the number of diseases are much less. But they have one or two serious diseases, which can affect the productivity of the plantation, should they spread in epidemic. Table 63 lists various serious as well as potentially serious diseases of the seven tree species surveyed in the state.

With a wide spectrum of serious diseases in different hosts, affecting the stocking in the nursery, chemical control becomes inevitable. To a great extent the damage to seedlings can be minimised by following standard nursery practices and applying appropriate prophylactic fungicidal treatments. If the disease still persists due to certain reasons, chemical control is the only proposition as it is economically feasible at the nursery stage. Standard management practices for eucalypts have been outlined in detail earlier (Anon., 1984). Control measures for 18 seedling diseases

Table 63. Serious and potentially serious diseases of various tree species recorded during the survey in Kerala

Sl-No.	Tree species	Diseases of serious nature and their causal organisms		Potentially serious diseases and their causal organisms (nursery-N/plantation-P)
		Nursery	Plantation	
1.	*Tectona grandis*	None	1. Die-back *(Phialophora richardsiae)* 2. Mistletoe *(Dendrophthoe falcata)*	1. Pink disease (P) *(Corticium salmonicolor)* 2. Phomopsis leaf spot (P) *(Phomopsis variosporum)* 3. Leaf rust (N) *(Olivea tectonae)* 4. Unkown etiology (N)
2.	*Bombax ceiba*	1. Collar rot *(Rbizoctonia solani* 2. Seedling blight *(Sclerotium rotfsii)*	None	Myrothecium leaf spot (P) *(Myrothecium roridum)*
3.	*Ailanthus triphysa*	1. Collar rot *(Rhizoctonia solani)* 2. Seedling blight *(Colletotricbum dematium)*		1. Stem canker (P) *(Botryodiplodia theobromae)* 2. Pink disease (P) *(Corticium salmonicolor)* 3 Shot-hole (P) *(Colletotrichum gloeosporioides)*
4.	*Gmelina arborea*	Seedling blight *(Colletotrichum* state of *Glomerella cingulata* and *Fusarium solani)*	Die-back *(Griphosphaeria gmelinae)*	Stem infection (N) *(Pboma nebulosa)*
5.	*Dalbergia latifolia*	None	None	None
6.	*Eucalyptus*	1. Damping-off *(Rhizoctonia solani. Cylindrocladium* spp. *Pythium* spp. and *Fusarium oxysporum)* 2. Seedling blight (*Cylindrocladium* spp.)	1. Pink disease *(Corticium salmonicolor)* 2. Leaf and shoot blight *(Cylindrocladium* spp.1	1. Cryphonectria stem canker (P) *(Cryphonectria cubensis)* 2. Cytospora canker (P) *(Cytospora eucalypticola)* 3. Web blight (N) *(Rhizoctonia solani)* 4. Wilt (P) *(Fusarium oxysporum)* 5. Botryodiplodia stem (root collar) canker (P) *(Botryodiplodia theobromae)* 6. Phaeoseptoria leaf *(Phaeoseptoria eucalypti)*
7.	*Ochroma pyramidale*	None	Die-back *(Calonectria rigidiuscula* and *Fusarium moniliforme)*	None

in various host species were worked out during this investigation and field tested for the efficacy of the fungicide and its dosage.

Chemical control of diseases in forest plantations is infeasible considering the low returns and other technical problems. However, at some stage it becomes essential to try chemical control measures, either to check the epidemic spread of a serious disease or to control it temporarily till the time a permanent solution such as resistant provenances, species, etc. of a disease is worked out. At present the most serious disease problems among all the plantation species are in eucalypts, especially those of ***E. tereticornis.*** The pink disease, prevalent in low elevations affects the productivity of the plantations considerably. The results of a pilot-scale chemical control trial in Kothamangalam Forest Division, clearly indicates that reccurrence of pink disease cannot be checked even though we have very effective systemic fungicides such as Calixin. In such case the only alternative left is either to continue the chemical treatment for at least first three years at which stage the plants are highly susceptible or switch over to other provenances or species of ***Eucalyptus*** having at least some degree of resistance to pink disease. With the view to find out resistant eucalypts for achieving a long-term solution of pink disease a bio-assay technique for screening eucalypt provenances in laboratory was standardized. Of the 23 provenances belonging to 11 species of ***Eucalyptus*** a few gave moderately resistant to moderately susceptible reaction. It is evident from the results that moderate resistance to pink disease in eucalypts exists even under intensive laboratory screening, which may prove to be quite promising under field trials.

REFERENCES

Anon. 1937. Report of the Director of the Avros Experimental Station, 1 August 1935 - 30 June 1936 and 1 July 1936 - 31 December 1936. Meded. alg. Proj. Avros Alg. Ser. 58.

Anon. 1955. Eucalypt for planting. FAO Forest Prod. Stud. No. 11 (Translated from French).

Anon. 1958a. Rep. Dep. Agric. Maurit. 1958.

Anon. 1958b. Principal pests and diseases of eucalypts outside Australia. Unasylva. Rome. 12.

Anon. 1982. *Eucalyptus grandis* one of the most suitable pulpwood species for Kerala. paper presented at the Forest Convention, Trivandrum. 8p.

Anon. 1983. Administration report of Kerala Forest Department for the year 1978-1980, Government Press, Ernakulam.

Anon. 1984. Nursery diseases of *Eucalyptus* in Kerala and their control. Kerala Forest Research Institute Information Bulletin 6, 16p.

Anon. 1985. Administration report of Kerala Forest Department for the year 1984-1985, Government Press, Ernakulam.

Agnihothrudu, V. 1962 Outbreaks and new records. Two species of *Pellicularia* parasitic on *Albizia falcata* in Assam. FAO Plant Prot. Bull. 10: 143-145.

Alfenas, A. C., K. Marsuoka, F. A. Ferreira, and C.S. Hodges. 1979. Identificao, caracteristicas Culturais e patogenicidade detres especies de *Cylindrocladium* isoladas de manchas de folha de *Eucalyptus* spp. Fitopatologia Brasileira 4: 445-459.

Al-Zarari, A. J., A. A. Attrackchi, A. M. Tarablib and S. H. Michail 1979. New hosts for *Hendersonula toruloidea.* Pakistan J. of Scientific and Industrial Research 22 (5): 251.

Arruda, S. C. 1913. Observacoes Sobre algumas doencas do eucalypto no estado de S. Paulo. Biologico 9: 140-144.

Arx, J. A. Von. 1957. Die Arten der Gattung *Colletotrichum.* Phytopath. Z. 29: 413-468.

Autter, S. H. 1943. Seven diseases and pests of forest nurseries including two of *Pinus radiata.* Bolon. For. Lat. Am. invest. Capacit. Merida 15: 3-59.

Aycock, R., 1966. Stem rot and other diseases caused by *Sclerotium rolfsii.* North Carolina Agricultural Experiment Station, Technical Bulletin No. 174, 202p.

Azevedo, N. F. dos S. de. 1971. Forest tree diseases. Laboratorio de Patologia Florestal Secretaria de estado da Agricultura. Oeiras, Portugal, 10-33.

Bagchee, K. 1952. A review work on Indian tree diseases and decay of timber and methods of control. Indian Forester 73: 540-546.

Baker, K. F. 1970. Types of *Rhizoctonia* diseases and their occurrence. Page 125. In: J. R. Parmeter Jr., ed. *Rhizoctonia solani:* Biology and Pathology. University of California Press, Berkely.

Bakshi, B. K. 1967. Quantification of forest disease losses. Report from Asia for XIV IUFRO Congr. Munich 1967. XIV Congr. Int. Un. For. Res. Org. Munich 1967, 5: 361-372.

Bakshi, B. K. 1972. Gummosis in *Eucalyptus* Indian Forester 98: 647-648.

Bakshi, B K. 1975. Forest Pathology. Principles and Practice in Forestry. Controller of Publications, Delhi. 400 pp.

Bakshi, B. K. and J. S. Boyce 1958. Water blister in teak. Indian Forester 85: 589-591.

Bakshi, B. K. and Sujan Singh 1964. Mortality of *Eucalyptus citriodora* Hook. Indian Forester 90: 15-18.

Bakshi, B K. and Sujan Singh 1967. Rusts on Indian Forest trees Indian Forest Rec. (N. S.). Forest Pathology 2: 139-198.

Bakshi, B. K., M. A R. Reddy, Y. N. Puri and Sujan Singh 1972. Forest disease survey (Final Technical Report). Forest Pathology Branch, F. R. I., Dehra Dun. 117p.

Barr, M. E. 1978. The Diaporthales in North America with emphasis on *Gnomonia* and its segregatzs. Mycol. Mem. 7: 1-232.

Batista, A. C. 1951. *Cylindrocladium scoparium* Morgan var. *brasiliensis* Batista and Ciferri em novo fungo do eucalypto. Boln. Sec. Agric. Ind. Com. Pernambuco 18: 188-191.

Baudin, P. 1956, Maladies parasitaires das Ignenies en Cote d'Ivoire. Rev. Mycol., Paris, 21 (Suppl. Colon 2): 87-1 11.

Bazan de Segura, C. 1969. *Corticium salmonicolor* and *Pellicularia koleroga* on various species of *Eucalyptus* in Turrialba. Turrialba 20: 254-255.

Beeson, C. F. C. 1941. The Ecology and Control of the Forest Insects of India and the Neighbouring Countries. Yasant Press, Dehra Dun. 1007p.

Behnke, M. 1979. Selection of potato callus for resistance to culture filtrates of *Phytophthora infestans* and regeneration of resistant plants. Theor. Appl. Genet. 55: 69-71.

Behnke, M. 1980. General resistance to late blight of *Solanum tuberosum* plants regenerated from callus resistant to culture filtrates of *Phytophthora infestans.* Theor. Appl. Genet. 56: 151-152.

Benito, M. J. 1957 The formation of a crown gall type of tumour on eucalypts cultivated in Spain. Bolon. Inst. For. Invest. Exp. Madrid 28: 76.

Berkeley, M. J and C. E. Broome 1875. Enumeration of the fungi of Ceylon. J. Linn. Soc. 14 (2): 29-140.

Bernard, C. 1908. Ziekten der thee planten. Dept. Land bouw. 1l.

Boerboom, J. H A. and P. W. T. Maas 1970. Cankers of *Eucalyptus grandis* and *E. saligna* in Surinam caused by *Endothia havanensis.* Turrialba 20: 94-99.

Booth, C. 1966. The genus *Cylindrocarpon.* Mycological Paper No. 104, Common wealth Mycological Institute, Kew, Surrey, England. 56p.

Booth, C. 1971. The genus *Fusarium.* Commonwealth Mycological Institute, Kew, Surrey, England. 237p.

Borum, D. E. and J. B. Sinclair 1968. Evidence for systemic protection against *Rhizoctonia solani* by systemic and non-systemic fungicides. Ann. appl. Biol. 82: 267-268

Browne, F. G. 1968 Pests and Diseases of Forest Plantation Trees. Clarendon Press, Oxford. 1330p.

Browne, C. E. and A. G. Davidson 1968. Forest Insect and Disease Survey. Data recording and retrieval manual. Forestry branch, Department of Fisheries and Forestry, Ottawa, Canada. 71p.

Bruner, **S.** C. 1917. Una Enfermidad Gangrenosa de les Eucaliptos. Boleteni No. 37, Estacion Experimental Agronomica, Santiago de las Vegas, Cuba. 38p.

Butler, E J. and G. R. Bisby 1960. The Fungi of India. Revised by R. S. Vasudeva. Indian Counc. Agric. Res., New Delhi. 552p.

Chandrasrikal, A. 1952. A preliminary host list of plant diseases in Thailand. Tech. Bull. Dept. Agric. Bangkok 6, 23p.

Christensen, C. M. 1973. Loss of viability in storage microflora. Seed Sci. and Tech. 1: 547-562.

Cockerill, J. 1955. The use of Thiram as a control for damping-off of red pine. Canada Dept. Agr., For. Biol. Div., Bi-Mon. Prog. Pap. 11 (4): 1

Cook, M. R. 1913. Diseases of Tropical Plants. London.

Corden, M. E. and R. E. Young 1962. Evaluation of eradicant soil fungicides in the laboratory. Phytopathology 52: 503-509.

Cox, R. S 1954. *Cylindrocladium scoparium* on conifer seedlings. Bulletin No. 301.

(Technical) Univ. of Delaware, Agric. Exp. St., Newark, Delaware, U. S. A., 40p.

Davison, E. M. 1982. *Endothia havanensis* on jarrah. Australas. Plant Pathol. 11: 10-11.

Davison, E. M. and F. C. S. Tay 1983. Twig, branch and upper trunk cankers of *Eucalyptus marginata.* Plant Disease 67: 1285-1287.

Deeley, J., W. A. Stevans, R. T. V. Fox 1979. Use of Dienes' stain to detect plant disease induced by Mycoplasma like organisms. Phytopathology 69: 1169-1171.

Dennis, R. W. G. 1981. British Ascomycetes. J. Cramer. 555p.

Doo, S. C. 1968. Bacterial wilt of teak seedlings. Union of Burma, J. of Life Sciences 1: 43.

Ellis, M. B. 1957a. Some species of *Corynespora.* Mycol. Pap. 65: 1-15.

Eusebio, A. M., F. P. Ilagan and M. J. Quimio Jr. 1980. Infection trend and control of canker of Molluccan Sau *(Albiziafalcataria* (L.) Back.) in Bislig, Surigao del Sur. Sylvatrop Philipp. For. Res. J. 5 (2): 99-122

Fergus, C. L. 1957. *Myrothecium roridum* on Gardenia. Mycologia 49: 124-127.

Fernandez Valida, M. V., M. Bakarcic and A. Turica 1954. Manual of fruit and forest tree diseases and pests in the delta of the Parna. Publnes. Minst. Agric. Ganad. Republica Argent. B. Aires.

F. A. and A. C. Alfenas 1977. Pink disease in *Eucalyptus* spp. caused by *Corticium salmonicolor* Berk. and Br. in Brazil. Fitopatologia Brasileira 2: 109-115.

Ferreira, F. A., A. C. Alfenas and A. L. de Freitas 1978. Determinacao da resistencia de 16 procedencias de *Eucalyptus* ao cancro causado por *Diaporthe cubensis* Bruner, no Vale do Rio Doce. Re Vista Arvore 2: 119-129.

Ferreira, F. A., M. S. Reis, A. C. Alfenas and C. S. Hodges 1977. Avaliacaoda resistencia de *Eucalyptus* spp. ao cancro causado por *Diaporthe cubensis* Bruner. Fitopatologia Brasileira 2: 225-241.

Figueiredo, M. B. and B. P. M. Curz 1963. Occurrence of *Cylindrocladium ilicicola* on *Eucalyptus* spp. in the state of Sao Paulo. Arg. Inst. biol. S. Paulo 30: 29-32.

Figueiredo, M. B. and T. Namekata 1967. Constalacao de *Calonectria quinqueseptata* n. sp. forma perfecta de *Cylindrocladium quinqueseptatum* Boedijn and Reitsma, sobre *Annona squamosa* L. e *Eucalyptus* spp. Arg. Inst. biol. S. Paulo 34: 91-96.

Gallagher, W. J. 1909. A preliminary note on a branch and stem disease of *Hevea brasiliensis.* Dept. of Agr., Federated Malay States Bull. 6, 6p.

Garces, O. 1964. Las enfermedades de 1os arboles forestales en la America Latina y su impacto en la produccion forestal. FAO/IUFRO Symp. int. dang. For. Dis. Insects, Oxford. 1964.

Ghosh, S, K., M. Balasundaran and M. I. Mohamed Ali 1984. Studies on the host-parasite relationship of phanerogamic parasite (s) on teak and their control. Kerala Forest Research Institute Research Report 21. 39p.

Ghosh, S. K., M. I. MohamedAli and M. Balasundaran 1985. Light and fluorescent microscopic studies on little leaf disease of Eucalyptus. Phytopath. Z. 110. 207-212.

Gibson, I. A. S. 1956. Sowing density as damping-off in pine seedlings. E. Afr. Agric. For. J. 21: 183-188.

Gibson, I. A. S. 1967. Influence of disease factors on forest production in Africa. Proceedings XIV Congrees of the International Union of Forest Research Organisation, Munich 5: 327-360.

Gibson, I. A. S. 1975. Diseases of Forest Trees Widely Planted as Exotics in the Tropics and Southern Hemisphere. Part I. Important members of the Myrtaceae, Leguminosae, Verbenaceae and Meliaceae. Department of Forestry, Oxford University and Commonwealth Mycological Institute, Kew. 51p.

Gibson, I. A. S. and F. B. Armitage 1979. Mission Report on Disease Problems in Eucalypt Plantations in Kerala State, India. Food and Agricultural Organization, Rome. 29p.

Gibson, I. A. S., and Jones, T. 1977. Monoculture as the origin of major forest pests and diseases. Pages 139-1961. In: J. M. Cherrett and G. R. Sagar, eds. Origin of Pest, Parasite, Disease and Weed Problems, Blackwell Scientific Publication, Oxford. 413p.

Goldthwaite, J. I and W. M. Laetsch 1968. Control of senescence in Rumex leaf discs by gibberellic acid. Plant Physiol. 43: 1855-1858.

Griffin, D. M. 1958. Influence of pH on the incidence of damping-off. Trans. Br. Mycol. Soc. 41: 483-490.

Grossmann, F., and D. Steckhan 1960. Nebenwerkungen ciniger Insektizide auf pathogene Bodenpilze. Zeitschr. Pflanzenkr. Pflanzenschutz 67: 7-19.

Hansford, C. G. 1957. Phaeoseptoria eucalypti Hansf., n. sp. Proc. Linn. Soc. N. S. W., 82: 225.

Hawkins, L. A. and P. B. Harvey 1919. Physiological study of the parasites of Pythium debaryanum Hesse on potato tubers. J. agric. Res. 18: 275-297.

Heather, W. A. 1965. Ph. D. Thesis, Australian National University, Canberra. 366p.

Hebert, T. T. and C. N. Clayton 1963. Pomegranate fruit rot caused by granati. Plant. Dis. Reptr. 47: 222-223.

Hepting, G. H. 1971. Diseases of Forest and Shade Trees of the United States. USDA Handbook No. 386, 658p.

Hewitt, W. 1977. Microbiological assay. An introduction to quantitative principles and evaluation. Academic Press, New York.

Hilton, R. N. 1958. The pink disease of *Hevea* caused by *Corticiunz salmonicolor* Berk. and Br. J. Rubb. Res. Inst. Malaya 15: 275-292.

Hodges, C. S. 1980. The taxonomy of *Diaporthe cubensis.* Mycologia 72: 542-548.

Hodges, C. S., T. F. Geary and C. E. Cordell 1979. The occurrence of *Diaporthe cubensis* of *Eucalyptus* in Florida, Hawaii and Puerto Rico. Plant Dis. Reptr. 63: 216-220.

Hodges, C. S. and L. C. May 1972. A root disease of pine, *Araucaria* and *Eucalyptus* in Brazil caused by a new species of *Cylindrocladiurn.* Phytopathology 62: 898-901.

Hodges, C. S. and M. S. Reis 1974. Identificaco do fungo causador de cancro de *Eucalyptus* spp. no Brasil. Brasil Florestal 5: 19.

Hodges, C. S., M. S. Reis, F. A. Ferreira and J. D. M. Henfling 1976. O cancro do eucalipto causado por *Diaporthe cubensis.* Fitopatologia Brasileira 1: 129-170.

Hsieh, H. J. 1980. Survey of disease of woody plants in Taiwan (3). Quarterly Journal of Chinese Forestry, 13 (3): 129-139.

Kalshoven, L. G. E. 1928. The injuries, diseases and pests of the teak forests of Java. Tectona 21: 593-623.

Karunakaran, C.K. 1982. A perspective plan for the period 1982-83 to 1975-96 on demand versus supply of important raw materials from forests in Kerala State. Kerala Forest Department, Trivandrum. 44p.

Kataria, H. R. and R. K. Grover 1976. Fungitoxicity of mineral oil against *Rhizoctonia solani* causing damping-off of inungbean *(Phaseolus aureus)* Ann. Appl Biol. 83: 79-85.

Kataria, H. R. and R K. Grover 1978. Comparison of fungicides for the control of *Rhizoctonia solani* causing damping-off of mungbean *(Phaseolus aureus).* Ann. appl. Biol. 88: 257-263.

Khan, A. H. 1951. Some diseases observed in teak plantations of the Punjab. Pakist. J. Sci. Res. 7: 92-99.

Kiely. 1949. Proc. Linn. Soc. N. S. W. 73: 259.

Kobayashi, T. 1978. Notes on the Philippine fungi parasitic to woody plants (1). Trans. Mycol. Soc. Japan 19(4): 373-381.

Kobayashi, T. and K. Ito 1956. Notes on the genus *Endothia* in Japan. I. Species of *Endothia* collected in Japan. Bulletin of the Government Forest Experiment Station, Meguro 92: 87-97.

Kristic, M. 1964. Cankers of forest trees. FAO/IUFRO Symp. int. dang. For Dis. Insects, Oxford.

Krupinsky, T. M. 1982. Growth and sporulation of *Botryodiplodia hypodermia* in response to different agar media and temperatures. Plant Disase 66 (6). 481-483.

Kulkarni, S., and A. L. Siddaramaiah 1979. Chemical control of powdery mildew of teak. Current Research 8: 192-193.

Leach, L. D. 1947. Growth rates of host and pathogen as factors determining the severity of pre-emergence damping-off. J. agric. Res. 75: 161-179.

Leach, L. D. and R. H. Garber 1970. Control of *Rhizoctonia.* Pages 189-198, In: J. R. Parmeter, ed. *Rhizoctonia solani.* Biology and Pathology. University of California Press, Berkely.

Lehman, S. G., Murakishi, H. and Goaham, J. H. 1951. A leaf spot of Soybean caused by *Sclerotium rolfsii.* Plant. Dis. Reptr. 35: 167-168.

Magnani, G. 1964. Diseases of *Eucalyptus.* Pages 159-167. In: Diseases of widely Planted Forest Trees. FAO/FORPEST 64, Oxford.

Massee, C. 1898. Fungi exotici. Kew Bull. NO. 119: 138 p.

May, L. C. 1973. A gomose de eucalipto no Brasil. Publicado I. F. No. 2, Instituto Florestal do Estado de Sao Paulo. 11p.

McClure, T. T. 1949. Phytopathology 39: 876-886.

McClure, T. T. 1950. Phytopathology 40: 769–775.

McClure. T. T. and W. R. Robbins. 1942. Resistance of cucumber seedlings to damping-off as related to age, season of year and level of nitrogen nutrition. Bot. Gaz. 103: 648-697.

McGuire, J. M. and W. E. Cooper. 1965. Interaction of heat injury and *Diplodia gossypina* and other etiological aspects of collar rot. Phytopathology 55: 231-236.

Meiffren, M. and M. Belin 1960. Cafe - Cacao - The 4: 150-158.

Mitchell, B. A. 1962. Bacterial wilt in teak, *Tectona grandis* Linn. Malay. Forester 25: 164-166.

Mitchell, R. E. 1984. The relevance of non-host specific toxin in the expression of virulence by pathogens. Ann. Rev. of Phytopath. 22: 212-245

Mordue, J. E. M. 1971. *Glomerella cingulata.* Set 32, No. 315. Descriptions of pathogenic fungi and bacteria. Commonwealth Mycological Institute, Kew, Surrey, England.

Mordue, J. E. M. and I. A. S. Gibson 1976. *Corticium salmonicolor.* Descriptions of pathogenic fungi and Bacteria No. 511. Commonwealth Mycological insititute Kew, England, 2p.

Nair, K. S. S. and R. V. Varma 1981. Termite control in eucalypt plantations. Kerala Forest Research Institute Research Report 6. 48p.

Natarajan, K. and B. Manjula 1976. Studies on *Lentinus squarrosulus* Lev. Pages 452-456. In: C. K. Atal, B. K. Bhat and T. N. Kaul eds. Indian Mushroom Science.

Newhook, F. J. and F. D. Podger 1972. The role of *Phytophthora cinnamomi* in Australian and New Zealand forests. Ann. Rev. Phytopathol. 10: 229-326.

Padaganur, G. M. and P. C. Hiremath 1973. *Phaeoseptoria eucalypti* Hansf a new record to India. Mysore. Agric. Res. J. 336-338.

Pawar, V. H , and M. J. Thirumalachar 1970. Mechanism of parasitism in *Myrothecium roridum* Tode. Pages 73-178. In: S. P. Raychaudhury *et al.*, eds. Plant Disease Problems. Proceedings of First International Symposium on Plant Pathology, Indian Phytopathological Society, New Delhi.

Petch, T. 1911. Physiology and diseases of *Hevea brasiliensis.* Macmillian and Co. Ltd., London. 209p.

Petch, T. 1921. The Diseases and pests of the rubber tree. Macmillian and Co, Ltd., London. 278p.

Piening, L. J. 1962. A checklist of fungi recorded in Ghana. Bull. Minist. Agric. Ghana No. 2.

Pretson, N. G. 1936. The parasitism of *Myrothecium roridum* Tode. Trans. Br, mycol. Soc. 20: 242-251.

Punithalingam, E. 1980. Plant diseases attributed to *Botryodipfodia theobromae* Pat. Bibliotheca Mycologica, Vaduz: Cramer, Band 11, 121p.

Raabe, R. D. 1962. Host list of the root-rot fungus, *Artnillaria mellea* Hilgardia, 33: 25-88.

Raciborski, M. 1900. Parasitischen Algen und Pilze Java's teil. 1: 28.

Radhakrishnapillay, P. N. and M. K. George 1981. Pink disease of rubber. Rubber Board Bull. 19: 8-10.

Rahaman, M. U., K. V. Sankaran, K. M. Leelavathy and S. Zachariah 1981. *Cy1indrocladium* root rot of nutmeg in South India. Plant Disease 65: 514-515.

Ramakrishnan, T. S. and K. Ramakrishnan 1949. *Chaconia tectonae* Ramakrishnan, T. S. and K. sp. nov. on teak. Indian Phytopath. 2: 17-19.

Ram Reddy, M. A. 1969. Damping-off in conifer nurseries in India. Indian Forester 95(7) : 475-479.

Rant, A. 1911. De djamoer oepas ziekte in het algemeen en bij kina in het bijzonder. Med. Dept. Landbouw. No. 13, Batavia.

Reddy, S. M. 1973. Perithecial stage of *Cylindrocladium ilicicola* Boed. and Reit. Curr. Sci. 43: 57-58.

Reddy, S. M. 1975. Some new leaf spot diseases caused by hyphomycetes. Proc. Nat. Acad. Sci. India, Sect. B, 45(2): 97-100.

Reis, M. S. and C. S. Hodges 1975. Status of forest diseases and insects in Latin America. FAO/FORPEST, D1/75.

Reitsma, J. and W. C. Sloof 1950. Rolf's sclerotium disease on *Hibiscus sabdariffa* L. var. Victor. Cont. of the Gen. Agr. Res. Sta., Bogor, Indonesia. 109: 27-33.

Roberts, M. and C. B. C. Boyce 1972. Principles of biological assay. Pages 153-190. In: J. R, Norris and D. W. Ribbons, eds. Methods inMicrobioIogy, Vol. 7A. Academic Press, New York.

Roldan, Emiliano F. and Primo P. Andres 1953. Bacterial wilt of teak seedlings *(Tectona grandis* Linn.). Phill. J. For. 9: 133-144.

Roth, L. F. and A. J. Riker 1943. Life history and distribution of *Pythium* and *Rhizoctonia* in relation to damping-off of red pine seedlings. J. agric. Res. 67: 129-148.

Rowan, S. J., T. H. Filer and W. R. Helps 1972. Nursery diseases of Southern hardwoods. USDA, Forest Pest Leaflet No. 137: 7p.

Rudolph, K. 1976. Non-specific toxins. Pages 170-315. In: R. Heitefuss and P. H. Williams, eds. Encyclopedia of Plant Physiology, New Series, Vol. 4, Physiological Plant Pathology, Springer-Verlag, Berlin and New York.

Saccardo, P. A. 1883. Sylloge Fungorum 2: 594.

Sastry, K. S. M., R. N. Thakur, J. H. Gupta and V. R. Pandotra 1971. Three virus diseases of *Eucalyptus citriodora.* Indian Phytopath 24: 123-126.

Scharif, G. 1964. Report on forest diseases in near and middle East. FAO/IUFRO Symp. int. dang. For. Dis. Insects, Oxford.

Scheffer, R. P. 1976. Host specific toxins in relation to pathogenesis and disease resistance. Pages 247-269. In: R. Heitefuss and P. H. Williams, eds. Encyclopedia of Plant Physiology, Vol. 4, Springer - Verlag, Berlin and New York.

Scheffer, R. P. and S. P. Briggs 1981. Introduction. A perspective of toxin studies in Plant Pathology. Pages 1-17. In: R. D. Durbin, ed. Toxins in Plant Disease. Academic Press, New York.

Schwarz, M. Beatrix 1925. Pink disease of teak. Meded. Inst Voor Piantenziekten 68:17p.

Sebastian, S., Quniones, and Maria P. Dayan 1981. Notes on the diseases of forest species in the Philippines. Sylvatrop Philip. For. Res. J. 6(2): 61-68.

Sehgal, H. S., M. J. Nair and S. J. Stanley 1978. Occurrence of *Cylindrocladium quinqueseptatum* Boedijn and Reitsma as a parasite of *Eucalyptus grandis* Hill ex Maiden and *Eucalyptus tereticornis* Sm. *(E.* hybrid or Mysore gum) in India. Southern For. Rangers Coll. Magazine, 62-65.

Seth, S. K., B. K. Bakshi, M. A. R. Reddy, and Sujan Singh 1978. Pink disease of *Eucalyptus* in India. Eur. J. For. Pathol. 8: 200-216.

Sharma, J. K. and C. Mohanan 1981b. Chemical control of *Cylindrocladium* causing damping-off, seedling and shoot blights of *Eucalyptus* in nursery. Paper presented at XI1 IUFRO World Forestry Congress, Kyoto, Japan, Sept. 1981, 10 p.

Sharma, J. K. and C. Mohanan 1982. *Cylindrocladium* spp. associated with various diseases of *Eucalyptus* in Kerala. Eur. J. For. Path. 12 (3): 129-136.

Sharples, A. 1936. Diseases and pests of the rubber tree. MacMillan and Co. Ltd., London, 480p.

Singh, S. and R. K. Tewari 1970 Role of a precursor fungus in decay in standing teak. Indian Forester 69: 874-876.

Sobers, E. K. and C. P. Seymour 1967 *Cylindrocladium floridanum* sp. n.associated with decline of peach trees in Florida. Phytopathology 57: 382-393.

Sommer, N. F. 1955. Sunburn predisposes walnut trees to branch-wilt infection. Phytopathology 45: 607-613.

Spaulding, P. 1961. Foreign diseases of forest trees of the world. Agric. Handbook 197, U. S. Dept. Agric. 361p.

Subramanian, C. V. 1952. Fungi imperfecti from Madras-1. Proc. Indian Acad. Sci. B 34: 43-53.

Sujan Singh and B. K. Bakshi 1964. Notes on some Indian tree rusts. Indian Forester 90(7): 469-472.

Sutton, B. C. 1980. The Coelomycetes. Commonwealth Mycological Institute, Kew, Surrey, England. 696p.

Tandon, R. N. and S. Chandra 1963-1964. Supplement to the list of Indian Fungi (1957-1962). University of Allahabad Publications, Senate House, Allahabad. 246p.

Taneja, M. and R. K. Grover 1982. Efficacy of benzimidazole fungicides against *Rhizoctonia solani* and *R. bataticola*. Ann. appl. Biol. 100: 425-432.

Taubenhaus, J. J. 1935. On a black crown rot of greenhouse snapdragons caused by *Myrothecium roridum* Tode. Phytopathology 25: 969-972.

Tereshita, T. and T. Takai 1955. Some notes on *Cylindrocladium scoparium* in Japan Bull. Govt. For. Exp. Stn. Meguro 87: 33-47.

Thirumalachar, M. J. 1950. Telia of the leaf rust on teak. Curr. Sci. 18: 175-177.

Thresh, J. M. 1960 Emp. J. Exp. Agric. 28: 193-200.

Tiwari, D. P., A. K. Rajak and Ku. M. Nikhra 1981. A new species of *Phomopsis* causing leaf spot disease on *Tectona grandis* L. Curr. Sci. 50: 1002-1003.

Troup, R. S. 1921. The Silviculture of Indian Trees. Vol. 11. Clarendon Press, Oxford. 783p.

Vaartaja, O. 1952. Forest humus quality and light conditions as factors influencing damping-off. Phytopathol. 42: 501-506.

Vaartaja, O. and W. H. Cram 1956. Damping-off pathogens of conifers and of Caragana in Saskatchewan. Phytopathol. 46: 391-397.

Vaartaja, O. and G. A. Morgan 1961. Damping-off etiology especially in forest nurseries. Phytopathology 51: 35-42.

Van der Westhuizen, G. C. A. 1965 a. A disease of young *Eucalyptus saligna* in northern Transvaal. J. S. Afr. For. Assn. 33: 53-56.

Van der Westhuizen, G. C. A. 1965 b. *Cytospora eucalypticola*. sp. nov. in *Eucalyptus saligna* from northern Transvaal. J. S. Afr. For. Assn. 54: 8-11.

Varma, K. S. and R. L. Munjal 1980. Studies on the development of necator state of *Corticium salmonicolor* causing pink disease of apple. Indian Phytopath. 33(3): 486-488.

Vasudeva, R. S. 1963. Indian Cercosporae. Indian Counc. Agric. Res., New Delhi. 245p.

Venkataramani, K. S. 1951. Report of the Botanist 1950-51. Ann, Rep. Sci. Dep. Uni. Pl. Ass. S. India 1950-51: 22-27.

Venkataramani, K. S. 1952. A tea root disease new to South India. Nature 169: 1099- 1100.

Venkataramani, K. S. and C. S. Venkata Ram 1964. A new species of *Cylindrocladium* parasitic on tea roots. Curr. Sci. 30: 186.

Wei, C. T. 1950. Notes on *Corynespora*. Mycol. Pap. 34: 1-10.

Weir, J. R. 1976. A pathological survey of the para rubber tree *(Hevea brasiliensis)* in the Amazon Valley. U. S. Dept. Agr Bull 1380: 1-130.

West, E. 1936 *Sclerotium rolfsii* in Florida - factors influencing its pathogenicity Florida Agr. Exp. Sta. Ann. Rep. for year ending June 30, 1936. 92-93.

Yoder, O.C. 1973. A selective toxin produced by *Phyllosticta maydis*. Phytopathology 63: 1361-1366.

Yoder, O.C. 1981, Assay. Pages 45-71. In: R. D. Durbin, ed. Toxins in Plant Disease. Academic Press, New York.

Zentmeyer, C. A. 1955. A laboratory method for testing soil fungicides with *Phytophthora cinnamomi* as test organism. Phytopathology 45: 308-404

APPENDIX-I

LIST OF FUNGICIDES EVALUATED AGAINST VARIOUS PATHOGENS

Sl. No.	Trade name	Common name	Chemical name	Remarks
1.	Bavistin	Carbendazim	Methyl-1 H-benzimidazole-2-yl-carbamate	BS, S
2.	Benlate	Benomyl	Methyl-1-(butylcarbamoyl)-2-benzimidazole carbamate	BS, S
3.	Bayleton	Triadimefon	I-(4-chloro-phenoxy)-3, 3-dimethyl-1-(1 H-1, 2, 4-triazol-l-yl)-2-butanone	S
4.	Calixin	Tridemorph	N-tridecyl-2, 6-dimethylmorpholine	S
5.	Daconil 2781	Chlorothadonil	Tetrachloroisophthalonitrile	BS
6.	Demosan	Chloroneb	1,4-dichloro-2, 5-dimethoxybenzene	S
7.	Difolatan	Captafol, Foltaf	Cis-N-(1,1,2,2,-tetrachloro ethylthio)-4-cyclohexane-l,-2-dicarboximide	FOL
8.	Dithane M-45	Mancozeb	Manganese ethylenebisdithiocarbamate+ Zn ions	BS, FOL
9.	Dithane 2-78	Zineb	Zinc ethylenebis (dithiocarbamate)	BS, FOL
10.	Emisan-6	—	2-methoxyethylmercuric chloride	SF
11.	Furmetamid	BAS 389	2,5-dimethyl-N-eoclohexyl-N-methoxy-3-flurancarboxamide	FOL
12.	Fytolan	—	Copper oxychloride-3 $Cu\,cl_2$ $Cu(0H)_2$	FOL, SF
13.	Hexacap	Captan	N-(trichloromethylthio)-4-cyclohexane-1,2-dicarboximide	FOL, SF
14.	Hexathir	Thiram, Thiride	Tetramethyl thiuram disulphide	FOL, SF
15.	"Vegfru" Kitazin-48	Kitazin	S-benzyl-0, 0-diisopropyl-phosphothionate	FOL
16.	Polyram Combi	Metiram	Zinc activated polyethylene thiuram disulphide	BS
17.	Saprol	Triforine	N, N'-Piperazinediyl-bis-(2,2,2-trichloro. ethylidene) 1-bis-formamide	S
18.	Syllit 65	Dodine	Dodecylguanidine acetate	FOL
19.	Tecto	Thiabendazole	2-(4-thiazolyl) H-benzimidazole	FOL
20.	Vitavax	Carboxin	5,6-dihydro-2-methyl-l, carboxani lide	S

BS—Broad spectrum; FOL—Foliar spray; S—Systemic; SF—Soil fungicide

APPENDIX- II

CULTURAL AND MORPHOLOGICAL CHARACTERS OF FUNGAL AND BACTERIAL PATHOGENS

TECTONA GRANDIS

1. Acremooiurn recifei (Leao & Lobo) W. Gams. IMI (284045); hyperparasite on Olivea tectonae.

Colony on PDA white, cottony; conidiophores slender, hyaline; conidia elliptical, hyaline, septate. 3.3-8.8 x 2.2-3.3 µm.

2. Cladosporium oxysporum Berk. & M. A. Curtis (IMI 284044).

Colony on PDA olive green; conidiophores dark, upright, conidia one-celled, ovoid to cylindrical 2.2-8.8 x 3.3 µm.

3. Colletotrichum state of Glomerella ciogulata (Stonem.) Spauld. & Schrenk (IMI 246479, 246480)

Colony on PDA off-white to greyish with aerial mycelium; acervuli, develop after a week, disc shaped with characteristic marginal black setae; conidial mass pink, conidia oblong, guttulate with slight constriction in the middle, 12.0-15.0 x 3.1-3.7 µm.

4. Marasmiellus ignobilis (Berk. & Br.) Pegler

The fructifications have a centrally depressed, smooth, white to pale cream, non-gelatinous pileus; gills soft deeply decurrent; stalk excentric to lateral; spores minute, hyaline.

5. Olivea tectonae (T. S. & K. Ramakr.) Mulder (IMI 273439): It is a micro-cyclic rust known only in telial and uredinial stages on teak.

Uredinial sori hypophyllous, subepidermal, orange-yellow uredinia covered with a peridium; peridium ruptures due to the pressure exerted by the urediniospores; urediniospores light orange yellow in colour, globose to oval, echinulate; echinulations arranged spirally on exine, 15.4-30.8 x 15.2-26.4 µm; paraphyses abundant, mostly marginal, light yellow, straight to slightly curved with swollen distal ends; no telial stage was observed.

6. Phialophora richardsiae (Nannf.) Conant (IMI 257551).

Colony on PDA white later turns light yellowish brown; hyphae initially non-septate but septation develops in old cultures, highly branched; conidiophores arise as a side branch on the main hyphae, erect, simple, non-septate, 12.35-19.76 x 2.4 pm; conidia numerous, produced terminally, one-celled, oval, 4.94-7.4 x 2.4-3.7 µm.

7. Phomopsis variosporum sp. nov.

Coloniae in agaro 'potato dextrose' primo albae vel flavidae et postremo transformatus atro-griseo-nigra. Pycnidia primo albae tun nigrescens, 1381-1551 µ m diam., stroma bene evolutus flavidobruneae, durus, unilocculariae, ostiolata centralis. Conidiophora hyalina 15.4-19.8 µ m longus; conidia, hyalina O-scptatae, biguttulatae 3.5-5.5x 1.08-1.98 µ m.

Habitatio : In folio vivo ex ***Tectona grandis***

Holotypus : Karulai, Nilambur, Kerala, India : J. K. Sharma, C. Mohanan, E. J. Maria Florence, 3-7-1982, IMI 269014.

Colony on PDA initially cottony white, then turns light yellow and finally dark greyish black; pycnidia develop after a month of incubation at 25°C in dark; pycnidia initially white turn black with the age of the culture, 1381-1551µ m in diam, stroma well developed, hard, yellowish brown, unilocular, ostiolate; conidiophores hyaline 15.4-19.8 µ m long; conidia ooze out in creamy mass, O-septate, biguttulate, hyaline 3.5-5.5x 1.08-1.98 µ m.

Habitat : On living leaves of ***Tectona grandis***

Holotype : Karulai, Nilambur, Kerala, India: J. K. Sharma, C. Mohanan and E. J. Maria Florence, 3-7-1982, IMI 269014.

8. Pseudoepicoccum tectonae sp. nov.

Coloniae in agaro 'potato dextrose' albae, reversum flavidus cum pallide nigra striatus. Conidiophoris synnamatibus pallidus brunneae vel nigra, erectus vcl flexuosa, laevia, incrassatus ad apices, 1.4 µ m, longus. Conidiis obovideus, 6.6-8.8 x 4.4-6.6 µ m, O-septatis, spinoformis structura ad apices subinde.

Habitatio : In folio vivo ex ***Tectona grandis***

Holotypus : Kondodi, Konni, Kerala, India : J. K. Sharma, C. Mohanan, E. J. Maria Florence, 2-6-1984, IMI 286905.

Colony on PDA white, reverse light yellowish with dull black striations; conidiophores synnematous, light brown to black, straight to flexuous, smooth, thickened at apex, 1.4 µ m long; conidia obovoid, 6.6-8.8 x 4.4-6 6 µ m, aseptate, occasionally with spine like structures at the apex.

Habitat : On living leaves of ***Tectona grandis***

Holotype : Kondodi, Konni, Kerala, India: J. K. Sharma, C. Mohanan and E. J. Maria Florence, 2-6-84, IMI 286905.

9. Sclerotium rolfsii Sacc. (IMI 271722).

Colony on PDA fast growing with abundant mycelium having thick strands; numerous white sclerotia, 0.5-1 mm in diam., develop-d within a week of incubation, which turned dark brown in due course.

10. Uncinula tectooae Salm.

Cleistothecia epiphyllous, dark black, globose, 150 to 215 mm in diam. with filamentous, aseptate-hyaline, appendages; asci hyaline, oval, 4-8 spored; ascospores hyaline, ellipsoidal, smooth, 19.2-26.0 x 8.0-10.0 µ m.

BOMBAX CEIBA

1. Cercospora bombacina T. S. & K. Ramakr. (IMI 290732).

On host, conidia hyaline to pale, allantoid, many septate, 17.5-87.5 x 3.5 µ m.

2. Colletotrichum gloeosporioides (Penz.) Sacc.

Colony on PDA greyish black with profuse floccose mycelium, reverse dark black; conidia, produced on cushion shaped acervuli, hyaline, straight, aseptate, guttulate, cylindrical, obtuse at ends, 16.5-26.5 x 4.5 µ m.

3. Corticium rolfsii Curzi (IMI 280236, 280239).

The fungus grows very fast on PDA; colony white, reverse dull light yellowish; abundant sclerotia produced within two weeks of incubation, initially white later turn brown, globose, hard, 1.5-2.0 µ m in diam.

4. Myrothecium roridum Tode ex Fr, (IMI 246401).

Colony on PDA initially white with cottony white mycelium later turning bluish-green in patches due to profuse sporulation; within 3-4 days discoid sporodochia develop in concentric rings; conidiophores erumpent, branched dichotomously, septate, light pale in colour; phialides hyaline to light pale, clavate; conidia spherical to oval, with distinct thick wall; immature conidia hyaline later turn olive green, 4.5-7.1 x 2.0-2.2 µ m.

5. Rhizoctonia solani Kuhn state of Thanatephorns cucumeris (Frank.) Donk (IMI 280235).

Colony on PDA fast growing, off-white turningto light brown with age, mycelia hyaline, later become pale and light brown; sclerotia develop after 7-10 days, dark brown, irregular in shape.

6. Uredo bombacis Petch (IMI 293349-50).

Strain 1: Uredinia orange-brown, hypophyllous, peridium 2-3 cells thick; paraphysis, spherical to oval with distal end obtuse, proximal end broadly narrow, 19.8-33.0 x 15.4-17.6 µ m.

Strain 2: Uredinia dull brown, hypophyllous, subepidermal, prominent peridium 4-5 cells thick; paraphysis not seen; urediniospores few, light yellow, echinulate, oval with distal end obtuse, 22.0-28.6 x 12.0-17.6 µ m.

AILANTHUS TRIPHYSA

1. Botryodiplodia theobromae Pat.

Colony on PDA initially dull white later turns dark greyish-black; pycnidia, produced after 2-3 weeks, globose, dark; conidia, oval, initially hyaline 1-celled, later turn 2-celled dark brown with characteristic longitudinal striations.

2. Colletotrichum dematium (Pers. ex Fr.) Grove (IMI 260692).

Colony on PDA dull white turning grey, mycelium floccose; acervuli disc shaped; conidia hyaline, 1-celled, 37.0-47.0 x 5.0-5.8 µm, falcate with acute apices; setae present, dark, tips light in colour; sclerotia present, black irregular to conical.

3. Colletotrichum state of Glomerella cingulata (Stonem.) Spauld. & Schrenk (IMI 278253).

Colony on PDA fast-growing, producing acervuli within a weak of incubation; conidia hyaline, guttulate, 1-celled, cylindrical, 8.8-17.5 x 3.6-5.3 µm.

4. Corticium salmonicolor Berk. & Br.

Only cobweb, pustule and perfect (pink) stages were observed. For details see *Eucalyptus*.

5. **Meliola ailanthii sp.** nov.

Coloniae in folius ex ***Ailanthus triphysa*** effusae, nigro velutinus; mycelium atro rubro-brunneae, formans inpervius reticulum superficiala, 11.8-14.1 µm latus, hyphopodia alternus in latera oppositus, cellulae basilaris brevis, cellulae terminalis, lobatus, globosus ad leviter reniformis, 11.0-13.0 x 11.0-21.0 µm. Seta mycelium atro brunneae, erecta, septata pallide brunneae versus apices. Conidiis portatus in mycelium, atro rubro-brunneae, quadriseptatis, constrictus ad septum, 11.0-13.75 x 38.5-44.0µm. Ascocarpis sphaericae, tectus cum crassa filum mycelium, 110-147.5µm in diametro; asci cylindrici, bitunicatis, 8-spori, 315-35x10.5µm; ascosporis hyalinae ellipsideus.

Habitatio : In folio vivo ex ***Ailanthus triphysa***

Holotypus : Kuttampuzha, Ernakulam, Kerala, India : J. K. Sharma, C. Mohanan, E. J. Maria Florence, 9-2–1982, IMI 270190.

Colonies on foliage of *A. triphysa* effuse, black, velvetty; mycellum dark reddish brown, superficial forming a close network, 11.8-14.1 µm wide, hyphopodia alternate on opposite sides, basal cell short, terminal cell swollen, lobed, globose to slightly reniform (curved on one side), 11.0-13.0 x 11.0-21.0 µm; mycelial setae dark brown erect, septate, light brown towards the apex; conidia born directly on the mycelium, dark reddish brown, 4-septate, constricted at the septum, 110- 13.75 x 38.5-44.0 µm; ascocarps present, spherical, covered with thick mycelial strands, 741-11.5 µm in diam; asci elongate, double walled, 8-spored 31.5-35.0 x10.5 µm; ascospores hyaline elliptical.

Habitat : On leaves of ***Ailanthus triphysa***

Holotype : Kuttampuzha, Ernakulam Dist., Kerala, India: **J.** K. Sharma, C. Mohanan and E. J. Maria Florence, 9-8-82, IMI 270190.

6. Pseudomonas sp. possibly P. solanacearum (E. F. Smith) E. F. Smith

Colony on nutrient agar off-white, slow growing, producing diffusible pigment; gram negative, rod-shaped, non-fluorescent.

7. Pythium sp.

Colony on rose bengal dextrose agar white, fast growing, hyphae highly branched, coenocytic, 2.4-4.9 µm wide; sporangia stalked, spherical with a prominent papilla, usually terminal, 7 4-11.11 µm in diam; oogonia present.

8. Rhizoctonia solani Kuhn state of Thanatephorus cucumeris (Frank.) Donk (IMI 267022).

Colony on PDA fast growing, white, vegetative hyphae 8 µm in diam., initially hyaline then turns pale yellowish brown due to production of sclerotia, highly branched, septate, branches arising at right angles with a constriction at the point of origin; sclerotia pale yellowish brown, produced singly or in groups, irregular in shape.

GMELINA ARBOREA

1. Colletotrichum state of Glomerella ciogulata (Stonem.) Spauld & Schrenk.

Colony on PDA greyish white, reverse greyish black; setae presant, dark light coloured near the tip; conidia produced in acervuli, straight, guttulate, cylindrical with rounded ends, 10.5-15.9x2.5-4.0 µm.

2. Coryoespora cassiicola (Berk. & M. A. Curtis) Wei (IMI 280237).

Colony on PDA dull green turning black with age; mycelium mostly immersed, hyaline to sub-hyaline, 3-5 µm wide; stroma absent; conidiophores erect, simple, straight to slightly flexuous, pale to dark brown, septate; conidia, subhyaline, cylindrical, 1-8 pseudoseptate, 20.0-77.0 x 5.5-6.6 µm; formed singly or in chains of 2-6.

3. Fusarium solani (Mart.) Sacc. (IMI 260689).

Fast growing cottony colony on PDA with carmine red pigmentation; conidia heterogenous, cylindrical, straight or slightly curved, 0-septate, 14.0-22.0 x 7.4 µm; 1- to 4-septate 17.0-29.0 x 7.8 µm; chlamydospores present, singly or in chains.

4. Griphosphaeria gmelinae sp. nov.

Ascomatibus piceus solitaris immersuam inferme evolutus stroma in ligno, pleaumgric tectus a clypeus et circumcinctus ad atro-brunneus hyphis; para physibus numerosus, longus, flexuosus;

asci unitunicati, amyloideus, 8-spori, 66.0-72.6 x 5.5-6.6 µ m; Ascosporis hyalina, fusiformis, acutae in ambabus extremitatibus, 17.6-19.8 x 4.4 µ m, l-septatis.

Habitatio : In caulis mortus ex parte ex ***Gmelina arborea***

Holotypus : Arippa, Quilon Dist., Kerala, India : J. K. Sharma, C. Mohanan, E. J. Maria Florence, 20-7-1981, IMI 261570

On the host, ascomata black, single, embedded in a weakly developed stroma in the wood often covered by a clypeus and surrounded by intramatrical dark brown hyphae; paraphyses numerous, long, flexuous; asci, unitunicate, amyloid (apices turn blue with iodine), 8-spored, 66.0-72.6 X 5.5-6.6 µ m; ascospores, hyaline, fusiform, sharply pointed at both ends, 17.6-19.8 X 4.4 µ m, l-septate.

Habitat : Partially died stem of *Gmelina arborea.*

Holotype : Arippa, Quilon Dist., Kerala, India: J. K. Sharma, C. Mohanan and E. J. Maria Florence, 20-7-81; IMI 261570

5. **Lentinus squarrosulus** Mont.

Colony on PDA cottony white, initially slow, later growing fast; sporophore-like structures are produced at the periphery of petri dish after I month's incubation in dark; mycelium hyaline, highly branched with prominent clamp connections.

Sporophores 10-15 cm in length; pileus 8-12 cm diam, convex to bell shaped, off-white to light brown, lobed with grooves, margin decurrent, entire, squarrose scales, dull brown, present on the entire surface, denser in the region between margin and centre; stipe usually central, occasionally eocentric, cylindric, solid with appressed scales, concolourous with pileus upto some distance; basidia 4-spored, numerous, 23.8-27.8 X 2.2-3.3 µ m, clavate; basidiospores borne on pointed sterigma, hyaline, ellipsoid, thin walled, 4.5-6.0 X 2 0 µ m.

6. **Phoma nebulosa** (Pers. ex S. F. Gray) Berk. (IMI 260690).

Colony on PDA variable, usually dull green to olivaceous brown; mycelium light olivaceous green and moderately felty: pycnidia dark brown, ostiolate; conidiophores short; conidia hyaline, l-celled, 4.6-7.0 X 2.0-2.4 µm, cylindrical to ellipsoid, biguttulate, straight to slightly curved.

7. **Pseudocercospora ranjita** (Chaudhury) Deighton (IMI 269020).

On the host, numerous conidiophores arise from stroma; conidiophores clustered, branched, pale to light brown; conidia long, hyaline to pale, pseudoseptate (2-7), often slightly constricted at the septum, 26.4-61.6 X 4.4-6.6 µ m.

8. **Sclerotium** sp.

Colony on PDA white, fast growing with thick corded mycelium; sclerotia, numerous, produced on seventh day of incubation, initially white later turn yellowish orange, round to globose, single or aggregate, varying in size greatly, 1-6 mm in diam.

9. **Thyronectria pseudotricha** (Schw.) Seeler

Conidiomata synnematous, 1-3 mm high, yellowish orange to light brown; conidial head round, 0.5 to 1 mm in diam; conidiophores numerous, branched; conidia minute, oval to spherical.

DALBERGIA LATIFOLIA

1. **Colletotrichum** state of **Glomerella cingulata** (Stonem.) Spauld. & Shrenk.

Colony on PDA off-white to light grey; mycelium slightly floccose; acervuli cushion shaped, waxy; setae present, dark blackish-brown; conidiophores simple, conidia, hyaline, 1-celled, guttulate, 11-15.4 x 3.96-4.84 µm.

2. **Phyllachora dalbergiae** Niessl. (IMI 293352).

Ascomata black, scattered on the adaxial surface, dark black, shiny, partially immersed in the leaf tissue; perithecia subglobose to globose with minutely papiollate ostioles; asci cylindrical, sessile to short pedicillate, 8-spored 35.0 **x** 7.0 µm; ascospores biseriate, I-celled, elliptical, hyaline, 10.5-14.G x 3.0-5.0 µm; paraphyses numerous, slender.

3. **Physalospora dalbergiae** sp. nov.

Perithecia sparsus in substrato folio immersum cum protrusus atro-brunneous vel nigrus clypeus, 72.0-76.8 µm in diam., asci plerumque affixes ad basi ex peritehcia, membranis non crassis, gracilis, sessilis, hyalina 35.0-42.0 x 7.08.75 µm; Ascosporae, biseriatae, ellipsoideae, 1-septatae 8.75-12.25 x 3.5 µm; paraphyses paucus.

Habitatio : In folio vivo ex *Dalbergia latifolia*

Holotypus : Vattapoyil, Wynad Dist., Kerala, India: **J.** K. Sharma, C. Mohanan, E. **J.** Maria Florence, IMI 286904

Perithecia immersed in the leaf tissue with protruding dark brown to black clypeus, scattered, 72.0-76.8 µm in diam., asci mostly attached at the base of perithecia, thin walled, slender, sessile, hyaline, 35.0-42.0 x 7.0-8.75 µm; ascospores biseriate, hyaline, ellipsoidal, 2-celled, 8.75-12.25 x 3.5 µm; paraphyses scanty.

Habitat : On living leaves of ***Dalbergia latifolia.***

Holotype : Vattapoyil, Wynad Dist., Kerala, India: J. K. Sharma, C. Mohanan and E. J. Maria Florence, IMI 186904

4 Uredo sissoo Syd. & Butler (IMI 273438).

Uredinia hyphophyllous, subepidermal; urediniospores borne on short l-celled stalk, ovoid or obovoid, light yellowish brown, 24.5–31.5 x 13.7-14.7 µm; peripheral paraphyses abundant, yellowish-brown to reddish-brown, thick walled, smooth, distal end swollen, clavate to subcylindrical, curved inward; paraphyses among urediniospores sub-hyaline, distal end swollen, slightly curved or straight; teleutosori absent.

OCHROMA PYRAMIDALE

1. Calonectria rigidiuscula (Berk. & Br.) Sacc. (IMI 257549); conidial state.

Colony on PDA dull yellowish-brown, floccose, aerial mycelium appears powdery after the formation of microconidia; microconidia borne on lateral branches, phialides simple, lateral or terminal, 25.0-36.0 x 4.0-5.0 µm; microconidia 0-1 septate, 7.4-16.6 x 2 0-2.3 µm; macroconidia produced in cream coloured pionnotes, conidiophores loosely branched with terminal phialids, macroconidia curved, fusoid, 4-6 septate, 32.0-56.8 x 4.9-6.7 µm; chlamydospores absent.

2. Fnasrium moniliforme var. subglutinans Wollenw. & Reink. (IMI 257550).

Colony on PDA purplish-violet, aerial mycelium floccose; microconidia found on branched conidiophores terminating in polyphialides, oval to ellipsoid, aseptate, hyaline, 5.6-7.4 x 2.4-3.7 µm; macroconidia few, produced in sporodochia, simple, 1-3 septate, 17.0-20.0 x 3.0-4.5 µm; chlamydospores absent.

EUCALYPTUS

1. Alternaria alternata (Fr.) Kiessler (IMI 246476).

Colony on PDA black to olivaceous black; conidiophores develop singly or in groups, simple or branched, straight, geniculate, pale olivaceous, yellowish-brown to dark brown, smooth with conidial scars; conidia formed in long chains, obclavate, ovoid to ellipsoid, often with a short cylindrical beak, with transverse and longitudinal septa, 30.0-65.0 x 12.0-15.0 µm.

2. Rotryodiplodia theobromae Pat. (Syn. Lasiodiplodia theobromae Pat.) Griff & Maubla. (IMI 246478).

Colony on PDA greyish black, fast growing; pycnidia black, ostiolate, immersed; young conidia oval, hyaline; mature conidia dark brown with longitudinal striations, 2-celled, 20-23 x 11.0-12.3 µm.

3a. Calonectria floridana Sobers (IMI 250220, 276603)

Colony on PDA yellowish-brown with appressed aerial mycelium; perithecia develop after 15 days of incubation, orange red, globose to oval, 408-418 x

301-312 mm with papillate ostiole; asci clavate, thin walled, hyaline, 8-spored, 71.1-142.4 x 15.3-22.8mm. Ascospores hyaline, l-septate, 24.7-34.6 x 4.9-7.8 µm.

b. Cyliodrocladium floridanum Sobers & Seymour (IMI 250219).

Colony on PDA initially yellowish orange with abundant white cottony aerial mycelium later turning dark orange-brown due to formation of chlamydospores and microsclerotia; conidiophores penicillate arising laterally from stipe; phialides hyaline, short; conidia hyaline, cylindrical, 1-septate, 32 0-45.0 x 2.7-4.0 µm; sterile filament upto 172 µm long terminates into a globose vesicle 14.8-17.2 x 5.1-6.1 µm.

4a. Calooectria ilicicola Boedijn & Reitsma (IMI 250212).

Perithecia produced on the host tissue after 20 days of incubation at 25 ± 2°C, ovoid to globose, yellowish-orange, pseudo-parenchymatous with papillate ostiole; asci clavate, hyaline, unitunicate, 4–spored; ascospores fusoid to falcate, curved I-4-septate (mostly 3-septate), hyaline, 47.4-66.7 x 4.9-5.4 µm.

b. Cyliodrocladium ilicicola (Hawley.) Boedijn & Reitsma (IMI 250214, 250215).

Colony on PDA light yellowish-brown with abundant aerial mycelium, with age colony turns darker due to production of chlamydospores and microsclerotia; conidiophore penicillate arising laterally from a stipe; conidia hyaline, cylindrical, 1-3 septate (mostly 3-septate) 39.5-61.0 x 4.0-5.6 µm; sterile filament terminating into a vesicle 14.0-18.7 x 3.2-4 9 µm.

5a. Calooectria quinqueseptata Figueiredo & Namekata

Perithecia superficial, oval to elliptical, orange to chest nut, 360–580 mm high, 300–400 Lm diam.; perithecial wall roughened by masses of large pseudoparenchymatous cells, leaving a smooth papillate ostiole; asci club–shaped, long stalked, hyaline, 8-spored, 76-126 x 13-22 µm; ascospores hyaline. variously curved, 1-6 septate, mostly 3 septate, 30-80 x 4-7 µm

b. Cyliodrocladium quiuqueseptatum Boedijn & Reitsma (IMI 246473, 250218, 250223, 276612, 200728-33, 280742).

Colony on PDA yellowish-brown with scanty aerial mycelium and irregular dull yellowish margin; colonies turned dark brown with age due to production of chlamydospores and microsclerotia; conidiophores penicillate arising from a stipe, phialides, hyaline; conidia hyaline, straight, cylindrical, 3-7 septate (usually 5-septate), 74.0-135.0 x 6.0-7.5 µm; sterile filament 192–345 µm long, terminating into a narrowly clavate vesicle, 2.8–3.4 µm diam.

6. **Calonectria theae** Loos and its anamorph **Cyliodrocladium theae** (Petch) Alf. & Sob. (IMI 280734-37; 280739-41).

Perithecia produced in culture after 2 weeks of incubation, superficial, scattered, arising from a stroma, globose to ovoid, yellowish orange, later turning reddish brown 208–420 µm diam, and 210–390 µm in height; asci club-shaped, hyaline, thin walled, long stalked, 8-spored, 68.7–128.2 x 16-3-24.1 µm; ascospores hyaline, elongate-fusoid, 3-septate, 47.0-68.1 x 4.9-7.8 µm.

Colony on PDA fast growing with abundant aerial mycelium; conidiophore branches arise from a stipe; phialides hyaline; conidia hyaline, cylindrical, 3-septate 42.0-66.0 x 3.9-6.6 µm; sterile hyphae septate, 242-385 µm long, terminates in a broadly clavate vesicle; chlamydospores and microsclerotia are produced in culture.

7. **Coniella castaneicola** (Ell. & Ev.) B. Sutton (IMI 278251)

Colony on PDA off-white to cream with numerous pycnidia developing within a week; pycnidia pale brown 126-176 x 110-176 µm; conidia hyaline, straight, slightly curved, fusiform, 11.0-15.4 x 2.8-3.3 µm.

8. **Coniella fragariae** (Oudem.) Sutton (Syn. **Coniella pulchella** Hohnel) (IMI 262984, 278252)

On host *(E. grandis)* conidiomata pycnidial, dark, lenticular to globose, ostiolate, simple, partially immersed in the leaf tissue; pycnidial wall light to dark brown made up of 2-3 layer of thin walled cells; conidiophores simple, subhyaline; conidia yellowish brown to light brown, thin walled, globose to napiform, 1-celled, 22.2-24.7 x 12.0-14.8 µm.

In culture (isolated from *E. tereticornis),* colony on PDA off-white to pale yellowish brown, slow growing; pycnidia produced after 10-15 days of incubation at 25 $\pm$ 2°C, brownish 180-300 x 175-275 µm; conidia, globose to napiform, thin walled, 6.6-13.0 x 6.1-8.8 µm. produced in conidial ooze.

9. **Coniella granati** (Sacc.) Petrak & Syd. (IMI 280238).

Colony on PDA pale brown with numerous black pycnidia developing after two weeks; pycnidia globose, ostiolate, 11.0-13.2 x 11.8-14.5 µm; conidia hyaline, straight or slightly curved to fusiform, 8.8-15.4 x 2.3-4.4 µm.

10. **Corticiom salmonicolor** Berk. Br.

C. salmonicolor produced four stages in its life cycle on eucalypts. They are described below in sequence of their development.

i. ***Cobweb:*** This is the first stage developed over the infected bark during the rainy season. It was characterized by silky white, corded interwoven arachnoid hyphae, appressed on the stem. The hyphae grew rapidly from the periphery and covered a large part of the stem. The bark beneath the cobweb got depressed and showed browning.

ii. ***Pustule:*** Pustule like bodies were formed initially on the older part of the cobweb mycelium. They are light pink to salmon, superficial, globose to irregular, sterile mycelial bodies measuring upto 2 mm in diam.

iii. ***Pink encrustation*** (perfect stage): This stage was characterized by sexual fructifications, light pink to salmon in colour developed over the entire surface of infected bark, where cobweb and pustules were formed earlier. Occasionally the pink stage developed on one side of the stem, side of the branches away from the afternoon sun.

The fructification (basidiocarp) resupinate, membranous, upto 500 μm thick, salmon pink when fresh but cracking and fading to light cream or whitish when dry; hyphae of the basidiocarp monomitic devoid of clamp connections, hyaline and smooth; hymenium composed of a layer of basidia obovoid to broadly clavate when young but becoming narrowly clavate to cylindrical when mature, thin walled, with four sterigma; basidiospores broadly ellipsoid with prominent apicule, thin walled, smooth, hyaline, 8.0–13.0 x 6.0-9.8 μm; paraphyses arranged in palisade manner.

iv. ***Necator:*** The fourth asexual spore-bearing stage generally developed only during the dry period on the dead bark, exposed to the sun. Initially it appeared in the form of small erruptions on the bark which later became orangish-red pustules due to profuse sporulation; conidia hyaline to sub-hyaline, unicellular, ovoid to irregular in shape, 8.0-20.0 x 6.0-12.5 μm.

11. **Cryphonectria cubensis** (Bruner) Hodges (IMI 254084, 261569, 274337, 274339, 274340-274342, 274344, 274345, 274347, 281616, 181617).

Colonies on PDA hyaline, later colour changed to light yellowish orange; numerous yellowish orange pycnidia produced after 7 days of incubation; no perithecia were observed even in cultures kept for 2 months.

Pycnidia usually scattered profusely on the dead bark, developedeither singly or in loose groups during the following monsoon when the cankers were nearly I-year-old. These superficial pycnidia with their bases slightly embedded in the bark were initially bright orange in colour but became darker except at the tip of the neck. They were cylindrical to broadly pyriform in shape with an attenuated

neck and measured 423-1100 x 112-282 µm with basal diameter of 183-634 µm Conidia were hyaline, one celled, broadly oval to clavate 2.46-4.1 x 1.9-2.4 µm and were extruded in bright yellowish tendrils upto 2-3 mm long, under high humidity (> 95%).

Perithecia also having long necks, were seen developing on 18-month-old cankers during the dry period (April-May) either singly or in groups of 2-8. During the rainy season the whole infected bark became covered with profuse growth of perithecia. Young perithecia were initially light brown and turn dark brown to black on maturity. As perithecia developed beneath the bark their bases were immersed and only the neck (584-846 x 98.7-1 13 µm) protruded out, piercing the bark. Asci, 27.5-33 x 4.0-7.15 µm were clavate and contained 8 ascospores arranged biseriately. Ascospores were two celled, hyaline, cylindrical, straight to slightly curved with rounded ends and measured 4.4-9.5 x I.9-3.0 µm.

12. **Cryphonectria gyrosa** (Berk. & Br.) Sacc. (Syn. **Endothia havanensis** (Bruner) Baur. (IMI 261575, 274336, 274343, 274346,281618).

Colony on PDA yellowish white turning cream and later light brown; mycelium slightly appressed; conidiomata pycnidial. developing on mycelial strands within two weeks of incubation; conidia ooze in long yellowish tendrils, O-septate, hyaline, ellipsoid, 2.5-3.5 x 1.1-2.5 µm; no perithecia developed in culture.

On host pycnidia stromatic, broadly pyriform, partially or completely immersed in the bark, ostiolate, papillate, initially bright orange-red turning brownish to black when mature, produced singly or in groups of 5-6, either scattered or arranged in short vertical linear rows depending upon the *Eucalyptus* species, 176-330 x 163-319 µm; conidia straight to slightly allantoid, O-septate 2.2-4.5 x 1.0-1.9 µm; spores extrud in long tendrils under high humidity.

Perithecia embedded in bright orange errumpant stromata develop during the dry period (December-April), later turning black, perithecial necks long protruding with tip light yellowish in colour, 2-4 mm; asci clavate, thin walled, 8-spored, 25.5-38.0 x 4.5-6.8 µm; ascospores irregularly biseriate, hyaline, 2-celled, slightly constricted at the septum with tapering ends.

13. **Cyliodrocarpon lucidum** Booth (IMI 267023).

Colony on malt agar yellowish orange with floccose aerial mycelium; phialides simple formed laterally on hyphae, terminal on simple lateral branches; phialospores 2-4 septate, hyaline, straight, cylindrical with rounded ends, 33.0-55.0 x 4.4-5.7 µm; chlamydospores brown, globose, single or in chains, intercalary or terminal.

14. **Cyliudrocladium camelliae** Venkataramani & Venkataram (IMI 262979).

Colony on PDA initially white, later turning light yellowish brown with profuse aerial mycelium and globose chlamydospores formed in chains; conidiophores penicillate, conidia cylindrical, hyaline, 1-septate, 20-30 x 2.7-5.4 μm with occasional swollen end cells; sterile filament aseptate 75-125 μm long, with lanceolate to ellipsoidal or spathulate vesicle, 10-35 x 5-7.5 μm.

15. **Cylindrocladium clavatum** Hodges & May (IMI 270185).

Colony on PDA yellowish-orange, later turning dark orange brown due to production of abundant chlamydospores and microsclerotia; conidiophores penicillate, conidia straight, cylindrical, hyaline, I-septate, 40-51 x 2.9-3.3 μm; slightly wider at the distal end; apical swelling of conidia was observed in old cultures; sterile hyphae septate, 143-174 μm long, terminating in a broadly clavate vesicle, 3.6-6 μm wide.

16. **Cylindrocladium curvatum** Boedijn & Reitsma (IMI 246474).

Colony on PDA yellowish-brown, with abundant cottony aerial mycelium, chlamydospores frequent and aggregate to form microsclerotia; conidiophores penicillate, varying in length, dichotomously branched at the apex; conidia cylindrical, hyaline, distinctly curved, 1 septate, 35-60 x 3.2-5 mm; sterile filament 86.5-17.3 x 2-2.3 μm terminating in a globose to ovoid vesicle, 9-12 x 6.4-8.6 μm.

17. **Cyliudrocladium parvum** Anderson (IMI 250221).

Colony on PDA light chocolate brown with a distinct white border, 4-6 mm wide, and characteristic concentric zones; aerial mycelium white, sparse; colony turns darker after 2-3 days due to development of chlamydospores and microsclerotia; conidiophores penicillate, varying in length, dichotomously branched near the apex; conidia cylindrical; hyaline, 1-septate, occasionally with swollen cells, 9.9-14.0 x 1.98-2.47μm; sterile filament 74.1-106.2 x 1.6-2.3 μm terminating into a clavate to spathulate vesicle, 9.9-33.5 x 4.9-9.9 μm.

18. **Cyliudrocladium scoparium** Morg.

Colony on PDA reddish brown with irregular white margin, 4 mm wide, aerial mycelium initially white turned reddish brown after 4-5 days of incubation, microsclerotia produced in abundance; conidiophore dichotomously branched near the apex; conidia straight, cylindrical, I-septate, occasionlly with one swollen cell, 51.0-62.0 x 4.6–6.1 μm; sterile filament terminates in a ellipsoidal to spear shaped vesicle, 14.8-19.8 x 6.2-9.9 μm.

19. Cytospora eucalypticola van der Westhuizen (IMI 284046).

Colony on PDA brownish black, reverse black, mycelium immersed, hyphae dark brown; conidiomata pycnidial, produced after two weeks of incubation, pycnidiospores ooze as light pale droplets, hyaline, O-septate, thin walled, allantoid, 3.5-7.7 x 0.5-1 μm.

On the host ***(E. tereticornis),*** conidiomata stromatic, pycnidia, black immersed in the bark, grouped 2-10, closely packed in 1 to 2 tiers; pycnidia with dark brown to black wall fused at many places, opening through a common ostiole, 275-577 μm wide and 165-412 μm in height; pycnidiospores hyaline, O-septate, allantoid 3.3-4.4 x 1.1-2 2 μm.

20. Cytospora eucalypti sp. nov.

Pycnidia in substrato caulius nigra 5-6 laxe aggregatae non compactus et evolutus circumcinctus interius zona cornpositus ex atro-brunneaus mycelium crassa tunicata, pariete pycnidia 2-3 cellulae crassa, fuscus vel atrobrunneus, non connatus vel unilatus tantum. Conidiophora simplicis vel ramosi; condia hyalina, levitier allantoideus cum rotunda tae O-septatae, 3.3-5.5x2.2 μm.

Habitatio : In caulis ex *Eucalyptus grandis*

Holotypus: Meenmutty Idukki, Dist., Kerala, India: J. K. Sharma, C. Mohanan, E. J. Maria Florence, IMI 261564.

On the host (E. grandis), conidiomata pycnidial, black, in loose clusture of 5-6, not compact or compressed and develop only in one tier surrounded within a zone composed of dark brown thick-walled mycelium; pycnidial wall, 2-3 cell thick medium to dark brown, not fused or partially fused only on one side; conidiophores simple or branched; conidia, hyaline, slightly allantoid with rounded ends, 33-5.5 x 22 μm.

Habitat : On stem of ***Eucalyptus grandis***

Holotype : Meenmutti, Idukki Dist., Kerala, India: J. K. Sharma, C. Mohanan and E. J. Maria Florence, IMI 257876, 261568.

21. Fusarium oxysporum Schlecht. (IMI 269013).

Colony on PDA white with pinkish tinge, fast growing, cottony with abundant dense aerial mycelium, floccose to felted; conidiophores simple, lateral found on aerial hyphae; microconidia aseptate clavate to fusiform with slightly truncate base, hyaline, 6.1-19.8 x 3.9-5.5 μm; macroconidia not observed.

22. Guigoardia citricarpa Kiely (IMI 287783)

Colony on PDA greyish black with greenish tinge, velvetty, slow growing with restricted irregular margin; conidiomata pycnidial, separate or aggregate, light

brown to black, 44.0-88.0 μm in diam., spherical to oval, ostiolate. slightly papillate; conidia hyaline, O-septate, thin walled, smooth, obovate to elliptical with truncate base: no perithecia formed in culture.

Perithecia developed on dead fallen leaves, solitary or aggregate, globose, immersed, dark brown to black, thin walled, outer side sclerotoid, ostiolate, non-papillate, circular; asci clavate to cylindrical, sessile to short, stipitate, 8-spored, 45.0-75.0 x 11.8-15.2 μm, ascus wall thick, bitunicate; ascospores aseptate, hyaline, multiguttulate, cylindrical, 15.5 x 3.3-5.5 μm, ends obtuse with small truncate, hyaline appendage.

23. **Hysterium angustatum** Alb. & Schwein (IMI 257895).

Hysterothecia superficial, scattered or aggregate 3-4 mm long, 440-467 μm wide; 412-423 μm high, black, carbonaceous, horizontally elongated, oblong or boat-shaped, pointed at ends, opening by a median narrow slit; asci cylindrical, long with small stalk, clavate; ascospores in 2-3 rows, elliptical or cylindrical, 27.5-30.25 x 10.5-12.25 μm, uniformly brown, 3-septate, slightly constricted at the septum.

24. **Lentinus squarrosulus** Mont.

Sporophores 12-16 cm in length; pileus 10-12 cm in diam., bell shaped, light brown, margin decurrent, entire with dull brown squarrose scales on the surface; stipe central, cylindrical, solid with appressed scales on the surface; basidia clavate; basidiospores 4, borne on pointed sterigma, hyaline, ellipsoid thin walled, 4.8-6.6 x 2.0 μm.

25. **Macrovalsaria megalospora** (Mont.) Sivan. (IMI 261566).

Ascomata stromatic, aggregated in 5-6 perithecia, immersed in bark, subcuticular with beaks protruding out, ostiolate, black; asci originating from flat base of perithecium, cylindrical, double walled, hyaline, 235-247 x 19.2-24.7 μm; ascospores 8 per ascus, linearly arranged, ellipsoid, immature ascospores hyaline, later turning moderate to dark brown, 2-celled, distal cell light brown, proximal cell dark brown with vertical striations.

26. **Microporus xaothopus** (Fries) O.Kuntze.

Hymenophore annual, solitary, coricaceous, attached by a central or eccentric stipe arising from a broad mycelial disc; pileus companulate, occasionally one side partly suppressed, orbicular or elliptical, 3-9 cm diam., 1-3 mm thick, pileus surface chestnut to amber coloured, strongly concentrically ridged or sulcate or alternating with concentric bands of varying shades of yellow, glabrous, cortex upto 55 μm thick, margin acute, plane, slightly inturned, brown or with yellow tinge, entire (immature) to crenate (mature); stem 1.5-13 cm long and 3-5 mm in diam., porous, cortex white;

hymenial surface decurrent, 1-2 μm deep; basidia clavate 8.0-10.5 x 4.5-5.0 μm, 4-spored; paraphyses subclavate; basidiospores elliptical, spiculate, 3.5-4.0 x 2.0-2.5 μm, smooth, hyaline.

27. Nattrassa tordoidea (Nattrass) Dyke & Sutton (IMI 261565).

On the host, conidiomata pycnidial, black, solitary, immersed first later bursting through the surface of bark, raised, unilocular, globose, 291 x 242 μm, wall dark brown made up of many layered thick walled cells; conidiophore hyaline, simple; immature conidia 1-celled, hyaline to yellowish later becoming 3-4 celled and dark brown, ellipsoid to ovoid, 14.8-19.7 x 6.0-7-4 μm.

Scytalidium state of N. tordoidea (IMI 267017).

In culture only the *Scytalidium* state was observed. Colony on PDA effuse, dark blackish-brown to black, mycelium immersed, hyphae dark brown, thick walled, septate, smooth; conidiomata eustromatic, irregular; conidiophores micronematous; conidia, arthroconidia, dark brown, smooth. mostly 0-septate, occasionally septate, cylindrical with obtuse apex and truncate base, eguttulate, 4.4-11.0 x 2.8-5.0 μm.

28. Pestalosphaeria elaedis (Booth & Robertson) van der Aa (IMI 268335.)

Colony on PDA white cottony with floccose mycelium, after the development of conidiomata felted; conidia 4 euseptate, straight or slightly curved, 19.8-30.8 x 6.6-6.8 μm, apical cell hyaline with 2-3 appendages, 17.6-33.0 μm long, median cells brown and thick walled, basal cell hyaline with a single appendage, 3.3-4.4 μm long.

29. Pestalotiopsis disseminata (Thum.) Steyaert (IMI 268328).

Colony on PDA white, dense, cottony, fast growing; conidiomata acervular, dark; conidia 4 euseptate, 24.2-28.6 x 6.0-6.8 μm, apical cell hyaline with 2 appendages, 13.2-19.8 μm long, basal cell hyaline with simple appendage, 4.4-4.62 μm long. median cells dark brown.

30. Pestalotiopsis guepinii (Desm.) Steyaert (IMI 268324, 268327, 268336).

Colony on PDA, white, cottony, dense; conidiomata acervular upto 210 μm; conidiophores simple, hyaline; conidia 19.8–26.3 x 4.8-6.82 μm, basal cell hyaline truncate with simple appendage, 4.4-6.6 μm long, apical cell conic, hyaline with 2-3 appendages, 15.4-26.4 μm long, median cells concolourous.

31. Pestalotiopsis maogiferae (P.Henn.) Steyaert (IMI 268326, 268330, 268334;

Colony on PDA dense white; aceruvuli develop from small clumps of hyphae and form conspicuous greenish black slimy spore mass; conidia 4 euseptate, oblong

to clavate, 17.6-28.6 x 4.6-6.16 μm, median cells dark brown, thick walled, apical cell hyaline with 2-3 appendages, 11.0-19.8 μm long, basal cell truncate hyaline with single appendage, 3.3-4.4 μm long.

32. Pesta1otiopsis neglecta (Thum.) Steyaert (IMI 268329,26833I, 268333).

Colony on PDA white, dense felted; conidiomata acervular; conidia 4 euseptate, 22.0-28.4x 4.62-6.60 μm, apical appendage 2-3 (usually 3), basal cell truncate with one simple appendage, median cells dark brown.

33. Pestalotiopsis versicolor (Speg.) Steyaert (IMI 268332).

Colony on PDA white, dense, fast growing; conidiomata acervular; conidia 4 euseptate, 24.2-28.6 x 6.6-68 μm, apical cell hyaline with 2-3 appendages, median cells dark brown, thick walled, basal cell hyaline with single appendage.

34. Phaeoseptoria eucalypti (Hansf.) Walker (IMI 246483).

On the host, pycnidia sub-globose to globose, embedded in the leaf tissue, narrowly ostiolate with membranous pseudoparenchymatous wall; conidiophores simple, hyaline to pale; conidia yellowish-brown to brown, cylindrical, straight to slightly fusiform with rounded to sub-truncate base, smooth, transversely two to several septate, 45.0-55.0 x 4 0-5.5 μm.

35. Pythium deliense Meurs. (IMI 267018, 281614).

Colony on PDA fast growing, cottony, floccose; hyphae hyaline, coenocytic, except where fructifications formed, 4 4-6 6 μm wide; sporangia filamentous, branched or unbranched, 17.5-52.0 x 9.8-10.5 μm; oogonia smooth walled, terminal or intercalary, 19.0-23.0 μm in diam; antheridia 1-2(2); oospore spherical, thick walled.

36. Pythium myriotylum Drechsler (IMI 268319, 268320).

Colony on PDA white, cottony, floccose; hyphae hyaline, coenocytic, 2.9-7.0 μm wide; sporangia inflated, filamentous greatly variable in size, 22.0-175.0 x 4.6-14.0 μm; oogonia spherical, terminal or intercalary; antheridia terminal or intercalary; oospore thick walled.

37. Pythium spinosum Saw (IMI 27661 1, 276605, 276608, 276609).

Colony white with abundant mycelium on oat meal agar, hyphae hyaline, 2.2-8.8 μm wide, branched, coenocytic; sporangia terminal or intercalary, spherical to pyriform 11.0-46.2x 15.4-24.2 μm; oogonia abundant, spherical, ornamented with projections, intercalary or terminal, 13.2-35.2 μm; antheridia 1-2; oospore spherical, thick walled, ornamented.

38. **Rhizoctonia solani** Kuhn state of **Thanatephorus cucumeris** (Frank.) Donk

Colony on PDA off-white to pale, fast growing; mycelium corded, thick, 17 μm in diam, branches arise at right angles of the main hyphae; sclerotia initially dull yellow later turning dull brown produced in loose groups on interwoven hyphae, irregular, variable in size.

39. **Sclerotium rolfsii** Sacc. state of **Corticium rolfsii** Curzi (IMI 269010, 160693).

Colony on PDA white, fast growing; mycelium abundant, corded, thick; sclerotia round, globose to ellipsoidal, initially white later turning pale and then dark brown, surface glabrous, reticulate, single or aggregated, 2-6 μm diam.

40 **Thyronectria pseudotricha** (Schw.) Seeler(IMI 252784, 257897).

Conidial state synnematous, 2-4 mm long, dull orange to light brown in colour; conidial head 0.5 to 1 mm in diam, conidiophores minute, profusely branched conidia, minute oval to spherical, hyaline.

Pseudothecia initially bright coloured later turning black, carbonaceous, horizontally elongated, oblong, boat-shaped, deeply ribbed, superficial, developing singly; asci clavate, ascospores 8 per ascus, hyaline, dictiosporous, 39.0-64.0 x 14.0-25.0 μm.

41. **Valsa eucalypti** Cook & Harkness (IMI 261564).

Ascomata deeply immersed in pseudostromata without dark zonations, compactly aggregate, flask shaped situated just below the layer of cork cells (subcuticular), 220–330 x 176–209 μm with long beaks upto 625 μm ostiole with thick membranous wall; asci clavate, 30.0-54.0 x 5.0-6.6 μm, thin walled 8-spored ascospores l-celled, hyaline, biseriate, allantoid, 7.4-9.8 x 2.0-2.5 μm.

42. **Valsa eucalypticola** sp. nov.

Ascomatibus profunde immersum in pseudostromatibus absque atrozonatus, aggregatae, crassa tunicata, 140-231 x 59-165 μm; rostro nigra usque ad 423 μm; asci clavati; membranis, noncrassis 8-spori, 15.0-26.4x7.0-8.8 μm; ascosporis, biseriatus, allantoideus; hyalina 3.3-6.6 x 1.1-2.6 μm.

Habitatio : In caulk ex ***Eucalyptus grandis***

Holotypus : Thrissillery, Wynad, Dist., Kerala, India: J. K. Sharma, C. Mohanan, E. J. Maria Florence, IMI 257896, 261568.

Ascomata deeply immersed in pseudostromata without dark zonations, loosely aggregated in groups of 4-5, thick walled, 140-231 x59–165μm; beaks uptq 423 μm, black, thick walled; asci clavate 15.0-26.4 x7. 0-8.8 μm; thin walled, 8-spored, ascospores biseriate, allantoid, hyaline, 3.3-6.6 x 1.1-2.6 μm.

Habitat : On stem of ***Eucalyptus***

Hofotype : Thrissillery, Wynad Dist., Kerala, India: J. K. Sharma, C. Mohanan and E. J. Maria Florence, IMI 257896; 261568.

APPENDIX-III

LIST OF PATHOGENS AND THEIR HOSTS

Sl. No.	Pathogen	Host (s)	KFR1 No.	IMI No.	ITCC No.
1.	*Acremonium recifei* (Leao & Lobo) W. Gams	hyperparasite on *Olivea tectonae*	1146	204045	3520
2.	*Agrobacterium tumefaciens (E.* F. Sm.) Conn.	*Eucalyptus*			
3.	*Alternaria alternata* (Fr.) Kiessler	*Eucalyptus*	024	246476	3522
4.	*Botryodiplodia theobromae* Pat.	*Eucalyptus* *Ailanthus triphysa*	050 958	246478	3496
5.	*Calonectriafloridana* Sobers	*Eucalyptus*	127a 894	250220 276603	
6.	*C. ilicicola* Boedijn & Reitsma	*Eucalyptus*	001-4 010, 012	250212-13 250216-17	
7.	*C. theae Loos* and its anamorph *Cylindrocladium theae* (Petch) *Alf.* & Sob.	*Eucalyptus*	1082(1) 1082(2) I063(1) 1100 1019 1091 495(D) 1162	280734-37 280739-41	
8.	*C. quinqueseptata* Figueiredo & Namekata	*Eucalyptus*	68		
9.	*C. rigidiuscula* (Berk. & Br.) Sacc.	*Ochroma pyramidale*	389a	257549	3511
10.	*Cercospora bombacina* T. *S.* & *K.* Ramakr.	*Bombax ceiba*	*0*54	290732	
11.	*Cladosporium oxysporum* Berk. & M. A. Curtis	hyperparasite on *Olivea tectonae*	1145	284044	3519
12.	*Colletotrichum dematium* (Pers. ex Fr.) Grove.	*A. triphysa*	420	260692	
13.	*Colletotrichum gloeosporioides* (Penz.) Sacc.	*Tectona grandis* *B. ceiba* *Eucalyptus*	36 260 067		

Condt.

1	2	3	4	5	6
14.	*Colletotrichum* state of *Glomerella cingulata* (Stonem.) Spauld. & Schrenk	*T. grandis* *A. triphysa* *Gmelina arborea* *Dalbergia latifolia* *Eucalyptus*	073 916 566 567 434	246479 278253 260689 269017 260691	3504 3508 3507
15.	*Coniella castaneicola* (Ell. & Ev.) Sutton	*Eucalyptus*	845	278251	
16.	*C. fragariae* (Oudem.) Sutton	*Eucalyptus*	019 888	262984 278252	
17.	*C. granati* (Sacc.) Petrak & Syd.	*Eucalyptus*	1044	280238	3501
18.	*Corticium salmonicolor* Berk. & Br.	*T. grandis* *A. triphysa* *G. arborea* *B. ceiba* *Eucalyptus*	78 108 203 609 33		
19.	*C. rolfsii* Curzi	*B. ceiba* *Eucalyptus*	1066 607	280236 280239	
20.	*Corynespora cassiicola* (Berk. & M. A. Curtis) Wei	*G. arborea*	861	280237	
21.	*Cryphonectria cubensis* (Bruner) Hodges	*Eucalyptus*	015 034 036-39 041 042 044 1061 1062 014	261569 274337 -339-42 -344-45 274347 281618 254084	
22.	*C. gyrosa* (Berk. & Br.) Sacc.	*Eucalyptus*	513 033 040 043 1059	261575 274343 274346 281618 274336	3532
23.	*Cylindrocarpon lucidum* Booth	*Eucalyptus*	640	267023	
24.	*Cylindrocladium clavatum* Hodges & May	*Eucalyptus*	484 517	260694 270185	3526 3527

Contd.

1	2	3	4	5	6
25.	*C. camelliae* Venkataramani & Venkataram	*Eucalyptus*	128b	262974	3525
26.	*C. curvatum* Boedijn & Reitsma	*Eucalyptus*	127	246474	
21.	*C. floridanum* Sobers & *C.* Seymour	*Eucalyptus*	025 127a 894	250219 250220 276603	3528
28.	*C. ilicicola* (Hawley) Boedijn & Reitsma	*Eucalyptus*	005 008 001 002 010 012	250214-15 250212-i3 250216-17	
29.	*C. parvunz* Anderson	*Eucalyptus*	128	250221	
30.	*C. guinqueseptatum* Boedijn & Reitsma	*Eucalyptus*	080 014 22l 977 579-84 1075	246473 250318 250223 276612 200728-33 280742	 3503 3529
31.	*C. scoparium* Morg.	*Eucalyptus*	125		
32.	*C. theae* (Petch) Alf. & Sob.	*Eucalyptus*	1082(1) 1082(2) 1063(1) 1100 1079 1091 495(D) 1102	280734-37 280739-40 280741	 3531
33.	*Cytospora eucalypti* sp. nov.	*Eucalyptus*		261564	
34.	*C. eucalypricola* van der Westhuizen	*Eucalyptus*	1147	284046	
35.	*Fusarium moniliforme* var. *subglutinans* Wollenw. & Reink	*O.pyramidale*	389b	257550	
36.	*F. oxysporum* Schlecht	*Eucalyptus* *E. grandis*	723 1136	269013 284047	3516 3518

Contd.

1	2	3	4	5	6
37.	*F. solani* (Mart.) Sacc.	*G. arborea*	486	260689	
38.	*Griphosphaeriagmelinae* sp. nov.	G. *arborea*	810	261570	
39.	*Guignardia citricarpa*Kiely	*Eucalyptus*	1214	287783	
40.	*Hysterium angustatum* Alb. & Schwein.	*Eucalyptus*	309	257895	
41.	*Lentinus squarrosulus* Mont.	*Eucalyptus* *G. arborea*	22,23	RBG	
42.	*Macrovalsaria megalospora* (Mont.) Sivan.	*Eucalyptus*	201	261566	
43.	*Marasmiellus ignobilis* (Berk. & Br.) Pegler	*T. grandis*	017	RBG	
44.	*Microporus xanthopus* (Fries) 0.Kuntze	*Eucalyptus*	24,25	RBG	
45.	*Meliola ailanthii* sp. nov.	*A. triphysa*		270190b	
46.	*Myrothecium roridum* Tode ex Fr.	*B. ceiba*	104	246451	3510
47.	*Nattrassa toruloidea* (Nattrass) Dyke & Sutton	*Eucalyptus*	110	261565	
48.	*OIivea tectonae* (T. S.& K. Ramakr.) Mulder	*T. grandis*	131	273439	
49.	*Pestalosphaeria elaedis* (Booth & Robertson) van der Aa	*Eucalyptus*	516	268335	
50.	*Pestalotiopsis disseminata* (Thum.) Steyaert	*Eucalyptus*	336	268328	3497
51.	*P. guepinii* (Desm.) Steyaert	*Eucalyptus*	333 338 530	268324 268327 268336	3506 3498
52.	*P. mangiferae* (P. Henn.) Steyaert	*Eucalyptus*	551 521 520 638 572	268326 268330 268334 268337-38	

Contd.

1	2	3	4	5	6
53.	*P. neglecta (Thum.)* Steyaert	*Eucalyptus*	328 374 570	268329 268331 268333	3505
54.	*P. versicolor* (Speg.) Steyaert	*Eucalyptus*	313	268332	
55.	*Phaeoseptoria eucalypti* (Hansf.) Walker	*Eucalyptus*	108 15	246482 246483	
	Phialophora richardsiae (Nannf.) Conant	*T. grandis*	390	257551	3572
57.	*Phoma nebulosa* (Pers. ex S. F. Gray) Berk.	*G. arborea*	403	260690	
58.	*Phomopsis variosporum sp.* nov.	*T. grandis*	641	269014	3499
59.	*Phyllachora dalbergiae* Niessl	*D. iatifolia*	060	293352	
60.	*Physalospora daibergia* sp. nov.	*D. latifolia*	247	286904	
61.	*Pseudocercospora ranjita* (Chaudhury) Deighton	*C. arborea*	579	269020	
62.	*Pseudoepicoccum tectonae* sp. nov.	*T. grandis*	1150	286905	3521
63.	*Pseudomonas* sp. (possibly *P. solanacearum)*	*T. grandis* *A. triphysa*	B106 B210		
64.	*Pythium sp.*	*A. triphysa*	74		
65.	*P. deliense* Meurs	*Eucalyptus*	629 1019	267018 281614	
66.	*P. myriotylum* Drechsler	*Eucalyptus*	714 703	268319 268320	3515
67.	*P. spinosum* Saw.	*Eucalyptus*	997 999 951 1000	276605 276608-9 276611	
68.	*Rhizoctonia solani* Kuhn state of *Thanatephorus cucumeris* (Frank.) Donk	*Eucalyptus* A. *triphysa* *B. ceiba*	637 396 1042	267022 257894 280235	3517
69.	*Sclerotium rolfsii* Sacc.	*Eucalyptus*	819	271722	

Contd.

1	2	3	4	5	6
70.	*S. rolfsii Sacc. state* of *Corticium rolfsii* Curzi	*Eucalyptus* *T. grandis*	430 752 392	260963 269010 160693	3524 3500
71.	*Scytalidium state* of *Nattrassa toruloidea*	*Eucalyptus*	602	267017	
72.	*Spiropes capensis* (Thum.) M. B *Ellis*	*hyperparasite* on *Meliola ailanthii*	025a	270190a	
73.	*Torula sp.*	*Eucalyptus*	I99		
74.	*Thyronectria pseudotricha* (Schw.) *Seeler*	*Eucalyptus* *G. arborea*	11 009	252784 257897	
75.	*Uncinula tectonae Salm.*	*T. grandis*	201		
76.	*Uredo bombacis Petch*	*B. ceiba*	057 058	293349 293350	
77.	*Uredo sissoo Syd.* & Butler	*D. latifolia*	130	273438	
78.	*Valsa eucalypti Cooke* & Harkness	*Eucalyptus*	0l0b	261564b	
79.	*Valsa eucalypticola* sp. nov.	*Eucalyptus*	028 014	257896 261568	

IMI No.: Herbarium Number, Commonwealth Mycological Institute, Kew, Surrey, U. K.

ITCC No.: Indian Type Culture Collection Number, Indian Agricultural Research Institute, New Dclhi, India.

KFRI No.: Culture Accession Number, Kerala Forest Research Institute, Peechi, Kerala, India.

RBG: Royal Botanic Gardens, Surrey, U. K.

SUBJECT INDEX