

DISEASES OF ALBIZIA FALCATARIA IN KERALA AND THEIR POSSIBLE CONTROL MEASURES

J.K.Sharma
K.V.Sankaran



KERALA FOREST RESEARCH INSTITUTE
PEECHI, THRISSUR

March 1987

Pages: 50

CONTENTS

	Page
Summary	1 r.47.2
Introduction	2 r.47.3
Review of Literature	3 r.47.4
Materials and Methods	6 r.47.5
Results and Discussion	14 r.47.6
References	44 r.47.7

SUMMARY

A total of five diseases were recorded during the survey conducted in numerous nurseries and five representative plantations of *Albizia falcataria* in Kerala. In nurseries only two diseases viz. web blight caused by *Rhizoctonia solani* and seedling wilt caused by *Fusarium solani* were observed. Of these, web blight was recorded commonly and it caused considerable mortality of seedlings in patches, if appeared within a month of emergence; seedlings > 3-month-old resisted the infection as it caused only premature defoliation. Two aerial strains of *R. solani* were found associated with the web blight. In saprophytic phase, the linear growth of the fungus was greatly affected by the moisture content of soil. In parasitic phase, penetration of leaves by the fungus took 12 h after the leaves were covered with the web of mycelium. Studies on incidence and spread of web blight in relation to isolate of *R. solani*, inoculum level and age of seedlings, indicated that isolate 783 was more aggressive than isolate 766 as it caused high mortality within a short period; younger (60-day-old) seedlings were found to be more susceptible than mature (75-day-old) seedlings. Disease severity did not differ significantly in two inoculum levels (1:50 and 1:200 on w/w basis, inoculum to soil). Of the 13 fungicides evaluated *in vitro* against two isolates of *R. solani*, Bavistin and Terraclor Super-X gave the maximum inhibition in growth. However, *in vivo* only Bavistin (1000 μ g a. i./ml), applied 1 wk before transplanting the seedlings in the infested soil, controlled the disease caused by both the isolates. Bavistin applied after the appearance of the disease was not very effective; Terraclor Super-X did not control the web blight at any stage.

Of the three diseases, namely Botryodiplodia die-back (*B. theobromae*), Phomopsis shoot die-back (*P. mendax*), and bacterial wilt (*Pseudomonas solanacearum*) recorded in plantations, only Botryodiplodia die-back was the most serious disease prevalent in all the *Albizia* growing areas of the state. Large-scale die-back of trees in patches due to girdling of stem by the progressing canker was recorded in Kattilappara-1980 and Nangachee-1974 (Thenmala For. Div.), Keezhayam-1979 (Punalur For. Div.) and Kollathirumedu-1979 (Vazhachal For. Div.) plantations. The incidence of die-back varied from nil (Vamanapuram-1980, to 66% (Kattilappara-1980) in 1983. It gradually declined to 13 to 25% over the next three years while the severity remained low throughout in these plantations. Intensive observations on progress and

spread of die-back in a plot with moderately severe infection indicated that the high incidence occurred during the dry-warm period, but during or just after the monsoon it declined as some of the affected trees recouped partially or completely; thus, the overall incidence gradually declined from 94.3% in June 1983 to 69.8% in May 1985. However, the percentage of mortality of the affected trees increased from 8.8% to 30.3% during the same period,

Phomopsis shoot die-back, reported from plantations affected by fire and bacterial wilt only from one plantation at Thundathil (Malayattoor For. Div.) were not common diseases.

INTRODUCTION

The genus *Albizia* comprises about 100 species, of which 14 occur naturally in India. So far only *A. falcataria* (L.) Fosberg and *A. lebbek* Benth. have been taken up for large-scale planting programmes around the world. *A. falcataria* syn. *A. moluccana* Miq., a native of Moluccas, New Guinea, New Britain and Solomon Islands, was introduced to South-East Asia, Burma and Philippines during 1870s (Anon., 1979). It is one of the fast growing tree species in the world suited for humid tropics, growing best on deep well drained, fertile, alkaline soils. In Kerala, planting of *A. falcataria* under afforestation programmes was initiated during the mid 1970 and so far 1350 ha of plantations have been raised by the Kerala Forest Department and Kerala Forest Development Corporation, mostly as monoculture and occasionally in mixture with *Ailanthus triphysa* (Denst.) Alston and *Bombax ceiba* L.

In India, pink disease caused by *Corticium salmonicolor* Berk. & Br. in plantations and web blight of seedlings by *Rhizoctonia solani* Kuhn. have earlier been reported on *A. falcataria* (Subba Rao, 1942; Agnihothrudu, 1962) In Kerala, pest arid disease problems came to forefront soon after the large-scale planting of *A. falcataria* began in 1974. First, a severe infestation of a bagworm, *Pteroma plagiophleps* Hampson was noticed in 1977 in a 3-year-old plantation at Vazhccchal (Vazhachal For. Div.) where it caused total defoliation in 5 ha of a 20 ha plantation (Nair *et al.*, 1981). A few years later in 1980 a die-back of *A. falcataria*, which caused extensive damage, was recorded at

Nangachee (Thenmala For. Div.) and Vazhachal respectively in 8- and 6-year-old plantations. The same year serious mortality of seedlings was also recorded in a nursery at Vazhachal. Since no information was available on diseases of *A. falcataria* in Kerala, studies were taken up to prepare a checklist of diseases in nurseries and plantations, to assess the level of infection of serious diseases and to work out control measures for diseases of major concern.

REVIEW OF LITERATURE

Though the amount of information available on the diseases of *Albizia* spp. is unusually large, they are known to be attacked by relatively few fungal diseases of significant importance (Gibson, 1975). Diseases of seedlings and root diseases of young plants are relatively few but a number of root and stem pathogens are recorded from older trees. A total of 15 diseases have been recorded on *A. falcataria* with which one algal and 27 fungal organisms are associated (Table I). Of these, eight diseases, namely Botryodiplodia root infection (*), violet root rot, Aglaospora root rot, Fomes stem canker (*), charcoal stump rot (*Ustilina zonata*), Phoma die-back, Macrophoma stem infection and foliar necrosis have been reported exclusively from India. Diseases recorded commonly in India and elsewhere are charcoal stump rot (*U. dusta*), die-back (*Botryodiplodia theobromae*): pink disease, leaf cast and web blight.

Though five diseases including those marked above with an asterisk and Botryodiplodia die-back, pink disease and leaf cast have been recorded on *A. falcataria* from Kerala (Subba Rao, 1939, 1942; Venkataram. 1950), precise details of these diseases are lacking. In most cases these reports include only occurrence and symptoms with either no mention of incidence/severity or it is described very vaguely; for some diseases even the symptoms are not described. Among the diseases recorded in India and elsewhere some account is available for pink disease and web blight. A high incidence of pink disease has been reported in 1-year-old trees from Assam (Agnihotrudu, 1982) In the Philippines, Eusebio *et al.* (1979) observed pink disease as the most serious disease of *A. falcataria*. An average of 76% trees were found infected with four or more infection points on stem in seven different localities. They indicated that if the disease is not contained it might affect the plantation development programme considerably. Web blight was also reported by Agnihotrudu (1962) in the same plantation in Assam where the pink disease occurred. It was observed that it attacked several 1-year-old trees and

Table 1. Diseases of *Albizia falcataria* recorded in India and other countries

Disease	Pathogen	India	Countries other than India
ROOT			
1 Root infection	<i>Botryodiplodia theobromae</i> Pat.	Wynad, Kerala (Venkataram. 1960)	—
2 Root rot	<i>Aglaospora</i> Sp.	North-East India (Sarmah, 1960)	—
	<i>Armillariella mellea</i> (Fr.) Karst.	—	Indonesia (Java), Tanzania, Zaire (Anon., 1950)
	<i>Ganoderma lucidum</i> (Leyss.) Karst.	—	Sri Lanka (Browne, 1968)
	<i>G. pseudoferreum</i> (Wakef.) Overeem	—	Sri Lanka (Bertus, 1961)
	<i>Irpex subvinosus</i> (Rerk. & Br.) Petch.	—	Sri Lanka (Browne, 1968)
	<i>Poria hypolateritia</i> (Berk.) Cooke	—	Sri Lanka (Browne, 1968)
3 Brown root rot	<i>Fomes noxius</i> Corner	—	Sri Lanka (Browne, 1968)
4 Black root rot	<i>Macrophomina phaseolina</i> (Tassi) Goid.	—	Sri Lanka, Indonesia (Java) (Steinmann, 1928), Uganda (Browne, 1968; Spaulding, 1961) North Africa (Scharif, 1964)
5 Purple root rot	<i>Helicobasidium compactum</i> Boedijn.	—	Indonesia (Spaulding, 1961)
6 Violet root rot	<i>Sphaerostilbe repens</i> Berk. & Br.	North-East India (Sarmah, 1960)	—
STEM			
7 Canker	<i>Botryodiplodia theobromae</i> Pat.	—	Sri Lanka (Browne, 1968)
	<i>Fomes</i> sp.	Peermade, Kerala (Subba Rao, 1939)	—
	<i>Nectria pulcherrima</i>	—	Sri Lanka (Bertus, 1961)

8	Charcoal stump rot	<i>Ustulina zonata</i> (Lev.) Sacc.	North-East India (Sarmah, 1960)	—
		<i>U. deusta</i> (Fr.) Petrak.	” ”	Indonesia (Java) (Anon., 1937)
9	Die-back	<i>Botryodiplodia theobromae</i> Pat, <i>Phorna</i> sp.	Wynad, Kerala (Venkataram, 1960) Nilgiris, Tamil Nadu, (Venkataram, 1964, 1966)	Indonesia (D' Angremond, 1948)
		<i>Physalospora rhodina</i> (Berk & Curt.) Cke. <i>Thyridaria tarda</i> Bancroft	— —	Indonesia (Sumatra) (Spaulding, 1961) Madagascar (Spaulding, 1961)
10	Pink disease	<i>Corticium salmonicolor</i> Berk. & Br.	Peermade, Kerala (Subba Rao, 1942)	Philippines (Eusebio <i>etal.</i> , 1979)
11	Stem infection	<i>Macrophoma theicola</i> Petch.	North-East India (Sarmah, 1960)	—
LEAF				
12	Foliar necrosis	<i>Camptorneris albizziae</i> (Petch.) Mason	Annamalai, Tamil Nadu, Assam (Venkataram, 1965)	—
13	Leaf spot	<i>Cephaleuros virescens</i> Kunz (Algae)	—	Malayasia (Sharples, 1930)
	Leaf cast	<i>Cercospora theae</i> Petch	Peermade, Kerala (Subba Rao, 1939)	Sri Lanka (Gadd, 1927, 1952)
	Yellow-brown spot	<i>Pleiochaeta albizziae</i> (Petch) Hughes	—	Indonesia (Java, Sumatra) Sri Lanka (Webster, 1952)
14	Powdery mildew	<i>Oidium</i> spp.	—	Indonesia (Java) (Bernard, 1926)
15	web blight	<i>Rhizoctonia Solani</i> Kuhn State of <i>Thanatephorus cucumeris</i> (Frank.) Donk	Assam (Agnihotbrudu, 1962)	Sri Lanka (Browne, 1968)

caused extensive defoliation. Elsewhere web blight has been recorded only from Sri Lanka (Browne, 1968).

A few diseases of other *Albizia* spp in India and other countries have also been recorded on *A. falcataria*. *A. falcataria* may not have any special susceptibility to the following pathogens which are associated with various diseases, namely *Armillariella mellea* (Bates, 1961), *Ganoderma lucidum* (Spaulding, 1961; Toole, 1966; Browne, 1968; Bakshi *et al.*, 1972; Gibson, 1975), *Macrophomina phaseolina* (Steinmann, 1928; Spaulding, 1961; Scharif, 1964; Browne, 1968), *Botryodiplodia theobromae* (D. Angremond, 1948), *Camptomeris albizziae* (Spaulding, 1961; Browne, 1968; Bakshi, *et al.*, 1972), *Cercosporella theae* (Gadd, 1927; Subba Rao, 1939) and *Oidium* sp. (Gadd, 1927); except foliar necrosis caused by *C. albizziae* others have a wide host range. From India only *Ganoderma* root rot (*G. lucidum*) recorded on *A. chinensis* (Bakshi *et al.*, 1972), *A. procera* and *A. lebbek* (Browne, 1968) and *Cercosporella* leaf spot (*C. theae*) on *A. lophantha* (Subba Rao, 1939) are known to occur on *A. faicataria*.

MATERIALS AND METHODS

DISEASE SURVEY

Nursery

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. Besides the date of appearance of disease, other relevant information pertaining to nursery practices such as sowing date, quantity of seeds per standard bed, watering schedule, type of shade, were collected from the field staff. Appropriate specimens of diseased seedlings were collected for isolation of the causal organism.

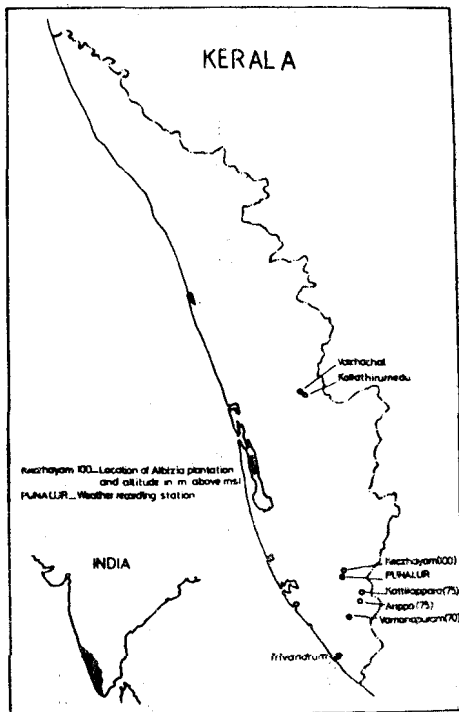


Fig 1. Location of plantations of *Albizia falcataria* in Kerala surveyed for disease occurrence.

Plantations

For assessing the disease situation, initially a reconnaissance was undertaken in most of the plantations of *Albizia falcataria* in Kerala. Based on these observations representative plantations of different age groups with some disease potential, easy accessibility and workable terrain were selected in southern Kerala (Fig. 1).

In each plantation, five observation plots of 15 x 15 trees (spacing 2m x 2m) were selected at random and trees in alternate rows paint marked. Thus in each plot observations were confined to only 120 trees out of a total of 225. However, at Keezhayam each observation plot consisted of 15 x 20 trees totalling 900 in three plots. Here all the trees were observed for incidence and severity of die-back. Observations on incidence and severity were recorded once in 1983, three times in 1984 and once in 1985.

Severity and incidence of a disease: Severity of a disease was rated on a numerical scale (1-5) of disease rating index as given in Table 2. The average severity index of a disease (DSI) in a plantation was calculated as follows:

$$\text{Average disease severity index (DSI)} = \frac{nL \text{ XI} + nM \text{ x2} + nMS \text{ x3} + nS \text{ x4} + nHS \text{ x 5}}{N}$$

Table 2. Disease index to assess the severity of diseases in plantations

Disease severity rating (DSR)	Disease severity index (Scale 0-5)	Symptoms	
		Shoot die-back % of shoots showing symptoms	Stem canker and die-back
Nil	0	Nil	Nil
Low (L)	1 (0. 1-1)	u p to 10%	1 canker, no apparent harm to tree, yellowing of leaflets, thinning of crown due to premature defoliation.
Moderate (M)	2 (1. 1--2)	> 10% to < 25%	1-2 cankers, die-back of up to 25% branches
Moderately severe (MS)	3 (2. 1-3)	> 25% to < 50%	1-2 cankers, die-back of up to 50% of branches.
Severe (S)	4 (3. 1-4)	> 50% to < 75%	1-2 cankers, die-back of up to 75% branches, epicormic shoots present.
Highly severe (HS)	5 (4. 1--5)	>75%	1-2 cankers > 75% branches dead, epicormic shoots dying, tree partially or completely dead.

where nL, nM, nMS, nS, represent total number of plants in all the observation plots with low, medium, moderately severe, severe and highly severe disease severity rating (DSR) and N total number of trees assessed in all the observation plots.

Percentage incidence of a disease in a plantation was calculated as a ratio of the total number of plants affected to the total number of plants observed in all the plots.

Progress and spread of die-back: For recording intensive observations on the progress and spread of die-back over a period of two years (1983-1985), a plot (14 x 30 trees) having high disease incidence (HD plot) was selected in 1980 plantation at Kattilappara containing a total of 152 trees; the remaining trees died due to die-back had already been removed by the local people. In June 1983 when the first observation was recorded 18 trees had already died due to die-back. Observations on progress of incidence and severity of die-back were recorded at an interval of 5 mo, except the second one which was after 3 mo.

Isolation and identification of causal organisms

Disease specimens collected from the field were brought to the laboratory in polythene bags. To avoid any saprophytic growth over the specimens, isolations were made within a week of collection. For isolation potato dextrose agar medium was used for fungi and nutrient agar for bacteria.

Diseased leaflets, pieces of rachis and tender stem were surface sterilized in 0.1% mercuric chloride for 2 min and washed in six changes of sterile water. Woody specimens were only flamed for a few seconds. These were plated on the medium and incubated at $25 \pm 2^{\circ}\text{C}$ for 1 to 2 wk. After isolating the causal organisms in pure culture, identification was attempted atleast up to generic level, based on cultural and morphological characteristics. For specific identification or confirmation the cultures were referred to CAB International Mycological Institute, Surrey, U.K.

Pathogenicity tests

The pathogenicity of the isolates was confirmed in artificial inoculation trials. For seedling diseases the tests were undertaken in the laboratory and for stem diseases in the field. 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 humidity chamber.

Seedling disease

Web blight: For testing pathogenicity of an isolate, inoculum was raised on sand-corn meal in culture bottles at $25 \pm 2^{\circ}\text{C}$ for 7 wk. Mycelial mats containing abundant microsclerotia were harvested from the culture bottles, air dried for

12 h and blended in a waring blender. For infesting the soil, 10g of this inoculum was mixed thoroughly with 2 kg of steam sterilized soil and transferred to an aluminium tray (30 x 30 x 5 cm) and incubated for 1 wk in the laboratory at $30 \pm 2^{\circ}\text{C}$. The soil in the tray was kept moist by spraying about 100 ml of sterile water every day. Seedlings of *A. falcataria* were raised from pre-soaked seeds in steam sterilized soil in large trays kept outdoors. Healthy seedlings aged 4 wk (5-8 cm in height) and 6 wk (11-18 cm in height) were pulled out gently and their roots thoroughly washed in sterile water. Thirty seedlings of one age group were planted 2.5 to 3 cm apart in each of the replicate trays. Separate trays were sown with 40 seeds each. Controls without inoculum were also maintained. Each set had three replicate trays. Two trays of each set were placed in a humidity chamber while one was kept on the laboratory bench. During the period of the experiment in the humidity chamber the r. h. varied from 92 to 100% and temperature 23 to 33°C while in the laboratory these were $40.5 \pm 65\%$ and $27.5 \pm 34^{\circ}\text{C}$, respectively. Observations on the disease development were recorded daily.

Tree diseases

Botryodiplodia die- back: For testing the pathogenicity of the isolate 3-year-old healthy trees of *A. falcataria* were selected in a 1981 plantation at Kattilappara where the disease incidence was very low. The bark of stem/root was cleaned with absolute alcohol and sterile water and inoculated either with or without wound. Wound inoculation was carried out in two ways, In one method, an inverted 'V'-shaped 1 cm deep cut was made in the bark with a sharp sterile chisel (2.5 cm wide). The cut flap was pulled gently and an agar disc (9 mm dia) bearing mycelium and fructifications from a 10-day-old culture of the isolate inserted between the bark flap and sapwood and flap pressed back gently. A sterile moist cotton swab was placed over the wound. In the second method, a wound of 1.5 cm^2 made in the bark was inoculated and the inoculated area covered with a sterile moist cotton pad. In inoculation without wound, a disc bearing mycelium and fructifications was placed upside down over the bark and covered with a moist sterile cotton pad. The site of inoculation was covered with a polythene sheet, the edges of which were sealed in close contact with the bark using beeswax. Control inoculations were made in the same way using PDA disc without the test fungus. The stem and roots of ten trees each were used for each type of inoculation during the dry period (February) and wet period (September/October). Observations on the infection and appearance of symptoms were recorded at frequent intervals.

Manifestation of disease through fire injury: Considering the susceptible nature of *A. falcataria* to fire, which results in injury at the basal part of the stem

and high incidence of die-back in plantation with a history of fire, manifestation of the disease through fire injury was also investigated. Five 2-year-old healthy trees of *A. falcataria* having similar girth (18-20 cm) were selected in an experimental plot in the Institute campus in February and inoculated after causing fire injury artificially. Inoculum of the pathogen was raised on dried tapioca (*Manihot utilissima* Pohl) stem chips with sand-corn medium. Tapioca, grown as a taungya crop (agriculture crop grown in forest plantations during the first few years of establishment) during the first 2-3 years in the plantations, was used as it was found to support pure luxuriant growth of *B. theobromae* (Fig. 2),

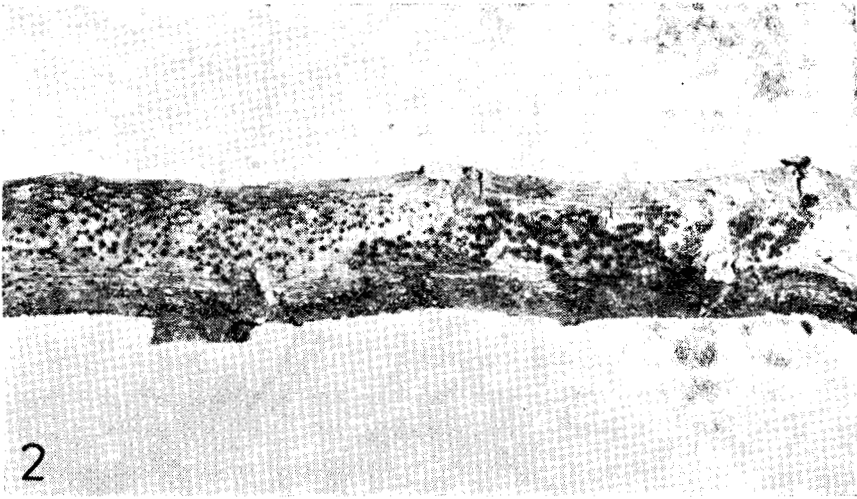


Fig- 2. A piece of tapioca (*Manihot utilissima*) stem, collected from an *Albizia* plantation, colonized by *Botryodiplodia theobromae*.

the possible causal organism of die-back of *A. falcataria*. One-month-old culture with profuse mycelial growth and abundant pycnidia was utilized in the experiment. For causing fire injury on one side of the stem, a stem guard (30 cm in length) made up of steel (2 mm gauge) was used. Each guard had a slit 10 cm long and 2 cm wide on one side. The guard was placed around the base of the stem in such a way that it did not come in contact with the stem. Equal amount of dried leaf litter was placed around the guard up to a height of 15 cm. The litter was lit and allowed to burn with flames for 2 min after which the fire was extinguished and stem guard removed. The bark at the place of slit in the stem guard appeared injured and charred with vertical fissures at some places; the part of the stem covered with the guard remained unaffected. The following day equal quantity of inoculum mixed with freshly dried chips of tapioca stem was placed up to 10 cm height around the base of the stem of

each tree and covered with a layer of moist soil. Water was sprinkled over the soil for a week to keep it moist. Observations on the development of infection were recorded every month.

Phomopsis die-back of shoots: A month old culture of the test fungus with abundant pycnidia was utilised for the pathogenicity tests. The shoots of 2-year-old *A. falcataria* at Kattilappara were wound inoculated by making inverted 'V' shaped cut in the bark and placing over it 9 mm disc from the culture containing abundant pycnidia. The inoculated part of the stem was covered with a sterile cotton swab and wrapped with a polythene sheet. Both the ends of the polythene were closed tightly with a twine. Suitable controls were also maintained without inoculum.

Host-parasite relationship studies of web blight pathogen

Infection process

Growth of mycelium over the leaves and infection processes were studied on 1-month-old seedlings of *A. falcataria* infected in artificial inoculation trials as detailed earlier.

Light microscopy: Leaves bearing different stages of mycelial growth were removed from the infected seedlings and mounted in lactophenol cotton blue under long coverslips. Separate slides were prepared for upper and lower leaf surfaces. Observations were recorded using a Leitz Dialux-20 microscope and photographs taken with Vario orthomat camera.

Scanning electron microscopy (SEM): Appropriate specimens were prepared for SEM after freeze drying and coating them with gold under vacuum. The specimens were observed under Hitache S-540 scanning electron microscope.

Influence of *R. solani* isolates, inoculum level and age of seedlings on severity of web blight.

Similar procedures for preparation of inoculum, infesting the soil and raising the seedlings as described under pathogenicity test were followed in this experiment.

Seedlings: Seedlings of *A. falcataria* aged 60 and 75 days were utilised. There were 30 seedlings in each replicate tray.

Rhizoctonia isolates: Two isolates of *R. solani* (KFRI Acc. No. 766 and were used in the study. Both the isolates were obtained by direct

isolation from the blighted foliage of seedlings collected from Vachumaram (Kollathirumedu For. Range) and Punalur (Anakulam For. Range).

Inoculum level: Two inoculum levels i.e., 10 and 40 g per 2 kg (1:200 and 150 on w/w basis respectively) were adjusted in the soil.

There were two replicate trays for combinations of different variables (isolate, inoculum level and seedling age). Appropriate controls were maintained with non-infested soil. Each tray was watered daily with 200 ml of water.

Observations on incidence and spread of web blight were recorded daily till the fifth day of incubation and later on alternate days till the eleventh day. At each observation seedlings having following symptoms associated with different stages of development of web blight were counted separately and percentage calculated.

Developmental stage	Web blight symptom
I	Growth of mycelium from soil to stem
II	Spread of mycelium from stem to first basal leaf
III	Spread of mycelium to second basal leaf
IV	Lateral spread of mycelium from one seedling to another
V	Seedlings dead

Statistical analysis of the data was carried out after appropriate transformations. This was subjected to three factor analysis of variance (Calinski and Corsten, 1985) to find out significant differences among different variable combinations,

Effect of soil moisture on the spread of *R. solani*

R. solani isolate 783 was used for studying the spread of mycelium under different soil moisture regimes in sterile soil. Since in *in vivo* chemical trials sterile soil was used this experiment was also to find out whether there was erratic growth of mycelium in sterile soil. As described earlier the fine-sieved soil was steam sterilised and after cooling 2 kg of it transferred to each aluminium tray. For maintaining different moisture levels 400, 500, 600, 700 and 800 ml of sterilised water was poured in separate trays which gave moisture percentages of 18, 19.8, 23.6, 25.8 and 30.2 respectively. Two replicates were kept for each moisture level. In the centre of each tray a 10 mm dia disc, taken from the margin of an actively growing colony of the fungus, was placed upside down with the mycelial side in contact with the soil. Trays were incubated in a humidity chamber maintained at about cent percent relative humidity; the temperature ranged from 26.5 to 33.5⁰C

during the incubation period. Observations on the radial spread of mycelium in soil were recorded daily up to the tenth day using a magnifying lens at three places in each replicate tray for a week.

A quadratic function was fitted to the data to represent the relation between moisture regimes and growth index. The growth index was taken as the value in equation, $Y = \alpha + \beta / X$, where Y is the radial growth and X number of days (Snedecor and Cochran, 1967)

Chemical control of web blight

Laboratory screening of fungicides

In vitro studies

Thirteen fungicides, namely Bavistin (carbendazim), Benlate (benomyl), Daconil 2787 (chlorothalonil), Difolatan (Captafol), Dithane M-45 (mancozeb), Emisan-6 (MEMC), Fytolan (copper oxychloride), Hexacap (Captan), Tecto (thiabendazole), Terraclor Super-X (quintozene + etridiazole), Terrazole (etridiazole) Topsin-M (thiophanatemethyl) and Vitavax (carboxin) were evaluated for their efficacy against two isolates of *Rhizoctonia solani* (KFRI Acc. Nos. 766 and 783), the web blight pathogens, following poison-food technique and soil method. LD₁₀₀, where the growth of the fungus was completely inhibited, was alone taken as the effective dose of a fungicide.

Poison-food technique: To obtain a desired concentration, an appropriate quantity of the test fungicide was mixed thoroughly with the sterilised 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 (8 mm dia.) taken from the margin of an actively growing colony of the test fungus. Inoculated Petri dishes were incubated at $25 \pm 2^\circ\text{C}$ and three to four observations of radial or diameter growth of the colony recorded till the fourth day, when the colonies in controls reached nearly to the periphery of the dish.

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 microsclerotia. The procedure has been detailed earlier by Sharma *et al.* (1985).

In vivo studies

The efficacy of the two most effective fungicides i. e., Terraclor Super-X (1170 and 2340 µg a. i./ml) and Bavistin (1000 and 2000µg a. i./ml) in soil

method was further tested in *in vivo* studies utilising both the isolates (766, 783) of *R. solani*. With the objective of standardising the time of fungicidal application in nursery beds three treatments were planned as follows:

- T I- First application of fungicide just after transplanting the seedlings in the infested soil and the second after 12 days.
- T II- First application of fungicide 5 days after transplanting the seedlings in the infested soil followed by the second after 7 days
- T III- One application of fungicide 6 days before transplanting the seedlings in the infested soil.

Procedure for preparation of infested soil was the same as described under pathogenicity test. For each treatment, two trays each containing 20 seedlings (7-week-old) were kept. Fungicide was applied by drenching the solution of appropriate concentration at different periods as shown above. Observations on percent seedlings affected and dead were recorded daily up to tenth day and later at different periodicities. The data were subjected to angular transformation and analysed statistically using 4-factor unweighted ANOVA (Keppel, 1973).

RESULTS AND DISCUSSION

Nursery diseases

In nurseries only two diseases viz. web blight and seedling wilt were observed. Of these, web blight was Common while seedling wilt only rarely observed.

WEB BLIGHT

Occurrence

Web blight was recorded in many nurseries surveyed during June-August. Highest incidence of web blight affecting >75% of seedlings (3-month-old) in seed beds was observed at Vachumaram (Kollathirumedu For. Range) during 1983. The mortality of seedlings due to web blight varied from locality to locality and it greatly depended on age of seedlings and their density; generally, it was high when the disease occurred in young seedlings (1-month to 2-month-old).

The disease appeared in seedbeds as irregular patches of web entangling seedlings. These patches enlarged rapidly from the periphery affecting the neighbouring healthy seedlings under high humidity and high seedling density. Occasionally, the disease covered the whole seedbed.

Symptoms

The disease was characterised by the formation of a web of mycelium which entangled a group of seedlings (Figs. 3, 4). Initially the infection caused flaccidity in healthy leaflets which was followed by development of water - soaked lesions. Gradually the infection also spread to the rachis resulting in drooping of the whole leaf. Soon leaves 'turned brown and premature defoliation and abscission of the rachis occurred. In most cases dead leaves covered with fungal mycelium could be seen hanging around the base of the stem. The disease spread in a seedling from lower to upper whorl of leaves and from seedling to seedling through contact. The higher the seedling density the greater was the spread of web blight in seedbed.

The younger seedlings (1 - to 2 - month - old) were killed outright due to infection but in the older seedlings only defoliation was observed.

Etiology

Rhizoctonia solani Kuhn. state of *Thanatephorus cucumeris* (Frank.) Donk (IMI 271579, 271880).

Pathogenicity

Within 24 h of incubation of trays in the humidity chamber, mycelium emerged from the infested soil and started to grow over the stem of all the test seedlings. At this stage the mycelium did not cause any apparent harm to the seedlings. Later, on the second day, the first lower leaf was attacked. The mycelium grew from the stem and became established on the leaflets. Initially, the mycelium grew epiphytically, however, within the next 12 h these leaflets showed flaccidity due to infection. Wilting of the leaflets occurred first and the whole leaf wilted soon after it was covered completely with a web of mycelium; wilted leaves gave a typical blighted appearance to seedlings (Fig. 3). The leaves of the younger seedlings were attacked earlier in comparison with older (seedlings as they were closer to the soil. Most of the 30-day-old seedlings died after 10, days of incubation and 45 - day - old after 15 days. The mycelium of the web, which was byaline in the beginning, turned light brown a few days later with abundant sclerotia developing, on it. The pathogen was reisolated from the infected seedlings

In laboratory, the growth of mycelium from soil to the stem of seedlings occurred only in a few cases. The spread of mycelium to the first lower leaf, observed only in 10% of the seedlings, was very slow as it took 10 days in comparison with two days in the humidity chamber. Further spread of the mycelium was not observed, probably due to unfavourable low humidity; none of the seedlings died.

The germination of seeds in infested soil occurred three days after sowing. The emerging seedlings appeared to be healthy in trays with infested soil, but within 24 h they were covered with fungal mycelium. Invading mycelium caused irregular, light brown, sunken, necrotic lesions on the cotyledons. Infected seedlings died within 3-4 days after emergence. The ungerminated seeds inside the soil, however, remained unaffected.

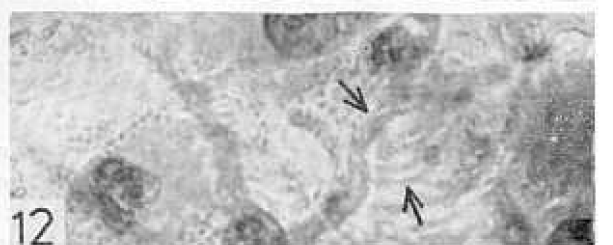
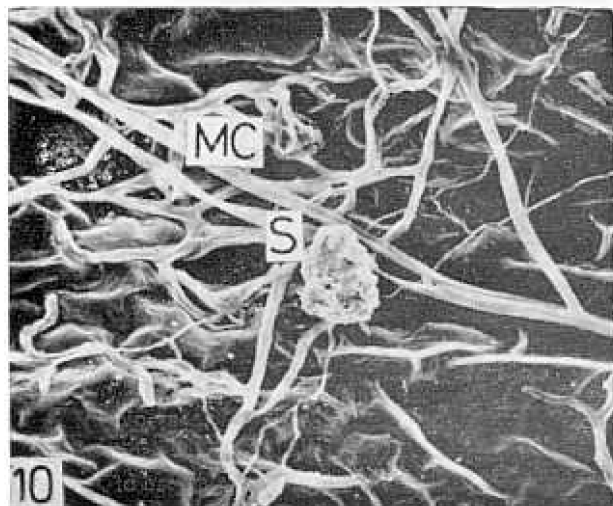
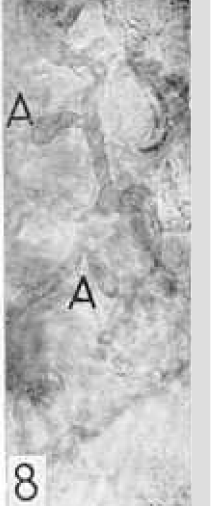
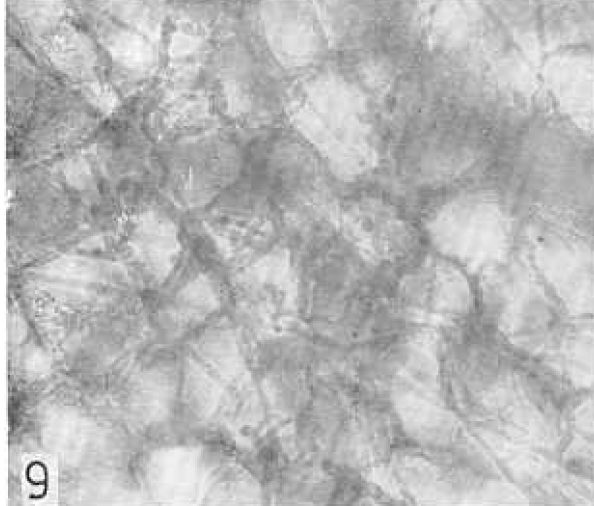
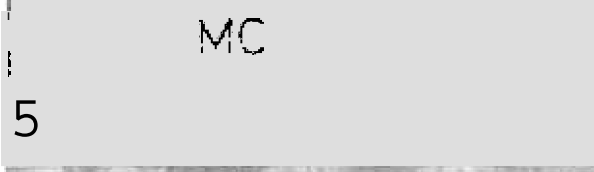
Host parasite relationship studies

Infection: The hyaline mycelia grew epiphytically over the epidermal cells of leaf touching the protuberances at the cell junctions. The hyphae of these mycelia were atypical in growth as compared to those seen on the agar medium as they grew in straight line with a very few branches at long intervals (Fig. 5). No definite pattern was observed in growth of these hyphae in respect to the orientation of the epidermal cells. The side branches emerged from these hyphae, formed a globular structure with dense cytoplasm upon touching the cell surface. This structure either gave rise to specialized infection structure, appressorium or mycelial cushion (Figs. 5, 6). Usually, the mycelial cushions were formed first and appressoria later. The appressoria were lobed, elongate, regular in outline, simple with dense cytoplasm (Figs. 7, 8). The mycelial cushions, made up of compact hyphae, usually developed at the junction of the cells. After the formation of these infection structures the side branches from the main hyphae branched profusely and formed a net of mycelium, pale yellowish to light brown in colour, over the leaf surface (Fig. 9). Commonly these hyphae followed the outline of a cell giving rise to mycelial net of different shapes (Figs. 9, 10). Some differences were observed on the upper and lower surfaces of the leaf. Upper surface of leaves had more mycelial growth than the lower. Appressoria on the lower surface were highly branched with bulbous lobes (Fig. 7) which were devoid of dense cytoplasm while on the upper they were mostly elongate with dense cytoplasm (Fig. 8). Also, fewer mycelial cushions, formed of loosely woven branched hyphae, were found on the lower surface.

Stomatal penetration was rarely observed (Fig. 11). In a number of instances either the appressorium or the growing mycelial tip was over the stomata



Figs. 3-4. Web blight of seedlings of *Albizia falcataria* caused by *Rhizoctonia solani*. 3, 60-day-old seedlings entangled with the mycelial web. Note the strands of hyphae arising from the soil (marked with an arrow) and climbing up the stem. 4, Overview of seedlings to show the spread of mycelial web from the stem to distal end of leaf.



Figs. 5-12. Infection of *Albizia* leaf by *Rhizoctonia solani*. 5, Early stages of formation of mycelial cushions (MC). Note two parallel running primary hyphae with a few branches at long intervals. 6, A magnified view of a mycelial cushion. 7, Formation of mycelial cushion on the lower leaf surface. Note vacuolated mycelium (VM) and appressorium (A) 8, Appressoria (A) on the upper leaf surface 9, A net of mycelia running along the cell walls. 10, SEM of lower leaf surface to show the course of mycelium through the cell walls. Note sclerotium (S) and young mycelial cushion (MC). 11, Appressorial penetration through the stoma (arrow). 12, Mycelium appears to avoid stomata (arrow).

but it seemed to make no effort in penetrating through it (Fig. 12). Penetration was direct through the epidermal cells. The appressoria gave rise to minute infection pegs which pierced through the cell wall and entered into the epidermal cell where it got enlarged in diameter. Branches emerging from these hyphae penetrated through the cell walls and infected the other adjoining cells. The chloroplast of the infected palisade cells got degenerated and turned brown and the cytoplasm disintegrated in to globular masses.

Influence of *R. solani* isolates, inoculum level and age of seedlings on severity of web blight

For both the isolates of *R. solani*, the treatment combinations were found to be significantly different in stage I, II and V of development of web blight and at stage III and IV. None of the factors and their interactions were significant (Table 3). In stage I, the interaction between isolate (I), inoculum level (L) and age of seedlings (A) i. e. I x L x A was significant at 5% level, the most susceptible combinations being 1 (766, 10, 60) and 4 (766, 40, 75). These combinations differed significantly from the others. In stage II also, the interaction between I x L x A was significant at 1% with combination 1 (766, 10, 80) alone differing from others at 5% level. At stage V, where the inoculum level (L) was not significant, combinations 1, 2, 3, 4, 5, 7 differed significantly from 6 and 8.

Table 3. Influence of *Rhizoctonia* isolates, inoculum concentration and age of seedlings on spread of web blight in *Albizia* seedlings

Combination	Factor			Mean Percent spread of web blight				
	Isolate (I)	Inoculum concentration (g) (L)	Age of seedlings (days)(A)	I*	II	III	IV	V
1	766	10	60	100a **	33.3a	13.7	11.4	8.5a
2	"	"	75	33.8b	15.5c	10.9	9.1	8.3a
3	"	40	60	28.2b	20.2b	8.2	7.3	7.5a
4	"	"	75	69.2a	22.5b	13.7	9.0	8.5a
5	783	10	60	39.8b	20.0b	9.1	8.4	8.8a
6	"	"	75	21.9b	11.1c	9.1	8.8	5.8b
7	"	40	60	47.9b	22.5b	3.1	8.4	8.0a
8	"	"	75	30.9b	11.1c	9.1	8.3	5.8b

*For explanation of developmental stage I, II, III, IV, V see p. 12

** Values superscribed by the same letter in each column are not statistically different

It is evident from the analysis of the data that isolate 783 was more aggressive than isolate 766, inoculum level of 10g caused more disease than 40g and 60-day-old seedlings were more susceptible than 75-day-old.

Effect of soil moisture on the linear growth of *R. solani*

Mthin 24 h of incubation the mycelium was seen growing rapidly inside as well as over the surface of soil. Due to high r.h. in the humidity chamber the aerial hyphae were seen impregnated with minute water droplets and, therefore, clearly visible as white cottony strands. The mycelial growth was found to be relatively slower in trays with 700 ml (25.8% moisture) and 800 ml (30.2%) of water as compared to 400 to 600 ml (18 to 23.6%) of water, thus indicating an adverse effect of high soil moisture on growth of *R. solani*. The regression of growth rate on soil moisture was significant with an F value of 9.914 ($p=0.05$). The predicted value of moisture regime at which maximum growth of *R. solani* is expected was 476.96 ml of water per 2kg of soil or at 18.95% soil moisture (Fig. 13)

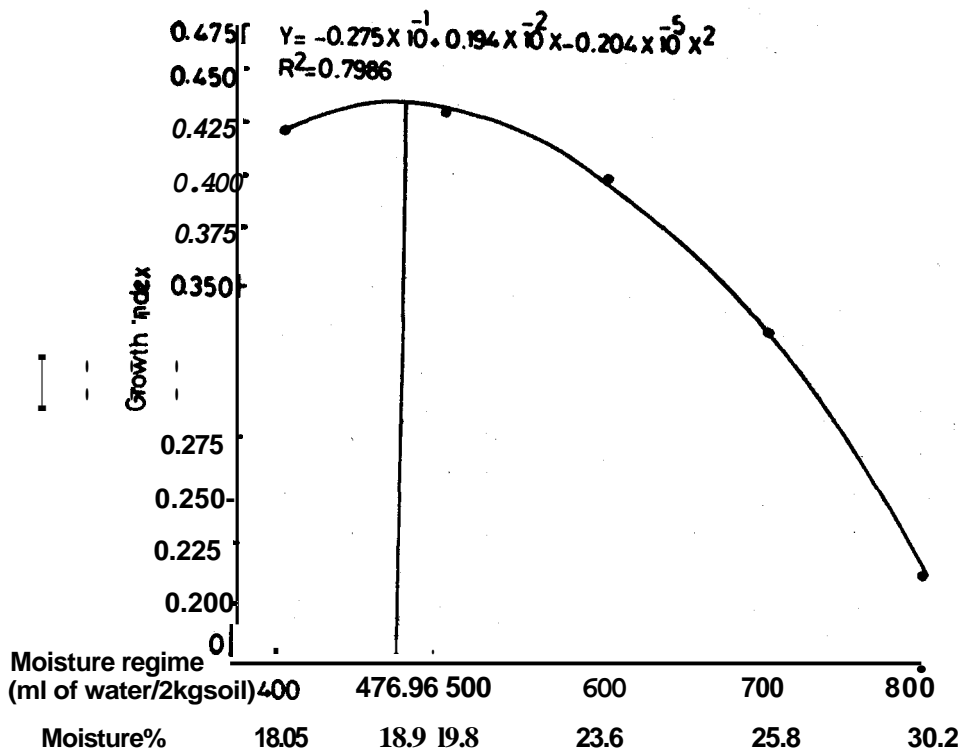


Fig. 13. Regression growth curve of *Rhizoctonia solani* under various soil moisture regimes.

Chemical control

In vitro studies: in poison-food technique Benlate, Emisan-6, Tecto, Terraclor Super-X, and Terrazole were the most effective fungicides in bringing about 100% inhibition of both the isolates of *R. solani* (Table 4). Bavistin and Vitavax were effective only at 1000 and 2000 μg a.i./ml. Hexacap, Daconil, Difolatan and Dithane M-45 inhibited the growth by 60 to 80% (2000 μg a.i./ml), while Fytolan did not show any inhibition even at this concentration. Behaviour of both the isolates was almost identical except in the case of Topsin-M where isolate 783 was completely inhibited at all the concentrations while for isolate 766 it was only at 3500 μg a.i./ml; at other concentrations the percentage inhibition varied from 67 to 79.

Results obtained in soil method were quite different from those of poison-food technique. Except Bavistin 2000 μg a.i./ml and Terraclor Super-X (1170, 2340 and 3510 μg a.i./ml) no fungicide inhibited the growth of both the isolates completely. Hexacap (2000 μg a.i./ml) was found to inhibit the growth of isolate 783 but not of isolate 766.

On comparison of two isolates in both the methods it was observed that Difolatan in poison-food technique and Vitavax in soil method have higher inhibition for isolate 766 than 783.

In vivo studies: In general, fungicides showed significant differences in controlling the disease throughout up to 28th day (Table 5). In the case of affected seedlings the interaction between fungicides (F), isolates (I) and treatments (T) was significant on the 7th day. None of the main effects, except fungicides was significant on the 14th and 28th day. On 21st day, the interaction between F x T was highly significant. For dead seedlings none of the treatments was significantly different on the 7th day. On the 14th and 21st days the pattern of differences was similar giving a significant F x T interaction. By the 28th day only the fungicides differed significantly in their effect. Of the two fungicides, Bavistin gave good protection against web blight depending upon the time of application. For both the isolates of *R. solani*, treatment III using Bavistin gave complete control (Tables 6, 7), as even after 28 days of transplanting of seedlings no disease developed. This was followed by treatment I which initially appeared to be promising for both the isolates but within three weeks percent seedlings got affected with >75% mortality. Treatment II was the least effective as within a week >70% of seedlings were found to be

Table 4. Percent inhibition in diameter growth of *Rhizoctonia solani* in various fungicides

Fungicides ^a	Concentration μg. a. i./ml	% inhibition in diameter growth over control No.			
		Poison-food technique		Soil method	
		Isolate No. 766	Isolate No. 783	Isolate No. 766	Isolate 783
Bavistin	100	79.5	93.8	0	0
	250	100.0	83.9	0	0
	500	94.0	93.3	0	0
	1000	100.0	100.0	60.0	87.0
	2000	100.0	100.0	100.0	100.0
Benlate	100	73.9	100.0	0	0
	250	96.3	100.0	0	0
	500	100.0	100.0	0	0
	1000	100.0	100.0	0	0
	2000	100.0	100.0	0	0
Emisan-6	100	100.0	100.0	0	0
	250	100.0	100.0	0	0
	500	100.0	100.0	0	0
	1000	100.0	100.0	0	0
	2000	100.0	100.0	0	0
Hexacap	100	72.7	71.2	0	0
	250	73.0	69.6	0	0
	500	74.3	73.0	0	0
	1000	78.0	71.6	0	0
	2000	76.2	80.8	0	23.6
Tecto	2500	100.0	100.0	0	0
	5000	100.0	100.0	0	0
	10000	100.0	100.0	0	0
	30000	100.0	100.0	5.5	5.8
Terraclor Super-X	292	100.0	100.0	0	0
	585	100.0	100.0	5.0	39.9
	1170	100.0	100.0	100.0	100.0
	2340	100.0	100.0	100.0	100.0
	3510	100.0	100.0	100.0	100.0
Terrazole	627	100.0	100.0	0	0
	1255	100.0	100.0	0	0
	2510	100.0	100.0	0	0
	3765	100.0	100.0	0	0
Topsin-M	700	67.5	100.0	0	0
	1400	69.1	100.0	0	0
	2100	75.6	100.0	0	0
	2800	78.6	100.0	0	0
	3500	100.0	100.0	0	0
Vitavax	100	75.3	76.4	28.6	7.9
	250	84.4	81.7	26.4	6.3
	500	81.7	80.2	32.2	0
	1000	100.0	100.0	41.9	39.1
	2000	95.0	100.0	50.4	38.5

^aFungicides inhibiting either 100% growth in each method or < 100% inhibition in both the methods are only included.

Table 5. Unweighted analysis of means of four variables influencing the incidence of web blight and mortality of *Albizia* seedlings at different periods of disease development

Variables and their interactions	F value							
	7th day		14th day		21st day		28th day	
	Seedlings affected	Seedlings dead	Seedlings affected	Seedlings dead	Seedlings affected	Seedlings dead	Seedlings affected	Seedlings dead
Fungicides (F)	159.65**	2.22	5.65*	198.47**	674.47**	89.63**	12.05**	12.07**
Isolates (I)	18.47**	0.42	0.30	0.57	4.23*	0.34	1.57	0.78
Treatments (T)	40.14**	2.48	2.03	21.13**	390.16**	43.92**	0.59	0.20
Fungicide concentrations (C)	1.02	0.01	0.02	0.54	0.54	0.34	0.45	0.79
F x I	4.24	1.08	0.03	0.57	4.26*	0.34	1.57	0.79
F x T	34.62**	0.78	2.03	23.69**	390.60**	43.92**	0.59	0.20
F x C	0.20	0.42	0.22	0.54	0.54	0.34	0.45	0.79
I x T	4.15	0.26	0.008	0.79	1.57	0.09	1.01	1.15
I x C	1.57	0.94	0.008	0.28	0.26	0.03	0.59	0.20
T x C	0.19	0.28	0.008	0.25	0.16	0.091	0.008	0.03
F x I x T	7.06**	0.12	0.008	0.15	3.69	0.096	2.10	1.15
F x I x C	0.17	0.22	0.008	0.29	0.61	0.032	0.59	0.20
F x T x C	0.81	0.28	0.008	0.25	0.16	0.092	0.008	0.03
I x T x C	0.55	0.21	0.009	0.66	0.07	0.07	0.036	0.20
F x I x T x C	0.96	0.11	0.009	0.66	0.16	0.07	0.036	0.20
Control x Treatments	12.83**	0.42	0.23	3.62	26.98**	0.68	—	0.10

* P=0.05

** P=0.01

affected and >90% died within two weeks. Terraclor Super-X, which was very effective in *in vitro* studies, did not show any promise in controlling web blight in any of the three treatments. Within two weeks cent percent mortality of seedlings was caused in all the treatments, including treatment III where no infection occurred in Bavistin treated seedlings; in all the treatments the seedling mortality was higher than in control. Surprisingly, no significant difference was observed with the two levels of both the fungicides, either in the appearance of the disease or in preventing the mortality.

Behaviour of the two isolates of *R. solani* in all the three treatments using both the fungicides showed significant differences only up to the 7th day of disease development; isolate differences were shown only by Bavistin on 21st day (Table 6). In general, the isolate 783 appeared to be more aggressive than isolate 766 as the former infected the seedlings first and also caused high mortality of seedlings in shortest period. Furthermore, web blight caused by the latter was easily controllable. This trend was also observed in the control sets of the two isolates as isolate 783 affected all the seedlings within 3 to 4 days whereas isolate 766 took 8 to 10 days.

Discussion

Rhizoctonia solani has gained the reputation of being a widespread, destructive and versatile plant pathogen capable of attacking a very wide range of host plants causing seed decay, damping-off, stem canker, root rots, and foliage diseases (Parmeter 1970; Baker 1970; Florence *et al.*, 1985). The web blight of *A. falcataria* was characterised by rapid vegetative growth over the foliage and production of abundant sclerotia under favourable climatic conditions. Our isolates of *R. solani* are possibly aerial strains (Baker, 1970) affecting only the aerial plant parts as no infection either of roots of the affected seedlings or ungerminated seeds in the infested soil was observed. Agnihothrudu (1962) reported that the infection of *R. solani* on *A. falcataria* in Assam originated on the branch and lower leaflets of the primary rachis were affected first. On the contrary, pathogenicity studies with our isolates clearly indicate that the mycelium originates from the soil and climbs up the stem and spreads to the foliage of seedling. Since the disease spreads through contact, crowding of seedlings always favoured rapid development and spread of web blight. Similar observation has also been made by Singh and Singh (1955). They found that closer the seedlings of *Cyamopsis psoralioides* the greater was the spread of aerial blight caused by *R. solani*. It is quite obvious from the results that the web blight can occur only when the humidity is above 95%, which is prevalent during the monsoon (June-August) in Kesala.

Table 6. Statistical significance of various combinations controlling the incidence of web blight and mortality of *Albizia* seedlings at different periods of disease development

7th day	Mean Percentage of seedlings affected (A) / dead (D) at different days of disease development								
	14th day			21st day					
Fungicide x isolate x treatment (FxIxT)	Fungicide x treatment		D	Fungicide x	A	Fungicide x	A	Fungicide x	D
	A	(F x T)		treatment		isolate		treatment	
				(F x T)		(F x I)		(F x T)	
B* 766** III***	Oa+	B III	0a	B III	Oa	B 766	59.8a	B III	Oa
B 783 III	Oa	B I	2.2a	B II	92.5b	B 783	68.0b	B II	89.3b
B 766 I	4.6a	B I I	80.0b	BI	96.1b	T 766	100.0c	B I	95.0b
B 783 I	10.6a	T I	97.4b	T I	100.0c	T783	100.0c	T I	100.0c
T 766 III	37.1b	T II	97.4b	T II	100.0c			T II	100.0c
B 766 II	74.1b	T III	100.0b	T III	100.0c			T III	100.0c
T 766 I I	76.0b								
T 783 I	95.6b								
B 783 II	92.8c								
T 783 II	96.8c								
T 766 I	98.1c								
T 783 III	100.0c								

***B**, Bavistin; T, Terraclor Super-X

**766, 783, isolates of *R. solani*

***I, II, III, time of application of fungicide * for details see p. 14.

+ Values superscribed by the same letter in one column do not differ significantly

Table 7. Effect of time of application on the efficacy of Bavistin and Terraclor Super-X against web blight of *A. falcataria* seedlings caused by two isolates of *R. solani*

<i>Rhizo- ctonia</i>	Treatment	BAVISTIN				TERRACLOR SUPER-X				CONTROL	
		1000µ g a. i./ml		2000 µ g a. i./ml		1170µ g a. i./ml		2340µ g a. i./ml		Day of 1st disease appearance	Day > 75% mortality
<i>solani</i> isolate No.	(time Of Application)	Day of 1st disease appearance	Day > 75% mortality	Day of 1st disease appearance	Day > 75% mortality	Day of 1st disease appearance	Day > 75% mortality	Day of 1st disease appearance	Day > 75% mortality		
766	I*	7	17	7	17	5	10	4	10	3	12
	II	2	10	2	8	2	10	2	10	2	10
	III	0	0	0	0	4	13	4	13	1	—
783	I	5	17	8	17	6	10	5	10	2	10
	II	2	10	2	12	a	8	2	10	2	8
	II	0	0	0	0	4	18	4	18	1	17

*For explanation of treatments I, II and III see p. 14.

Host - parasite relationship studies: The rapid growth of mycelium of *R. solani* from the infested soil over the aerial parts of *A. falcataria*, especially leaves within 24 h and its further spread to give webby appearance shows the susceptible nature' of this species. This possibly could be due to plant exudates which are known to influence the development of *R. solani* (Kerr and Flentje, 1957; DeSilva and Wood, 1964) and high r. h. The latter gets support from earlier reports that *R. solani* grows optimally at 100% r. h. and its growth is retarded at 99.5% r. h. (Roth and Riker, 1943; Schneider, 1953). Faster growth of mycelium in young seedlings as compared to old seedlings may also be due to exudates as DeSilva and Wood (1964) found that exudates from younger seedlings caused a greater stimulation of growth of *R. solani* than did exudates from older seedlings.

Pattern of growth of hyphae is known to be greatly influenced by the nature of the surface on which the fungus grows. In *A. falcataria* no particular pattern of hyphal growth was observed in relation to cell walls. However, the growth of hyphae which gave rise to cushions was distinctly different from other hyphae. This conform to earlier report by Flentje *et al.* (1963) who found marked differences between hyphae, which give rise to branches forming cushions and normal vegetative hyphae. SEM and light microscopic studies clearly indicated that penetration was direct through epidermis and no stomatal penetration was observed as reported for *R. solani* by some workers (Townsend, 1934; Ullstrup, 1936). Penetration of the intact cuticle and epidermis by *R. solani* has been reported (Dodman and Flentje, 1970) but in a very few cases have these studies provided detailed observations on the actual means of entry. In *A. fulcataria*, penetration by mycelial cushions was common, though lobate appressoria were also observed.

Effect of soil moisture on the linear growth of *R. solani*: It is evident from the results that the saprophytic linear growth of *R. solani* in sterile soil is greatly affected by the moisture regime. The growth is best at low moisture regimes and as soil moisture increases it declines. Similar observations that *Rhizoctonia* is favoured by intermediate moistures and often operates in relatively dry soil and that excess soil moisture inhibits its growth have also been made by various authors (Blair, 1942; Roth and Riker, 1942; Rushdi and Jeffers, 1956; Radha and Menon, 1957; Das and Western, 1959; Papavizas and Davey, 1961). Slow growth of *R. solani* at high moisture levels was possibly due to lack of aeration and accumulation of CO₂, as has been reported by Durbin (1959), and Papavizas and Davey (1962). On the tenth day linear growth of mycelium of *R. solani* in sterile soil with 18% moisture was ca. 5 to 6 cm, which is comparable to earlier findings of Sanford (1938), and Rushdi and Jeffers (1956) with 10 to

19.5% soil moisture. However, Radha and Menon (1957) recorded growth of 21.3 cm at 50% moisture after 21 days. This discrepancy in behaviour of *R. solani* could be due to differences in isolate and growth techniques used.

Availability of rapidly assimilating nutrients in the agar discs in the initial stages of growth may possibly explain the intensive saprophytic activity of *R. solani* during the initial six days. As quickly available substrate decreased, the saprophytic activity also declined. Since the sterile soil was used decline in growth is not related to antagonistic activity of soil microorganisms.

Influence of *R. solani* isolates, inoculum level and age of seedlings on web blight: Seedlings in none of the stages of disease development, except in stage I, achieved cent percent infection. This appears to be due to longer time taken by the mycelium to climb up the foliage and infect plant parts away from the ground. Since stages III and IV, which represented spread of mycelium to second leaf and from one seedling to another respectively, are not found significantly different, it possibly means that for web blight of *A. falacataria* only the initial stages i. e., I and II and the last stage V, when the seedlings are killed, are sufficient to reflect significant differences between various disease parameters. This appears to be also logical because if there are differences due to interactions between isolate (I), inoculum level (L) and age of seedlings (A) these, especially that of I x L would be evident clearly in the initial stages due to inherent characters. During the period of secondary spread of the disease due to overlap these interactions are unlikely to be significant as confirmed in statistical analysis of results, where I x L x A interaction is significant only for stages I and II and not for V. In stage V, significant difference in I x A interaction indicates that isolate and age of seedlings affect the seedling mortality more than the inoculum concentration. However, in most of the combinations the disease incidence was more in low inoculum level i. e., 10g/2 kg of soil than in high (40g). Sanford (1941) and Das and Western (1959) have also observed reduction in pathogenicity/ virulence of *R. solani* in sterile soil containing high concentration of inoculum. Incidentally, in the present experiment also sterile soil was used. However, no explanation is available for this behaviour of *R. solani* in sterile soil. It is possible that the differences between the two concentrations were not large enough to be significantly different from each other. As regards the age, younger seedlings (60-day-old) were more susceptible than 75-day-old as has also been reported by Bateman and Lumsdan (1965) and, Mildenhall and Williams (1973). This also confirms field observations that seedlings develop resistance to web blight on maturity. Higher susceptibility of younger seedlings could be due to exudates, which possibly favoured growth and infection

by *R. solani* (DeSilva and Wood, 1964), or else, the soft nature of tissues of young seedlings would have favoured the infection and spread of web blight.

Among the two isolates, 783 behaves more aggressively than isolate 766, which also showed significant differences in symptom development. Earlier, Shatla and Sinclair (1963) have also reported similar results where under greenhouse condition nine isolates of *R. solani* varied in their pathogenicity from slightly to highly pathogenic.

Chemical control: Of the 13 fungicides screened against *R. solani*, only Bavistin (2000 μ g a. i./ml) and Terraclor Super-X (1170,2340 and 3510 μ g a. i./ml) inhibited the growth completely of both the isolates in soil method. This is in contrast to poison-food technique where cent percent inhibition of growth was caused by Benlate, Emisan-6, Terraclor Super-X, Bavistin and Vitavax; the latter two were effective only at 1000 and 2000 μ g a. i./ml). This clearly indicates that for sclerotial fungi like *R. solani* soil method is more reliable than poison-food technique. This may be one of the reasons for obtaining erroneous results in *in vivo* studies using effective fungicides screened through poison-food technique (Martin *et al.*, 1984a, b). Terraclor Super-X, a formulation of PCNB, was found effective against both the isolates of *R. solani*. With the development of organic fungicides, pentachloronitrobenzene (PCNB, quintozone, Terraclor) became very popular and it has been used widely to control *Rhizoctonia* diseases for the last three decades (Georgopoulos and Wilhelm, 1962; Livingston *et al.*, 1964; Wright, 1968; Souza Filho, 1979; Galindo *et al.*, 1982; Bains and Jhooty, 1983; Schneider and Potter, 1983; Gurkin and Jenkins, 1985). Other effective fungicide was Bavistin (carbendazim), which has been also reported earlier to be promising against *Rhizoctonia* diseases on various crops (Shehata *et al.*, 1983; Grover and Kataria, 1985). However, results with Vitavax, which is generally known to provide good protection against *R. solani*, (Borum and Sinclair, 1968; Allam *et al.*, 1969; Martin *et al.*, 1984b), were not encouraging for web blight pathogen as also has been observed by Bains and Jhooty (1983) working with different isolates of *R. solani*. Similarly, certain other fungicides, namely Difolatan (Oyeken, 1979), Daconil 2787 (Seoud *et al.*, 1982; Schneider and Potter, 1983; Martin *et al.*, 1984b), Topsin-M (Shehata *et al.*, 1982; Chase, 1982), which have been reported to be effective against *R. solani* were not promising in inhibiting the growth of web blight pathogen. These findings are in agreement with earlier observations that in spite of the fact that quite a large number of fungicides have been tried and found useful against *R. solani* there appears to be lack of agreement between different reports on the efficacy of a particular fungicide (Grover and Kataria, 1985),

In both the methods of screening, response of two isolates of *R. solani* i. e., 766 and 783 to certain fungicides such as Hexacap (2000 μ g a. i. / ml) in soil method and Topsin-M in poison-food technique was significantly different. This type of differential behaviour of isolates of *R. solani* has also been reported earlier by various workers (Thomas, 1962; Bains and Jhooty, 1983; Martin *et al.*, 1984 a). Sinclair (1960) reported significant differences in the degree of sensitivity among five isolates and suggested that this may account for the apparent lack of uniformity of disease control in the field.

In vivo studies reveal that Bavistin is the only effective fungicide for controlling web blight caused by both the isolates of *R. solani*, provided it was applied before transplanting the seedlings in the infested soil; it was not effective when applied at the time of transplanting or after the appearance of the disease. Bavistin, (carbendazim) applied as soil drench or foliar spray has earlier been reported to control sheath blight of rice (Dev, 1980; Dev and Satyarajan, 1980; Shehata *et al.*, 1982). Efficacy of carbendazim is further established by the fact that it is also known to persist in soil for a significantly longer time and at higher concentrations in the leaves of pepper grown in treated soil (Yarden *et al.*, 1985). On the contrary, Terraclor Super-X, which was equally effective in *in vitro* studies failed to provide any protection against web blight in any of the three treatments. This type of anomaly in *R. solani* where *in vitro* tests of different isolates are not correlated with *in vivo* tests, is not uncommon and for which various reasons have been ascribed (Grover and Kataria, 1985). Wright (1968) found that Terraclor Super-X suppressed growth of *R. solani* and thereby reduced incidence of potato stem canker in clay soils. However, even very high rates of this fungicide did not control the disease in muck soils. Shatla and Sinclair (1962) reported a correlation between pathogenicity and sensitivity of the isolates of *R. solani* to quintozone while Maier (1962) found that the differential *in vitro* sensitivity of 12 isolates to quintozone + thiram showed no correlation in the field tests. A lack of correlation between *in vitro* inhibition of growth of different isolates of *R. solani* and disease control with fungicide treatments was also found by Jhooty and Bains (1973). Similarly, Kataria and Grover (1978) compared 41 fungicides *in vitro* against an isolate of *R. solani* and showed that these results could not be correlated in every case with the control of the pathogen on the host plant.

Reasons for ineffectiveness of Terraclor Super-X in *in vivo* experiments are not clearly understood but they could be composition of medium on which inoculum was raised, and temperature and pH differences in *in vitro* and *in vivo* studies. As regards the inoculum medium is concerned, disease control by PCNB is known to be most affected by inocula grown in different substrates while Bavistin (carbendazim)

the least. Quintozene (PCNB) is also reported to be very sensitive to temperature and pH. Kataria and Grover (1976) found that the optimum temperature for *in vitro* inhibition of growth of *R. solani* by quintozene was 25°C at pH 5.6 whereas in pot trials it was 30°C at pH 5.4. In our studies, *in vitro* screening was done at 25°C but during *in vivo* the temperature ranged between 30 and 35°C and the soil pH between 5.8 and 6.0.

The results clearly suggest that for affording effective protection against web blight of seedlings of *A. falcata* the soil of the nursery beds should be treated with Bavistin before raising the seedlings. And also, it will be advisable to prepare beds for raising *Albizia* seedlings at different sites every year because Bavistin is known to degrade more rapidly in soils with previous history of Bavistin treatment than without (Yarden *et al.*, 1985).

WILT

Occurrence

The disease was recorded in 3-month-old seedlings at Kollathirumedu (Vazhachal For. Div). during April 1980. In seedbeds the disease occurred in patches affecting about 50% of the seedlings.

Symptoms

The lower leaves initially turned yellow and got defoliated. Gradually the yellowing proceeded towards the growing shoot. The affected seedlings, appeared to be stunted with only 1-2 leaves remaining near the apex, died within a month. The roots of such seedlings showed prominent discoloration.

Etiology

Fusarium solani (Mart) Sacc.

Control measures

Agallol (MEMC) (2500 µg a. i./ ml), Dithane M-45 (3000 µg a. i. / ml), Difolatan (3000 µg a. i./ml), Hexathir (Thiride) (3000 µg a. i./ml) and Bavistin (2000 µg a. i./ml) were applied separately in half part of three seedbeds. The remaining half was untreated as control. Observations recorded after two weeks indicated that Dithane M-45 and Bavistin were the most effective fungicides. In seedbeds treated with Bavistin the disease was completely controlled. However, after two weeks fresh seedlings were found affected in Dithane M-45 treated beds. Second treatment of Dithane M-45 (1000 µg a. i./ml) applied immediately controlled the disease.

Diseases in Plantations

Three diseases were observed in plantations. Amongst them Botryodiplodia die-back was the most common disease followed by Phomopsis shoot die-back, A bacterial partial wilt was recorded only from one plantation.

BOTRYODIPLODIA DIE-BACK

Incidence and severity

This was the most serious disease of *A. falcataria* prevalent in plantations throughout the State. Large-scale mortality of trees due to this disease was recorded usually in patches in plantations at Nangachee-1974, Kattilappara-1980 (Thenmala For. Div.) and Keezhayam - 1979 (Punalur For. Div.). The year after the name of the locality refers to year of planting.

Though the die-back occurred in most of the observation plots at Kattilappara- 1980,-1981, Keezhayam- 1979, Arippa-1979, high incidence of the disease was localised in some patches of these plantations where mortality of trees was observed. No die-back was recorded in plantations at Vamanapuram-1980 (Table 8). During 1983 the disease incidence in plantations at Kattilappara-1980, Arippa-1979 and Keezhayam-1979 was about 50% or a little over, which gradually decreased between 13 and 25% over the next three years. Asimiiar trend was observed with respect to disease severity and death of trees. Average disease severity rating (DSR) in these plantations was initially low, which subsequently declined further to very low by 1985. Percentage of trees killed also declined considerably. Exceptionally, the incidence in Kattilappara- 1981 plantation remained almost static at around 24% except during December 1984 when it declined to 17%. In this plantation, though DSR remained low, DSI showed a slight increase from 0.26 to 0.4 with no mortality recorded during the observation period.

Progress and spread of die-back

Progress of die-back monitored in the HD plot at Kattilappara is given in Table 9. Distribution of trees with different disease severity and progress in their severity over time showed a pattern of spread of disease from tree to tree, particularly in trees surrounding a few severely affected or dead trees. Though the disease severity remained constant at moderately severe (MS) level, the incidence showed a decline from 94.32% in June 1983 to 69.83% in May 1985. However, the mortality of the affected trees increased from 8.8 to 30.3% during the period. Generally, high incidence of die-back occurred during the dry-warm period. But during or just after the monsoon the incidence apparently

Table 8. Incidence and severity of Botryodiplodia die-back in representative plantations of *Albizia falcataria* in Kerala during 1983—1985

Locality and year of planting (Total area in ha)	Total number of trees in three observation plots	Disease parameters	Month of observation					
			June '83	April '84	July '84	Dec. '84	May '85	
Arippa ^a - 1979 (41.8)	675	360	% incidence	59.7	-d	25.7	21.4	25.7
			% of diseased trees killed	1.9	—	0.97	0	0
			Disease severity DSI ^b	1.3	—	0.39	0.33	0.46
			DSR ^c	L	—	L	L	L
Kattilappara-1980 (10.5)	675	360	% incidence	66.4	41.5	18.5	20.6	13.3
			% of diseased trees killed	0.4	0.4	0.4	0	0
			Disease severity DSI	1.4	0.67	0.28	0.32	0.23
			DSR	L	L	L	L	L
Kattiiappara-1981 (10.0)	675	360	% incidence	23.8	23.8	23.5	16.6	23.9
			% of diseased trees killed	0	0	0	0	0
			Disease severity DSI	0.26	0.36	0.3	0.28	0.4
			DSR	L	L	L	L	L
Keezhayam-1979 (27.0)	900	900	% incidence	49.2	39.2	31.5	24.7	25.8
			% of diseased trees killed	12.9	2.1	1.5	0.3	0.5
			Disease severity DSI	1.3	0.72	0.54	0.38	0.47
			DSR	L	L	L	L	L
Vamanapuram-1980 (27.9)	675	360	% incidence	0	0	0	0	0
			% of diseased trees killed	0	0	0	0	0
			Disease severity DSI	0	0	0	0	0
			DSR	Nil	Nil	Nil	Nil	Nil

^a First observation recorded in September 1983

^b Disease severity index

^c Disease severity rating

^d Observation not recorded

declined as some of the trees with low severity rating recouped partially with the production of new shoots. About 40.0% of the trees, which were diseased at the time of first observation, recouped and became healthy by May 1985; this included even some trees with DSR of 4.

Table 9. Incidence and severity of *Botryodiplodia* die-back in a HD plot of *Albizia falcataria* at Kattilappara, Kerala during 1983-1985

Disease parameter	Date of observation					
	16 June '83	17 Sept. '83	22 Feb. '84	18 July '84	13 Dec. '84	7 May '85
% incidence	94.3	89.4	91.2	86.2	81.7	69.8
% of diseased trees killed	8.3	10.4	13.5	18.7	21.9	30.3
Disease severity DSI ^a	2.4	2.5	2.5	2.3	2.5	2.3
DSR ^b	MSc	MS	MS	MS	MS	MS

a Disease severity index

b Disease severity rating

c Moderately severe

Symptoms

The initial symptom of die-back was appearance of a stem canker in the form of a depressed greyish-black area, generally near the ground level, during the dry period (Fig. 14). This was followed by yellowing of leaflets, which gradually defoliated prematurely (Fig. 15). Slowly shoots in the upper crown of the tree showed symptoms of die-back. Under favourable conditions the canker spread lengthwise to several centimeters as the infection progressed further (Fig. 16). The bark over the canker splitted longitudinally and got separated. The wood of the affected trees showed greyish-black discolouration in streaks (Fig. 17) running vertically due to profuse mycelial growth in ray cells. As the canker advanced further, more branches died, including the main terminal shoot and the tree appeared to be almost dead (Fig. 18). However, during the following monsoon numerous epicormic shoots developed from the living part of the stem (Fig. 19) and callus growth from the margins over grew the canker covering it partly or completely depending upon the extent of canker, girth of trees and favourable condition i. e., prolonged wet period. Some of the shoots grew rapidly giving somewhat healthy appearance to trees. However, during the next dry-warm period the cankers progressed further resulting in the death of more shoots. By this time the canker usually had also spread downwards into the root system (Fig. 20). This process of



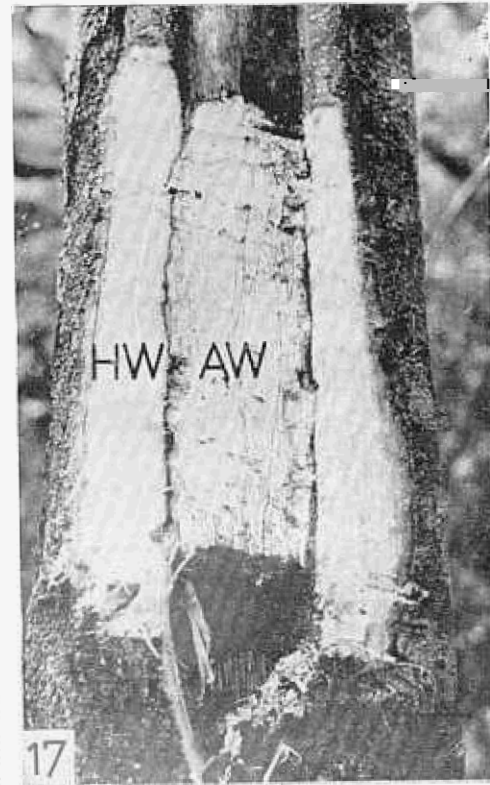
14



15



16

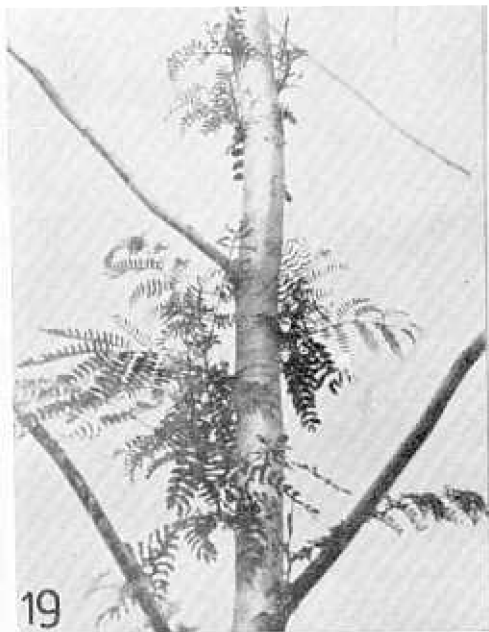


17

Figs. 14-17. *Botryodiplodia* die-back of *Albizia falcataria*. 14, A young stem canker (C) and a portion of exposed decayed root (marked with an arrow). 15, A shoot in the upper part of the crown showing premature defoliation. 16, Several meters long stem (canker initiated from the base of the stem). 17, A part of the canker showing healthy (HW) and adjoining affected wood (AW) exposed to show the discolouration in the affected part due to extensive mycelial growth.



18



19



20



21

Figs. 18-21. Botryodiplodia die-back of *Albizia falcataria*. 18, Four-year-old trees severely affected by the die-back. 19, Development of epicormic shoots on a severely affected tree during the wet season. 20, A stem canker extending up to the root system. 21, A stem canker initiated near the ground level being callused over during the wet season.

development of epicormic shoots and callusing over of canker (Figs. 20, 21) during the wet period and further spread of canker and killing of shoots during dry-warm period continued for 2-3 years till the progressively spreading canker girdled the stem completely and also affected most of the root system, which consequently led to death of trees.

In some cases the initiation of infection was from the roots rather than the stem. This was observed in trees with partially exposed roots (Fig. 14). The infection from the root canker spread to feeder roots and other large roots leading to stem collar. The infection progressed further upwards into stem and gave rise to stem canker.

Pathogen

In a plantation of *A.falcataria* at Nangachee, *Hypoxylon* was suspected to be the pathogen of die-back as profuse growth of fructifications of *Hypoxylon rubiginosum* (Pers. ex Fr.) Fr. var. *tropicum* Miller (IMI 254086) and *H. bovei* Speg. (IMI 254085) were observed on the stems of partially dead and completely dead trees. However, in most of the other plantations association of *Hypoxylon* spp. with trees affected with die-back was not found to be consistent, thus ruling out the possibility of being the pathogens of the disease. Furthermore, saprophytic growth of *Hypoxylon* on dead fallen branches and twigs during the wet period and negative pathogenicity tests with *Hypoxylon* spp. also strengthened this view. Later, a closer examination of the canker revealed profuse growth of pycnidia of *Botryodiplodia theobromae* immersed in the bark, at Nangachee and other places. During the monsoon pycnidia are hidden in the bark of the canker and appear as small protuberances over the surface but during the dry period they erupt producing black powdery mass of conidia. Consistent isolation of only *B.theobromae* (IMI 280241) from the diseased specimens further gave positive indication of it being the die-back pathogen.

Etiology

Botryodiplodia theobromae Pat.

Pathogenicity

In pathogenicity tests, conducted during the wet period, none of the inoculation methods was successful as the wounds callused over without producing any discolouration in wood or canker. However, tests carried out during the dry period with relatively high temperature gave positive results.

In trees inoculated with a 'V'shaped cut, the disease appeared in 2 to 3 month's time. At the infected site the bark became depressed and turned greyish-black, developing into a canker. After 6 to 7 months the infection had spread 5 to 8 cm longitudinally and 2 cm vertically in the stem. No infection occurred in trees which were inoculated either with bark injury alone or with no injury; in the former case the wound callused over in the following monsoon. Reisolation of *B. theobromae* from the infected tissues, several centimeters away from the inoculated site, confirmed the pathogenic nature of the isolate.

Infection through fire injury: Of the five trees only three developed infection within two months of inoculation; *B. theobromae* was reisolated from the infected discoloured wood. The other two trees died within one month of inoculation, possibly due to excessive fire injury as no infection could be noticed in the stem.

Discussion

Botryodiplodia theobromae is a ubiquitous, facultative, wound pathogen widely distributed in tropics and subtropics. It is reported to cause several types of diseases such as dampingoff, seedling blight, die-back, stem canker, stump rot, root rot, leaf spot and pre- and post-harvest fruit rots (Punithalingam, 1980) thus affecting almost all the parts of plant. *Botryodiplodia* die-back of *A. falcataria* recorded from India (Kerala) (Venkataram, 1964) and Indonesia (D'Angremond, 1948) and other two diseases, viz. *Botryodiplodia* root infection (Wynad, Kerala, India) (Venkataram, 1960) and *Botryodiplodia* stem canker (Sri Lanka) (Browne, 1968) known earlier in the literature are possibly similar to die-back reported here. It appears that while reporting the latter two diseases emphasis had been placed on the part of the tree infected rather than the ultimate symptoms produced. Production of stem canker is the first stage of die-back, which may or may not be accompanied with root infection. This is followed by yellowing of leaflets, defoliation and die-back of smaller shoots in the crown. Field observations indicate that the death of trees due to die-back depended upon age of the tree at which infection occurred, its girth and season, besides the severity of infection including extent of canker. If the tree gets infected at the age of 2-3 yr, as possibly happened in the HD plot at Kattilappara, the survival of trees by recouping during the monsoon depended mainly on the extent of stem canker and the girth of trees; trees with smaller girth (20-25 cm) got girdled easily by the rapidly spreading canker and succumb. In trees of bigger girth (45-60 cm) rapid callusing over the canker helped in resisting the infection. On the contrary, adverse environmental conditions contribute to rapid development of canker and thus spread of infection.

The time of infection also affects greatly the severity of the disease and survival of trees. Infections occurring in April/May are usually callused over during the following monsoon (June- September) healing the cankers completely, while those during dry-warm months (January/February) resulted in large cankers which usually did not callus over completely. Pathogenicity trials conducted during the dry and wet periods also confirm these observations. These findings are also in conformity with the earlier observations on the effect of month of inoculation and subsequent development of stem canker diseases caused by *Botryodiplodia*. Riffle(1978) reported that though all trees of *Ulmus pumila* L. inoculated with *B. hypodermia* (Sacc.) Petr. & Syd. during March-September cankered, inoculation during July-September gave highest percentage of successful infections; during March-May or November-February most of the cankers callused over. He also suggested that warm weather with deficient rainfall increased the susceptibility of *U. pumila* to *B. hypodermia*. Possibly these factors contributed to moisture stress in the bark and, hence, conditions were more favourable for the growth of *B. hypodermia*. Successful infections during dry-warm period and through the fire injury are also likely to be due to moisture stress. Also, in the latter case the tissues dead due to fire will facilitate easy colonization by *B. theobromae*. Role of moisture stress in pathogenesis has also been found true for some other canker pathogens (Bier, 1964), such as *Cystospora* on *Populus* (Bloomberg, 1962; Bloomberg and Farris, 1963), *Hypoxylon pruinaum*. (Klotsche) Cke. on *Populus tremuloides* Michx. (Bagga and Smalley, 1969) and *Nectria cinnabarina* Tode ex Fr. on elm (Munch, 1909). Rapid development of cankers during dry-warm weather is also supported by reports that *Botryodiplodia* spp. are adapted for optimum growth at relatively high temperature (Riffle, 1978; Sharma *et al.*, 1984).

Of the five plantations surveyed, die-back was recorded only in four, where the severity remained low throughout the period of study. However, the percent incidence declined considerably, except in a younger plantation at Kattilappara- 1981, where it remained almost static. The reason for the decline in incidence is possibly recouping of the affected trees; in some instances trees even with DSI of 4 recouped. Similar trends were also observed in the HD plot, where the severity of die-back was recorded to be moderately severe throughout the study period, but the mortality of the affected trees increased. A number of factors are likely to contribute to the initial high incidence and its subsequent decline accompanied with increase in number of dead trees. First of all, since this part of the plantation was easily accessible, the frequent grazing of cattle after the extraction of taungya crop in 1982 caused extensive bark injuries. This coupled with high inoculum on leftover tapioca stem, which formed a good substrate for *B. theobromae* during the dry-warm period and

CLIMATIC DATA AT PUNALUR

—●— Monthly mean maximum temperature (°C)
 - - -● - - - minimum " " " "
 □ total rainfall

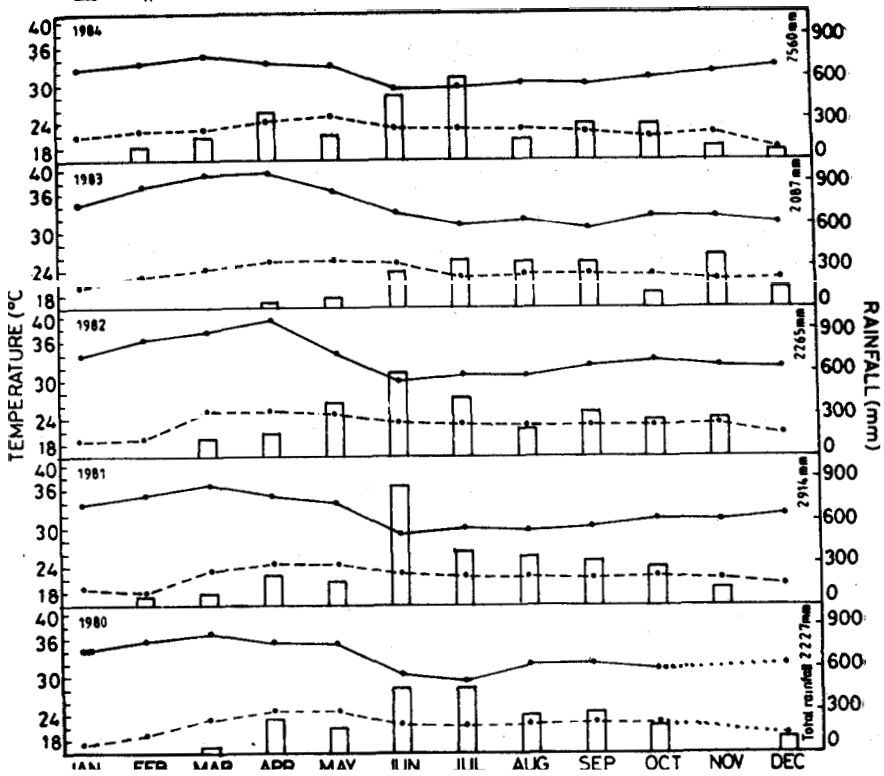


Fig. 22. Rainfall and temperature data for the years 1980-1984 recorded at Punalur.

severe drought during 1982 and 1983 (Fig. 22) led to severe infection in this part of the plantation. It may be worth noting here that *B. theobromae* is known to cause a severe stem rot of cassava (tapioca), where the infection is either through injury or natural cracks present in the bark (Hopkins, 1950). At the time of the first observation itself 18 trees already had died in the HD plot and 19 had basal and above ground cankers, some several meters long. These were the trees which could not recoup in subsequent monsoons with normal or above normal rainfall and died slowly and gradually. In a few instances coppice shoots, developed from the stem collar region of the apparently dead trees, helped the tree to survive. Hence, the incidence and severity of die-back are greatly influenced by the season. Similar observations are that of Shaw (1921) who found that the incidence and severity of black band disease of jute in India caused by *B. rheobromae* vary from season to season, high relative humidity and high temperature being the major

A pattern of declining incidence of die-back with low severity in all the four affected plantations, where there was no history of fire, shows that most of the trees will get recouped within the next few years unless the situation is aggravated due to drought. High disease incidence in plantations at Nangachee-1974 and Vazhachal-1974 was possibly the result of frequent fire in the area. Absence of die-back in Vamanapuram-1980 plantation may be due to absence of cattle grazing, fire and tapioca cultivation. This provides sufficient evidence that *Albizia* plantations subjected to fire and debarking by domestic and wild animals, which weaken the trees, and those with a taungya crop of tapioca are more prone to *Botryodiplodia* die-back than without. For *Botryodiplodia* die-back of *Araucaria cunninghamii* D. Don. also, Griffiths (1966) found a close relationship of trees weakened either by environmental changes or an injury by a primary invader to development of the disease. As it is not easy to limit the sources of inoculum surviving on decaying litter in tropics (Weststeijn, 1966) and the high inoculum pressure in *Albizia* plantations because of tapioca cultivation, any bark injury caused by fire or animals or even agricultural implements while doing soil work for the tapioca crop will form potential site for the manifestation of the disease. Since *B. theobromae* is primarily a wound pathogen *Botryodiplodia* die-back of *A. falcataria* can be avoided to a considerable extent by protecting the plantations from biotic factors and fire and also removal of tapioca stem from the plantation after the harvest of tubers. If possible, instead of tapioca some other taungya crop may be grown in *Albizia* plantations.

PHOMOPSIS SHOOT DIE-BACK

Occurrence

The disease was recorded in plantations at Vazhachal-1980 and Kattilappara-1981 where plants were affected by fire and or *Botryodiplodia* die-back.

Symptoms

The first symptom of the disease was yellowing of leaves which led to premature defoliation. This was followed by death of the terminal shoot. At this stage prominent cankers were observed on the affected branches. Under humid conditions often fructification of the pathogen developed over the cankers. The disease usually caused death of young branches and twigs in upper half of the tree crown.

Etiology

Phomopsis mendax (Sacc.) Trav. (IMI 87, 270188, 290729)

Pathogenicity

In pathogenicity trials conducted on 2-year-old trees at Kattilappare infection developed only in the wounded shoots. This indicated the weak pathogenic nature of *P. mendax*.

Discussion

Die-back of *A. julibrissin*, *A. Iebbek*, *A. versicolor*, *A. odorotissima* and *A. petersiana* caused by *P. mendax* has been reported by Gibson (1975) from the herbarium specimens from Pakistan, India, Tanzania, Zambia and Malta deposited in the herbarium of CAB International Mycological Institute, U. K. No other details on symptomatology and incidence of the disease are available. This is the first record of *P. mendax* causing shoot-die-back of *A. falcataria* in India.

The shoot die-back does not appear to be a serious disease as it usually occurred in trees weakened either by fire or Botryodiplodia die-back. If protection is afforded to plantations against these two factors the disease incidence can be minimised considerably.

PARTIAL BACTERIAL WILT

Occurrence

The disease was only recorded in a 2-year-old plantation at Thundathil (Kodanad For. Div.). The incidence of the disease, which occurred in patches, was estimated to be ca. 2%.

Symptoms

In the affected plants,) initially leaves of lower branches on one side turned yellow and finally wilted and dried up. Such plants had decayed feeder as well as primary roots which became greyish black. As the root infection proceeded further towards the stem, more side branches were killed.. Finally, when the infection had already reached above ground, appearing as greyish black sunken canker on one side of the stem the terminal shoot got killed. From the healthy side of the stem numerous epicormic branches developed, which did not survive for long as the stem was completely girdled by the spreading canker down below at the base.

When a small piece of stem/root taken from the affected trees was dipped in water streaks of bacterial ooze from the vascular elements were noticed. This confirmed the association of a bacterium with the disease.

Etiology

Pseudomonas sp. (IMI 139597), resembling *P. solanacearum* (E. F. Smith) E. F. Smith.

Bacterium gram negative, rod shaped, motile. Colonies on nutrient agar whitish or opalescent turning darker, small, irregular, smooth, wet and shiny.

Discussion

Extensive cattle damage was noticed in the plantation where bacterial wilt was recorded. It is not known whether bark injuries helped in the manifestation of the disease. As the disease was observed only in one plantation and the incidence was low, it does not appear to be of serious concern. There are only a few reports of bacterial wilt of trees (Browne, 1968). Partial bacterial wilt of *A. falcataria* is a new record.

RECOMMENDATIONS

The two serious diseases of *Albizia falcataria* viz. web blight caused by *Rhizoctonia solani* in nursery and Botryodiplodia die-back caused by *B. theobromae* in plantations can be managed effectively to minimize the losses. Web blight can be controlled by applying a prophylactic treatment of Bavistin at 500 µg a. i. / ml, a week before sowing the seeds in the nursery beds. After the appearance of the disease, however, at least two applications of Bavistin (1000 µg a. i./ml, at weekly interval will be necessary. As the disease manifests and spreads when the seedling density and relative humidity are high it is recommended to avoid crowding of seedlings and over watering the seedbeds.

Affording protection to *Albizia* plantations from fire and biotic factors could prevent bark injuries, which form potential site for infection by the wound pathogen *B. theobromae*. Removal of tapioca stem from the plantations after the harvest of tubers could reduce the inoculum pressure considerably, thus minimising the disease hazards. These two measures as a part of the management system will certainly contribute towards avoiding the development of die-back disease in *Albizia* plantations.

REFERENCES

- Anonymous, 1937. Verslag van der Directeur van het Algerneen Proefstation der A.V.R.O S. over het tyjdoak I Juli 1935-30 June 1936 on het tydvak I Juli 1936-31 December 1936. Meded. alg. Proefst. Avros. Alg. Ser 58: 48 p.
- Anonymous, 1950. Rapport annuelle pour l' exercice 1949. Publ. *Inst. nat. Etude agron.* Cong. Belge 1950 (hors series), 306 p.
- Anonymous, 1979. *Tropical Legumes: Resources for the Future*. National Academy of Sciences, Washington, D.C. 331 p.
- Agnihotrudu, V. 1962. Outbreaks and new records. Two species of *Pellicularia* parasitic on *Albiziafalcata* in Assam. FAO Pl. Prot. Bull. 10: 143-145.
- Allam, A.I., J.B. Sinclair and P.E. Schilling 1969. Laboratory and greenhouse evaluations of four systemic fungicides. *Phytopathology* 59: 1659-1662.
- Bagga, D.K. and E.B. Smalley 1969. Factors affecting canker development on *Populus tremuloides* artificially inoculated with *Hypoxyylon pruinaum*. *Can. J. Bot.* 47: 907-914.
- Bains, S.S and J.S. Jhooty 1983. Sensitivity of fungitoxicants, cultural behaviour and pathogenicity of *Rhizoctoniasolani* isolates naturally occurring in Punjab. *Indian J. of Ecology* 10: 274-278.
- Baker, K.F. 1970. Types of *Rhizoctonia* diseases and their occurrence. In: J.R. Parmeter Jr. (ed.). *Rhizoctonia solani: Biology and Pathology*. University of California Press, Berkeley, USA pp. 125-148.
- Bakshi, B.K., M A R. Reddy, Sujan Singh and P.C. Pandey 1970. Disease situation in Indian forests. 1. Stem diseases due to *Corticium salmonicolor* and *Monochaetiaunicornis*. *Indian Forester* 96: 826-829.
- 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. 117 p
- Bateman, D.F., and R.D. Lumsdan 1965. Relation of calcium content and nature of the pectic substances in bean hypocotyles of different ages to susceptibility to an isolate of *Rhizoctonia solani*. *Phytopathology* 55: 734-738.
- Bates, G.R. 1961. Branch of Botany, Plant Pathology and Seed Testing. Rep. agric. Rhod Nyasald 1959-1960: 50-57.

- Bernard, C. 1926. Verslage over het Algemeen Proefstation voor Thee over het jaar Meded. Proefst, Voor Thee 45, 26 p.
- Bertus, A. L. 1961. Fungi recorded on the leaves, stems, flowers and fruits of forest trees in Ceylon. Ceylon Forester (NS) 5: 101-113.
- Bier, J. E. 1964. The relation of some bark factors to canker susceptibility. Phytopathology 54: 250-253.
- Blair, I.D. 1942. Behaviour of the fungus *Rhizoctonia solani* Kuhn. in the soil. Ann. 'Appl. Biol. 30: 118-127.
- Bloomberg, W.J. 1962. Cytospora canker of poplars: the moisture relations and anatomy of the host. Can. J. Bot. 40: 1281-1292.
- Bloomberg, W.J. and S.H. Farris. 1963. Cytospora canker of poplars: bark wounding in relation to canker development. Can. J. Bot. 41: 303-310.
- Borum, D.C. and J. B. Sinclair 1968. Evidence for systemic protection against *Rhizoctonia solani* with Vitavax in cotton seedlings. Phytopathology 58: 976-980.
- Browne, F.G. 1968. *Pests and Diseases of Forest Plantation Trees*. Clarendon Press, Oxford. 1330 p.
- Calinski, T. and L.C.A. Corsten 1985. Clustering means in Anova by simultaneous testing. Biometrics 4: 39-48.
- Chase, A. R. 1982. *Rhizoctonia* aerial blight's effects on leather leaf ferns and pittosporums. American Nurseryman 156: 75-76.
- Cordon, M. E. and R. E. Young 1962. Evaluation of eradicant soil fungicides in the laboratory. Phytopathology 52 : 503-509.
- D'Angremond, A. 1948. Verslag van der Directeur van het Algemeen Proefstation der A. V. R. O. S. ove het tydvlak 1 Januari 1940-31 December 1940. Meded alg. Proefst, Avros. Alg. Ser. 69 :59 p.
- Das, A. C. and J. H. Western 1959. The effect of inorganic measures, moisture and inoculum on the incidence of root disease caused by *Rhizoctonia solani* Kuhn. in cultivated soil. Ann. Appl. Biol 47 :37-48.
- DeSilva, K. L. and R. K. S. Wood 1964. Infection of plants by *Corticium solani* and *C.praticola* · effect of plant exudates, Trans. Br. Mycol Soc.47 :15-24.

- Dev, V. P. S. 1980. Sheath blight control with soil fungicides. Int. Rice Res. Newslr. 5 : 14-15.
- Dev, V. P. S. and P. K. Satyarajan 1980. Efficacy of certain fungicides in the control of Sheath blight disease of rice. Ag. Res. J. of Kerala 18 : 113-115.
- Dodman, R. and M. T. Flentje 1970. The mechanisms and physiology of plant penetration by *Rhizoctonia solani*. In : J. R. Parmeter Jr. (ed.). *Rhizoctonia solani : Biology and Pathology*. University of California Press, Berkeley, U. S. A. pp. 149-160.
- Durbin, R. D. 1959. Factors affecting the vertical distribution of *Rhizoctonia solani*, with special reference to CO₂ concentration. Amer. J. Bot. 46 : 22-25.
- Eusebio, M. A., M. J. Quimio Jr. and F. P. Ilagan 1979. Canker of Moluccan Sau (*Albiziafalcataria*) in Bislig. Sylvatrop Philipp. For. Res. J. 4 : 191-214.
- Flentje, N. T., R. L. Dodman and A. Kerr 1963. The mechanism of host penetration by *Thanatephorus cucumeris*. Aust. J. Biol. Sci. 16 : 784-799.
- Florence, E. J. M., J. K. Sharma, K. V. Sankaran and C. Mohanan 1985. Some diseases of forest tree seedlings in India caused by *Sclerotium rolfsii* and *Rhizoctonia solani*. Eur. J. For. Path. 15 : 187-190.
- Gadd, C. H. 1927. Report of the Mycologist. Ann. Rep. Tea Res. Inst. of Ceylon for the year 1926 (Bull. 7) : 7-15.
- Gadd, C. H. 1928. Report of the Mycologist. Ann. Rep. Tea Res. Inst. of Ceylon for the year 1927 : 7-18.
- Galindo, J. J., G. S. Abawi, H. D. Thurston and G. Galvez 1983. Effect of mulching on web blight of beans in Costa Rica. Phytopathology 73 : 610-615.
- Georgopoulos, S. G. and S. Wilhelm 1962. Effect of non sterile soil on *Rhizoctonia solani* mycelium in the presence of PCNB. Phytopathology 52 : 361.
- 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. 51 p.
- Griffiths, D. A. 1966. "Die-back" of *Araucaria cunninghamii* caused by *Botryodiplodia theobromae* Pat, in Malaya. Malayan For. 29 : 154-162.
- Grover, R. K. and H. R. Kataria 1985. Management of *Rhizoctonia solani* diseases with chemicals. Proc. Ind. Acad. Sci., Pl. Sci. 94 : 415-431.

- Gurkin, R. S. and S. F. Jenkins 1985, Influence of cultural practices, fungicides, and inoculum placement on southern blight and *Rhizoctonia* crown rot of carrot. *Plant Dis.* 69: 477-481.
- Hopkins, J. C. F. 1950. Summary of Annual Report of the Chief Botanist and Plant Pathologist for the year ended 31st December 1949. *Rhod. Agric. J.* 47: 356-363.
- Jhooty, J. S. and S. S. Bains 1975. Studies on *Rhizoctonia solani*. 111. Reaction of various isolates to different fungitoxicants *Indian J. Microbiol.* 13: 27-32.
- Kataria, H. R. and R. K. Grover 1978. Comparison of fungicides for the control of *Rhizoctonia solani* causing damping-off of mung bean (*Phaseolus aureus*). *Ann. Appl. Biol.* 88: 257-263.
- Keppel, G. 1973. *Design and Analysis: A Researchers Handbook*, Prentice-Hall, London. 658 p.
- Kerr, A. and N. T. Flentje 1957. Host infection of *Pellicularia filamentosa* controlled by chemical stimulates. *Nature (London)* 179: 204-205.
- Livingston, C. H., N. Oshima and M. D. Harrison 1964. Terraclor as a control for various levels of *Rhizoctonia* inoculum in potato soil. *Am. Potato J.* 41: 239-243.
- Maier, C. R. 1962. Response of selected *Rhizoctonia solani* isolates to different chemicals in cultural tests. *Phytopathology* 52: 19.
- Martin, S.B., L. T. Lucas and C. L. Campbell 1984a. Comparative sensitivity of *Rhizoctonia solani* and *Rhizoctonia-like* fungi to selected fungicides *in vitro*. *Phytopathology* 74: 778-781.
- Martin, S. B. Jr., C. L. Campbell and L. T. Lucas 1984b. Response of *Rhizoctonia* blight of tall fescue to selected fungicides in the green house. *Phytopathology* 74:782-785.
- Mildenhall, J. P. and P. H. Williams 1973. Effect of soil temperature and host maturity on infection of carrot by *Rhizoctonia solani*. *Phytopathology* 63; 276-280.
- Munch, E. 1909, Untersuchungen uber immunitat und Krankheitsan fallingkeit der holz pflazen. *Natura. Z. Forst. Land Wirtsch.* 7: 54-75, 87-114, 129-160.

- Nair, K. S. S., G. Mathew and M. Sivarajan 1981. Occurrence of the bagworm, *Pteroma plagiophleps* Hampson (Lepidoptera, Psychidae) as a pest of the tree, *Albizia falcataria* in Kerala, India. Entomon 6: 179-180.
- Oyekan, P. T. 1979. Chemical control of web blight and leaf spot of cowpea in Nigeria. Plant Dis. Rep. 63: 574-577.
- Papavizas, G. C. and C. B. Davey 1961. Saprophytic behaviour of *Rhizoctonia* in soil. Phytopathology 51: 693-699.
- Papavizas, G. C. and C. B. Davey 1962. Activity of *Rhizoctonia* in soil as affected by carbon dioxide. Phytopathology 52: 759-765.
- Parmeter, J. R., Jr. 1970. *Rhizoctonia solani: Biology and Pathology*. University of California Press, Berkeley, 255 p.
- Punithalingam, E. 1980. *Plant Diseases Attributed to Botryodiplodia theobromae* Pat. Bibliotheca Mycologia, Band 7 I, J Cramer Vaduz. 121 p.
- Radha, K. and K. P. V. Menon 1957. The genus *Rhizoctonia* in relation to soil moisture. I. Studies on *Rhizoctonia solani* and *Rhizoctonia bataticola*. Indian Coconut J. 10: 29-36.
- Riffle, J. W. 1978. Development of canker on *Ulmus pumila* related to month of inoculation with *Botryodiplodia hypodermia*. Phytopathology 68: 115-119
- Roth, L.F. and A. J. Riker 1943. Influence of temperature, moisture, and soil reaction on the damping-off of red pine seedlings by *Pythium* and *Rhizoctonia*. J. Agric. Res. 67 : 273-293.
- Rushdi, M. and W. F. Jeffers 1956. Effect of some soil factors on efficiency of fungicides in controlling *Rhizoctonia solani*. Phytopathology 46 : 78-90.
- Sanford, G. B. 1938. Studies on *Rhizoctonia solani* Kuhn. IV. Effect of soil temperature and moisture on virulence. Can. J. Res. (C) 16 : 203-213.
- Sanford, G. B. 1941. Studies on *Rhizoctonia solani*. V. Virulence in steam sterilized and natural soil. Can. J. Res., Ser. C. 19 : 1-18.
- Sarmah, K. C. 1960. Diseases of tea and associated crops in North East India. Indian Tea Assn., Tocklai Experimental Station, Memorandum No. 26 : 68p.
- Scharif, G. 1964. Report on forest diseases in near and middle East. Symp. Int. dang. For. Dis. Insects, Oxford.

- 'Schneider, R. 1953. Untersuchungen über Feuchtigkeitsansprüche parasitischer Pilze. *Phytopathology Z.* 21 : 63-73.
- 'Schneider, C. L. and H. S. Potter 1983. Efficacy of some fungicides in controlling *Rhizoctonia* crown rot of sugarbeet. *J. of the Am. Soc. of Sugarbeet Tech.* 22 : 54-59.
- Seoud, M. B., K. A. El-Alfy, A. T. Thoma and A. A. El-Dib 1982 (published 1984). Further studies on the control of peg and pod rots of peanuts in Egypt, *Agricultural Research Review* 60 : 127-139.
- 'Sharma, J. K., C. Mohanan and E. J. M. Florence 1984. A new stem canker disease of *Eucalyptus* caused by *Botryodiplodia theobromae* in India. *Trans. Brit. Mycol. Soc.* 83 : 162-163.
- Sharma, J. K., C. Mohanan and E. J. M. Florence 1985. Disease survey in nurseries and plantations of forest tree species grown in Kerala. *Kerala Forest Research Institute Res. Rep. No.* 36 : 268 p.
- Shatla, M. N. and J. B. Sinclair 1962. Tolerance of pentachloronitrobenzene and pathogenicity correlated in naturally occurring isolates of *Rhizoctonia solani*. *Phytopathology* 52 : 752p.
- Shatla, M. and J. B. Sinclair 1963. Tolerance of pentachloronitrobenzene among cotton isolates of *Rhizoctonia solani*. *Phytopathology* 53 : 1407-1411.
- Shaw, E. J. F. 1921. Studies in diseases of the jute plant. I. *Diplodia corchori* Syd. *Mem. Dept. Agric. India, Bot. Ser.* 11 : 37-58.
- Shehata, M. R., H. M. Sheir, M. A. E. -Goorani and S. M. El Allaf 1982. Control of wilt and stem rot diseases of carnation *Acta Phytopath. Acad. Scient-Hung.* 17 : 233-237.
- Sinclair, J. B. 1960. Reaction of *Rhizoctonia solani* isolates to certain chemicals. *Plant Dis. Rep.* 44 : 474-477.
- Singh, R. S and B. Singh 1955. Root rot and wilt of *Cyamopsis psoralioides* in relation to thick and thin sowing of the crop. *Agra Univ. India J. Res. (Sci)* 4: 379-385.
- Snedecor, G. W. and W. G. Cochran 1967. *Statistical Methods.* Oxford & IBH Publications Co., New Delhi. 553 p.
- Souza Filho, B.F. de 1979- Effects of fungicides, inoculum density and inoculum depth on isolation of *Rhizoctonia solani* Kuhn. from soil. *Fitopatologia Barsileira* 4: 417-421.

- Spaulding, P. 1961. *Foreign Diseases of Forest Trees of the World*. Agric. Handbook 197, U.S. Dept. Agric., Washington. 361 p.
- Steinmann, A. 1928, Voorloopige mededeeling omtrent het optreden van *Rhizoctonia bataticola* (Taub.) Butler op Java en Sumatra. Arch. Voor Theecult. Nederl. Indie 2: 74-86.
- SubbaRao, M.K. 1939. Report of the Mycologist 1938-39. Ann. Rep. Tea Sci. Sect., Un. Pl. Assn. S. India, 1938-39 pp. 28-42.
- SubbaRao, M. K. 1942. Report of the Mycologist 1941-42. Ann. Rep. Tea Sci. Sect., Un. Pl. Assn. S. India, 1941-42: 27-33.
- Thomas, W.B., Jr. 1962. Reaction of biotypes of *Rhizoctonia solani* to different fungicides. Phytopathology 52: 366 (Abstr.)
- Toole, E.R. 1966. Root rot caused by *Polyporus lucidus*. Plant Dis. Rep. 50: 945-946,
- Townsend, G.R. 1934. Bottom rot of lettuce. New York (Cornell) Agr. Expt. Sta. Mem. 158, 46 p.
- Ullstrup, A.J. 1936. Leaf blight of China aster caused by *Rhizoctonia solani*. Phytopathol. 26: 981-990.
- Venkataram, C. S. 1960. Report of the Plant Pathologist 1959-60. Ann. Rep. Tea Sci. Dept., Un. Pl. Assn. S. India, 1959-1960: 19-32.
- Venkataram, C. S. 1964. Report of the Plant Pathologist 1963-64. Ann. Rep. Tea Sci. Dept., Un. Pl. Assn. S. India, 1963-64: 21-35.
- Venkataram, C. S. 1965. Report of the Plant Pathologist 1964-65. Ann. Rep. Tea Sci. Dept., Un. Pl. Assn. S. India, 1963-1964: 18-78.
- Webster, B.N. 1952. A note on pathological matters. Tea Quart. 23: 84-85.
- Weststeijin, G. 1966. Other pod diseases of cocoa. Ann. Rep. Cocoa Res. Inst. Nigeria (1964-1965). 71 p.
- Wright, N.S. 1968. Evaluation of Terrachlor and Terrachlor Super-X for control of *Rhizoctonia* on potato in British Columbia. Can. Plant Dis. Survey 48: 77-81.
- Yarden, O., J. Katan, N. Aharonson and Y. Ban-Yephet. 1985. Delayed and enhanced degradation of carbendazim and benomyl in disinfested and fungicide-treated Soils Phytopathology 75: 763-767.
- Zentmeyer, C.A. 1965. A laboratory method for testing soil fungicide with *Phytophthora cinnamomi* as test organism Phytopathology 45: 308-404.