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DISEASES OF GINGER

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I. INTRODUCTION

As an Indian folklore goes, Vararuci, one of the nine gems in the fabled court of Vikramaditya was on the look-out for a beautiful, intelligent and sharp witted maiden to become his bride. While accepting the hospitality of a brahmin at Varanasi, he chanced to meet the host's beautiful daughter. Trying to fathom her knowledge, Vararuci posed a request that he be served with a meal containing not less than 100 herbs. Accepting the challenge, the hostess served him with a delectable dish made of ginger along with other items for his noon-day repast. Vararuci's search for the bride was thus rewarded. He chose the woman who knew that ginger was treated as equivalent to 100 herbs.

So much for the importance of ginger in the oriental cuisine. Widely cultivated m many parts of the world like India, Jamaica, Sierra Leone, Nigeria, Southern China, Japan, Taiwan and Australia, it is grown mostly as a kitchen garden crop, and on a limited scale as a cash crop. Thus, the area occupied by the crop is very limited.

Right from sprouting through the growing season and to seed storage, ginger is affected by a succession of fungal, bacterial and viral diseases. In this paper, the diseases have been organised based on their importance and destruction potential.

II. RHIZOME ROT

As the name implies this disease affects the rhizomes which are the economically important produce. Losses of even more than 50% have been reported to be caused by Rhizome rot (Joshi & Sharma, 1982). This disease is caused by a number of species of Pythium (Table 1). When infection takes place through heavily contaminated seed, sprouts fail to grow resulting in pre-emergence damping off. When the disease strikes after sprouting, intial symptoms are manifested in the collar region as water soaking. Soft-rot sets in spreading upwards and downwards. Leaflets become pale, and the borders turn yellow. Lowermost leaflet is affected first. Soon yellowing spreads to all the leaves of the plant from bottom

Table 1. Pythium species reported to be causing soft rot of ginger

ORGANISM	PLACE	REFERENCE
Pythium Sp.	Rangpur, Bihar	MC Rae (1911)
Pythium Spp.	Hawalii	Paris (1939)
P. aphanidermatum	Pusa, Bihar	Mitra and Subramanian (1928)
Edson Fitz.		
-do-	Hyderabad	Vaheeduddin (1955)
-do-	Nagpur	Sahare and Asthane (1968)
P. butleri	Ceylon	Park (1934)
-do-	Malabar, S. Kanara	Thomas (1938)
P. complactens	Ceylon	Park (1937)
P. deliense Meurs	Jabalpur, M.P.	Haware and Joshi (1974a)
P. gracile	Bengal,Gujarat,	Butler (1907)
(de Bary) Schrent	Malabar	
-do-	Assam	Sen (1930)
-do-	Fiji	Parham (1935)
P. graminicolum Subram.	Ceylon	Park (1935)
P. myriotylum Drech	Taiwan	Lin *et al.* (1971)
	Ceylon	Park (1937)
-do-	Poona	Uppal (1940)
-do-	Bombay State	Patel *et al.*, (1949)
-do-	Hongkong	Bertus (1942)
-do-	Nagpur (India)	Sahare & Asthana (1968)
P. pleroticum. T.	Solan, H.P.	Sharma & Dohroo (1980)
P. Vexans de Bary	South India at places above 3,000 ft.	Ramakrishnan (1949)
P. zingiberum	Osaka, Japan	Takahashi (1954)

upwards. Simultaneously, soft tissues of the collar disintegrate and the plant topples. The lignified vascular strands are unaffected. As the rot affects the rhizome, it softens. Other fungi and bacteria follow and hasten the deterioration. Foul smell emits from the rhizomes. Rotting attracts dipteran flies like *Mimegralla* and *Eumerus* species. These flies lay eggs in the tissue and the maggots grow feeding on the rotting contents of the rhizome. Such rhizomes finally have only the jacket intact. The whole inside turns into a bag of maggots, unfit for consumption or planting. Various *Pythium* species have been reported to be attacking ginger and causing soft-rot. The available information has been summarised in Table 2. In most of the cases, *Pythium aphanidermatum* and *P. myriotylum* have been found to be reponsible for the symptoms. *P. vexans* has a restricted geographic distribution. It is prevalent at high altitudes of 1170 meters or above in the Wynad District of Kerala, India. This fungus grows at cooler temperatures while *P. aphanidermatum* & *P. myriotylum* are commonly encountered in the plains where the temperatures are warm. The optimum temperature for gorwth of the fungus 34°C while the maximum is around 40°C. The optimum for *P. vexans is 28°C and no growth takes place beyond 34°C.*

The hyphae of P. aphanidermatum are colorless, much branched threads upto 4μ in diameter. They penetrate cells freely forming usually irregular swellings and the rotting takes place usually in advance of penetration. The non-septate mycelium of the branching hyphae form long tapering zoosporangia by the swelling (Figs. 1 & 2) of the hyphae, when the branches extend to the outside where there is plenty of moisture, or when they are placed in water. The zoosporangium is not cut off from the rest of the hyphae by a septum. After certain internal changes each apical swelling blows out into a vesicle into which the protoplasm migrates. The zoospores (Fig.3) about thirty-five in number are formed in the vesicle which rupture the sporangial wall and escape. These are kidney-shaped, biciliate and slightly depressed at the hilum end, measure 8-12 μ x 6-8 μ in diameter when swimming and 7-11 μ when encysted. Zoospores, after swimming for some time encyst to form walled structures which germinate directly to produce mycelium (Subramanian, 1919).

Mycelium of *P. myriotylum* is broad, upto 8.5 μ in diameter and during the course of growth it forms numerous clavate knob-like appressoria. Sporangia are either terminal or intercalary. As in *P. aphanidermatum* the hyphal tips may swell slightly and form long tapering zoosporangia upto 300 μ in length with swollen or lobulate elements attached laterally. Germination of the sporangium is accompanied by the formation of a vesicle. Reniform zoospores are liberated by the rupture of the vesicle. A zoospore, after a period of free-swimming comes to rest and encysts within a wall and later germinates by producing a single germ-tube.

Table 2. Fungi affecting ginger in storage

	Fungi	Ref.
1.	*Acrimonium kiliense*	Haware, Joshi and Sharma 1974
2.	*A. Strictum*	"
3.	*Alternaria alternate*	"
4.	*Aspergillus flavus*	"
5.	*A. niger*	"
6.	*A. niger*	Iyer *et al.*, 1983
7.	*Cunninghamella echinulata*	"
8.	*Cuminghamella* sp.	Raju *et al.*, 1985
9.	*Fusarium moniliforme*	Rath *et al.*, 1978
10.	*F. Solani*	"
11.	*Fusarium* sp.	Simmonds 1955, 1956 Raju *et al.*, 1985
12.	*Gliocladium* sp.	"
13.	*Macrophomina Phaseolina*	Iyer *et al.*, 1980
14.	*Memnoniella echinata*	Sharma and Joshi, 1976a
15.	*Nectria inventa*	Sharma and Joshi 1976
16.	*Penicillium* species	Iyer *et al.*, 1983
17.	*Pseudopapilaspora Kendrickii*	Sharma, 1977
18.	*Pythium* sp.	Anon. 1953, 73, 74a
19.	*Rhizoctonia bataticola*	Sharma and Jain 1977, Park, 1932 Iyer *et al.*, 1983
20.	*R. Solani*	Park, 1932
21.	*Rhizopus* sp.	Raju *et al.*, 1985
22.	*Sclerotium rolfsii*	Sundaram, 1951. Raju *et al.*, 1985
23.	*Trichurus spiralis*	Iyer *et al.*, 1984

Sexual reproduction may take place within the host, within the scales or tissues. Oogonia are either terminal or intercalary, spherical, smooth, thin walled and 22-41 µ in diameter. Antheridia are typically diclinous and are upto 30 µ long and 4-8 µ wide. They are paragynous, and to each oogonium a number of antheridia (upto 10) may be attached. Oospores are formed singly in each oogonium. They live freely inside the oogonia. They are slightly yellowish, sub-spherical and 16.5 to 30 µ in diameter (Mundkur,1949).

The Hyphae of *P. vexans* are very fine and freely branched. They usually taper at the ends, particularly in irregular manner. Sporangia are usually terminal and rarely intercalary. They are irregularly pyriform to ovoid, rarely globose and 17 to 24 µ in diameter. Zoospore formation is rare. Sporongia germinate directly by germ tubes. Oogongia are mostly terminal and very rarely intercalary. They arise laterally on short branches or may be sessile. Oogonia are 22-25 µ in diameter. Antheridia are clavate, paragynous. One antheridium is usually closely applied to each oogonium. Oospores are smooth, round, yellowish lying free in the oogonium, 20-22 (average 18 µ) in diameter. Germination may be direct or by production of zoospores.

In *Pythium pleroticum*, zoospores are rarely or never produced. The vegetative hyphal bodies are spherical, or ellipsoid and these are less than 40 µ (average 30 µ). The oogonia measure 15-18 µ. The oospores are plerotic or nearly so.

In the case of *Pythium zingiberum*, it has been treated as similar to *P. volutum.*, In this fungus the appressoria consists of lateral falcate structures with rounded ends. The mycelium is mostly intracellular in host tissue. Sporangia consist of small lobulations or torulorid buds formed only rarely in aqueous culture. Discharge tube is usually about 50 µ long and 3-4 µ wide, zoospores biflagellate bean-shaped about 0-14 µ. Oogonia are smooth, subspherical, dark brown, terminal on short side stalks or rarely intercalary, formed copiously in culture,but a large percentage remains sterile. Average diameter 150 µ; antheridia 3-10; each oogonium, crooknecked, sometimes curved or even straight,with narrow apical contact, usually arising from adjact hyphae, each of which supplies one to four antheridia, or more rarely arising from oogonial stalk. Antheridial branches commonly entwine about the oogonial stalk on liquid media, but less frequently on solid media. Oospores smooth, spherical to oblong, usually free within oogonium. Average diameter 27.7 µ, central globules, 14-24, refringent spot 8.5 x 2.2 µ. Oospore wall 2.0 µ and oblong.Oospores average 3.9 x 19.2 µ (Waterhouse 1967,68)

There are two ways in which the disease perpetuates. Firstly through infested soil and secondly through infected seed. Mc Rae (1911) & Butler (1918) considered the use of diseased rhizomes to be the principal factor in the dissemination of the disease. Oospores germinate when rain and temperature favour disease

development. Infected rhizomes contain mycelia and fruiting bodies inside. Oospores are often seen in the scales of the seed rhizomes (Thomas 1938).

Iyer Mishra & Koya (1984) has reported that *Pythium aphanidermatum* could be isolated after one year's fallow period from sick plot. Therefore, control of the disease has many facets viz. controlling the infection in seed, reducing the inoculum in soil, and checking the spread of the disease.

With regard to seed infection control, the best method of course is to use disease-free rhizomes for planting (Bulter, 1918; Park, 1941; bertus, 19420. Hence, collection of seed ginger from a place free of disease has been over-emphasised by workers like Butler (1918), who had suggested that even in such cases, seed material should be carefully selected before planting. Even at the time of storing the seed material for the subsequent season's planting; in CPCRI, Ann Rep. 1962) caution should be exercised. Studies conducted at CPCRI (Iyer *et al*, 1984.) showed that even when 100% apparently healthy seeds were stored, recovery of wholesome seed was 60%. When 10% visibly rotten rhizomes were mixed with 90% apparently healthy looking seeds,the recovery was only to the tune of 18%. Hence the signficance of seed selection and protection prior to storing and planting. It is necessary, therefore, to enforce programmes of seed certification (Joshi & Sharma, 1982) for controlling this disease.

Since the disease is internally seed-borne also, seed treatments can reduce the infection only to a limited extent. Various types of seed treatments including chemical and hot water have been tried by different workers. These seed treatments have been tried prior to storing and also prior to planting. At CPCRI, trials were conducted to compare hot water 42°C for 20 minutes, and cowdung slurry dip followed by smoke treatment independently and in combination (Iyer et al. 1984 CPCRI Ann. Rep. 1982). The crop stand was better in the hot water (alone) treated group in comparison to the others. Various chemicals have also been tried as seed protectants prior to planting. Many workers used 0.1% mercuric chloride against *P. graminicolum*. Thomas (1940-41) used mercuric chloride (0.1-0.25%) for seed treatment. He (1940) also tried ceresan 0.25% treatment for seed materials. At CPCRI, trials were conducted using pyroxychlor (1000-2000 ppm), Agallol (Methoxy ethyl mercuric chloride) 0.25%, Bavistin 0.3%, Benlate 3%, Captafol 0.36%, Dithane M-45 0.3%, Ridomil 0.2% and Terrazole 0.2%. Out of these, Bavistin 0.3%, Dithane M-45 0.3%, Agallol 0.25%, and terazole 0.2% were on par and better than other treatments. Initial moistrue level of the seed material was 81.4% whereas at the time of sowing it was as low as 56.7%. Temperature in the pits varied from 24.5°C to 34°C. Two varying durations of dip were tried viz. 30 minutes and 60 minutes. Both were only equally effective (Iyer *et al.* 84, 85; Raju *et al.*, 1985).

Apart from chemicals, various methods of preservation were also tried by Mishra and Iyer (1981) at CPCRI, Kasaragod. These included dipping the material in cowdung slurry and smoking, preserving the seed in polythene lined pits; in refrigerator (6° +/- 1°C); heaped on the floor; in pits lined with sand, sawdust, leaves of neem, tapioca, *Glycosmis pentaphylla* etc. Of these, it was found that storing the seed in sand lined pits was best. Sharma & Dohroo (1982) used Daconil (0.2%), Emisan 0.1%, Dithane M-45 (0.2%), Blitox-50 (0.3%), Mercuric chloride (0.1%), Panoram (0.2%), Panolil (0.2%) and Anthracol (0.2%).Of these Emisan-6, Blitox-50 and Mercuric chloride were highly effective and on par with Dithane M-45 in controlling *Pythium pleroticum* rot. In case of *Fusarium equisetti* rots, Dithane M-45, Emisan-6, Blitox-50 gave 50, 25 & 25 % disease control respectively. Dithane M-45 & Daconil were highly effective in controlling rots caused by combined infection, recording 67.6, 67.6, 58.8 and 51.4% disease control followed by Vitavax + Thiram, Anthracol and Euparca-M with 47.0, 47.0 and 41.2% respectively (Sharma & Dohroo, 1982).

During the storage of seed ginger a number of organisms were found to colonize it. In addition to potential pathogens, saprophytes were also seen. A list of these is given in Table.

Next and most important source of infection is an infested soil. It has been seen that the fungus can be successfully isolated from sick soil even after one year fallow period (Iyer *et al*, 1984). Hence, the importance of crop rotation is obvious. Butler (1918) recommended crop rotation, In many of the ginger growing tracts, crop rotation is practiced as a routine. In Kerala, normally 3-5 year rotation is common.

This fungus mulitiplies in water and spreads through free flowing water, Hence, it is necessary to reduce the chances of stagnation and flooding and improve drainage (Butler, 1918). Sarma *et al.* (1979) compared growing ginger on ridges and raised beds with reference to the incidence of soft rot. They found that once disease makes its appearance, it spreads with equal ease on ridges and as well on beds.

It is the experience of cultivators that by sowing the crop early in the season, soft rot is checked to a certain extent. By the time the pathogen builds up sufficient inoculum potential, the crop is hardy and chances of infection and aggravation of symptoms is less. From experiments done by Iyer *et al.* (1984) on the effect of date of sowing on the incidence of soft rot it was seen that for Calicut region in Kerala State of India, the whole of May and early June were the best seasons. Next best period was last week of April.

Table 3. Effect of application of soil amendments on the percentage of incidence of soft rot (Rhizome affected)Transformed sin *P* where P is the proportion of soft rot incidence

Treatments	Year 1980-81					Year 1981-82				
	CK[1]	FYM[2]	PC[3]	NC[4]	Mean	CK	FYM	PC	NC	Mean
CHECK	7.5	15.8	10.0	5.5	9.7	4.6	7.5	10.2	8.7	7.7
Lime	8.6	36.3	20.9	9.5	18.8	9.7	39.3	22.5	9.6	20.3
Fertilizer	12.3	12.6	10.9	16.8	13.1	8.5	12.9	13.2	16.5	12.8
Lime+Fertz.	31.5	27.1	23.8	17.4	25.0	37.3	27.5	23.2	17.1	26.3
Mean	15.0	23.0	16.4	12.3		15.0	21.8	17.3	12.9	
CD (5%)	Main plot 8.18			Sub plot 6.20		Main plot 8.94			Sub plot No-	

1. Check - Control
2. FYM - Farm Yard Manure
3. PC - Pongamia cake
4. NC - Neem cake

Another method of manipulating the situation of the standing crop is by the application of various soil amendments. These amendments alter the soil reaction, change the spectrum of soil microflora and thus affect the population of pathogens existing in soil. A field experiment aimed at studying the influence of soil amendments like neemcake, saw dust, dolomite and burning the soil in situ before planting was conducted by Sarma *et al.* (1979). This study was in-conclusive. Experiment was, therefore, again conducted at the Farm in Peruvannamuzhi, Kerala using the following treatments namely (1) lime @ 1.5 tonnes/ha; (2) Fertilizers (Nitrogen @ 75 Kg/ha, Phosphorus @ 50 kg/ha and Potash @ 50 kg/ha); (3) Combination of lime and fertilizers and (4) Control. This was conducted for two years. In both years, incidence of rhizome rot was lower in the organic amendment treated plots. Among these, neemcake and pongamia cake gave significantly lower incidence of rhizome rot when compared to control of FYM applied plot. Incidence of soft rot was lowest in the control plot and highest in plot receiving a combination of lime and fertilizer treatment. In addition, it was found that organic amendments had also increased the availability of mutrients in the soil during crop growth (Sadanandan & Iyer, 1986). Often this amelioration is effected due to the alteration in microbial flora, either due to the increase of antagonists or due to the prevention of the increase of the pathogen or both.

Experiments have been conducted using several of the well established antagonists. Thomas (1939) suggested biological control of *Pythium* spp. using Trichoderma lignorum. In mixed cultures, *T. lignorum* increased acidity of the medium which was unfavourable for the growth of *Pythium*. Sarma *et al.* (1979) tested 81 isolates of fungi, bacteria and actinomycetes for their antagonistic effects against *Pythium* species and isolated two as yet unidentified actinomyctes which showed distinct inhibition zones in culture plates. These when tested in field, did not afford any clear protection. Nambiar and Sarma(personal communication) also tried inoculating *Trichoderma* directly in the plot and tried methods of enriching the population by adding sulphur to soil.

Another aspect of controlling the disease in the field is through fungicides. Ever since the disease was discovered, field trials using chemicals have also been tried.Department of agriculture, Bombay recommended soil drenching using Bordeaux mixture 3:3:50 one week before planting and subsequently at monthly intervals during monsoon. Shahare and Asthana (1962) recommended application of 8 gallons of 0.15% zineb (65%) per plot of '8 X 5' before planting followed by three similar applications at 3-weekly intervals.

Sarma *et al.*, (1974) reported that the incidence of soft rot was reduced to 45.8 % in ceresan (WP) treated plots and against 71.4 % in control. Sarma *et al.*, (1979) reported 22.7 % soft rot in Aureofungin-sol. (200 ppm) treated plots as against 28.34 % in Captafol and 68.8 % in control. Though field control

trials have been repeatedly tried at two locations in Kerala, namely Kasaragod and Calicut, it was observed that Methoxy ethyl mercuric chloride (0.25%) drenched plots fared better in comparison to other fungicides tried at Kasaragod. At Calicut, Dithane M-45 (0.3%) drenched plots had an edge over other fungicides, viz. Captafol (0.2%), Mothoxy mercuric chloride (0.25%), Dexon 0.1% , Ridomil (0.2%), Bordeaux mixture (1%), Heptane antibiotic (200 ppm), Captafol (0.2%), Maneb + Zinc (0.3%), Dithane M-45 (0.36%), Dexon(0.1%), Alliette (0.01%), Terrazole (0.2%), Formalin (0.1%), Bordeaux Mixture (1%), Cheshunt compound (1%). Soil drenching with fungicides does not give consistent results year after year. Hence, it is necessary to resort to other measures of control as well to reduce damage due to the disease.

Once a clump becomes infected, it is difficult to eradicate the infection. Hence, it is necessary to resort to measures of preventing the spread of the disease. The role of free flowing water has already been well established in the spread of the disease.

It has been observed that dipteran flies that are found in the ginger field help in disseminating the disease (Iyer *et al.*,1981). *Pythium* had been successfully isolated from the foregut of field collected *Mimegralla* adult flies. However, the fungus could not be isolated from any other portion of the gut proving that the fungus does not colonise in the insect. Similarly, isolations were attempted from the droppings of maggots that were found within the rotting rhizomes. This also was not successful. These findings point to the adult fly as the positive disseminating agent. The peculiar scratching habit of the fly of the surface of the plant at the base of the clump must be aiding in transmission of the pathogen. Experiments were conducted on the effect of insecticides, singly and in combination with fungicides, at CPCRI. Agallol + Solvirex and Dexon + Lindane (Iyer *et al.*,1982), oftanol + Agallol, Carbofuran, Garvox,5, BHC 50% WP (Iyer *et al.*,1983), Aldicarb, Sovimol, Phorate, Thiodematon (Iyer *et al.*,1984) and Methyl parathion were tested. Of these, Methyl parathion (0.05%) proved to be the best, followed by Carbofuran 0.05%. Taking lead from the various experiments, comprehensive field trials were laid out by Iyer *et al.*, (1983) and Raju *et al.*,(1985), at different locations so as to evolve a package of practices of plant protection to be adopted. An integrated control measure is, therefore, suggested (Fig. 4).

III. BACTERIAL WILT

After rhizome rot, the bacterial wilt assumes considerable importance wherever ginger cultivation is undertaken. Causal organim of this disease is *Pseudomonas solanacearum*. biotype III (Hayward) and biotype IV. This disease was reported from Mauritius in 1953 by Orian for the first time. Later, this was reported from Australia (Hayward *et al.*,1967), Queensland (Hayward and Moffet, 1971),

rom Western hemisphere (Ishii and Aragaki, 1963; Quinon, Aragaki and Ishii, 964), Philippines (Pordesimo and Raymundo, 1963; Zehr, 1969), and Malaya Navarathnam, 1967). From Madras, this disease was reported in 1941 by Thomas. ubsquently from Kerala, this was reported by Sarma, Indrasenan and Iyer (1978), nd by James Mathew *et al.*,(1979).

Earliest symptoms appear as water-soaked patches or linear streaks on the collar egion of the pseudostems followed by yellow to bronze coloration of the margin f the lowermost leaf which gradually progresses upwards. Later, the leaves become laccid with intense yellowish bronze color and droop, exhibiting typical wilt symptoms. The ligules and leaf sheaths in the infected plants appear yellowish to dull reen. Finally the leaves roll up and the whole plant dries up (Fig.5). Pseudostems come off casily with a gentle pull. They break off at the base also very asily . At the advanced stage, the pseudostem is slimy to touch. Affected plants tand persistently and do not collapse and fall prostrate (Fig.5). A milky bacterial exudate oozes out on pressing the rhizome gently. When infected tissues re steeped in clear water for a while, the water turns cloudy and milky (Fig.6).

Of the causal prganism *Psedudomonas solanacearum* (Smith), Both biotype II and Biotype IV whereas have been described, III from India, III and IV from Australia. Biotype III causes slow wilt.Biotype IV causes rapid wilting and death.

P. solanacearum biotype III has a host range restricted to ginger Only,*Pseudomonas solanacearum* biotype IV has a wide host range including tomato, potato, apsicum, *Physalis peruviana, P. minima, Solanum mauritianum, Solanum nigrum, Solanum spp., Crassocephalum crepidibides,* brinjal, groundnut and tobacco. Qunion *et al.* (1964) described 3 strains of *P. solanacearum* which differed in heir pathogenicity to tomato, groundnut, edible ginger and bird of paradise. The ginger strain was found to be only weakly pathogenic compared to the tomato and bird of paradise strains (Quinon *etal.*,1964). Lum's (1973) isloate did not produce symptoms on tomato, tobacco and groundnut. The organism could be isolated even after a gap of 20 months from infected plots. Pegg and Moffet (1971) suggested that, to check the disease, destruction of alternate hosts should be given extra emphasis. Crop rotation and treatment of seed using 0.6% mercurial seed protectants had been recommended by Pordesimo and Raymundo (1963). Ishii and Aragaki (1963) observed that soil fumigation with methyl bromide at the rate of 3 lbs/100 sq. ft. checked the disease.

Scientists at CPCRI (Anon.1986), Calicut, India reported that the incidence of bacterial wilt was delayed by more than a month when seed rhizomes treated with streptocycline (200 ppm) or plantomycin (200 ppm) were planted. These fared better when compared to untreated control or hot water treatment at 45°C for 30 minutes at Calicut (India).

IV. PHYLLOSTICTA LEAF SPOT

This was first described in 1942 from Godavari District in Andhra Pradesh by T.S. Ramakrishnan. Subsequently, it has been reported from Philippines (Chaniongco, 1966), Mauritius (Anon, 1971) and Sarawak (Anon, 1972). In India, it has been reported from Kerala (Anon, 1974), Maharashtra (Kaware, 1974) and Himachal Pradesh (Sohi *et al.* 1964).

Initial symptoms on the leaves show presence of small oval to elongated spots, measuring 1-10mm x 0.5 to 4 mm. As the spot grows old, it develops a white papery centre and dark brown margin with a yellowish halo surrounding it. Closely located spots grow and coalesce to form large lesions. Leaves become shredded and disfigured and suffer extensive desiccation. Young leaves are affected first. As the crop puts forth fresh leaves, the these get infected subsequently. The crop attains a grey dishevelled look as a result of infection.

A. Pathogen: The causal organism is *Phyllosticta zingiberi* T.S. Ramakr. Pycnidia of the fungus are found in the central white portion of the spot. They are first immersed below the epidermis and later become erumpant. They are distrinctly ostiolate and measure 3.7 - 7.4 µ x 1.2- 2.5 µ, averaging 4.3 µ x 1.6 µ. Hyphae are hyaline. Pycnidia are formed in old cultures. Pycnidiospores (conidia) are hyaline, oblong with rounded ends and are often biguttulate.

B. Epidemiology : Primary inoculum is the infeced leaf or seed. In the leaf, pycnidiosperes and mycelia remain viable for 14 months under laboratory conditions. Under field conditions, pycnidia survived in the leaf debris throughout summer season (30-33°C). Pycnidiospores were viable in soil even at 25°m depth after 6 months. Plants raised in unsterile soil from heavily infected fields as well as those grown in sterilized field soils inoculated with fungus mass showed disease symptoms. Seedlings raised in pots the surface of which was covered with infected leaf debris developed disease in the very early stage itself (Figs. 7 & 8) Thus, the primary inoculum consists of infected seed and as well inoculum lurking in the debris in an infected field (Brahma & Nambiar, 1982). Pycnidia are abundant in the fallen leaf. The number of pycnidia per lesion increased with increase in size of lesion, and so also the number of spores (Table 4). Spore release from pycnidium depended upon its contact with water drops. Pycnidiospores were released in the form of cirrus through ostioles. Dew drops collected in the morning hours contained a large number of germinating spores. For the subsequent dispersal, these had to depend upon rain splash (Fig. 9). Pycnidiospores trapped at different distances and heights from the focus of infection under different amounts of rainfall are shown in Tables 5 & 6. The extent of dispersal either horizontally or vertically was dependent upon the intensity of preciptation. High intensity of rain accompanied by wind seems to exert greater impact on target leaf,

Table 4. *Phyllosticta zingiberi*.Number of Pycinidia under different lesion sizes in leaf spot of ginger

Lesion area (mm^2)	No. of Pycinidia	No. of Pycinidiospores (10^5)
≤ 2.50	8.10	6.50
2.50 - 5.00	9.93	8.00
5.00 - 7.50	11.73	9.70
7.50 - 10.00	12.39	10.25
10.00 - 12.50	15.63	13.00
12.50 and above	17.04	14.10

Table 5. *Phyllosticta Zingiberi*. Horizontal Splash Dispersal of Pycnidio spor

Date of Observation	Rainfall (mm)	Distance (cm)	Spores Trapped (000'ml)
1-10-1980	6.4	30	20
		60	20
		90	10
		150	—
1-10-1980	24.3	30	100
		60	50
		90	30
		120	10
		150	—
3-10-1980	15.2	30	80
		60	60
		90	30
		120	10
		150	—
11-10-1980	42.8	30	90
		60	60
		90	30
		120	20
		150	—
13-10-1980	5.2	30	20
		60	10
		120	—
		150	—
23-10-1980	26.4	30	60
		60	30
		90	20
		120	10
		150	—

Table 6. *Phyllosticta zingiberi* Vertical splash dispersal of pycnidiospores

Date of observation	Rainfall (mm)	Height (cm)	No. of spores trapped (000 ml)
25-10-1980	8.3	30	20
		60	10
		75	—
		90	—
17-11-1980	19.8	30	30
		60	20
		75	10
		90	—
19-11-1980	11.3	30	20
		60	10
		75	—
		90	—
20-11-1980	5.5	30	20
		60	10
		75	—

so that ring drops are splashed to greater distances, resulting in liberation of greater amounts of spores and increasing disease incidence (Table 7) (Brahma & Nambiar, 1984).

The disease begins to appear in traces towards the end of June, though the plants are at the most susceptible stage (3-4 leaf stage) and would have received high cumulative rainfall (1003.13mm) for disease spread. During the first fortnight of the month, because of drier conditions, build up of inoculum is not sufficient. During this period, the temperature varies between 23.4 - 29.6°C and relative humidity is between 83.3-90.1%. Later in July when the number of rainy days and the total rainfall increase, the disease aggravates and also spreads fast. A temperature range of 23.0 - 28.0oC is found to be suitable for the fungus. As the number of rainy days decrease.and days warm up, disease spread decreases (Table 7) (Brahma & Nambiar, 1984). Ginger plants upto the age of 6-7 months are susceptible to the disease. Two weeks-old leaves are more susceptible than those of six weeks age.

Table 7. Mean temperature, relative humidity, rainfall and number of rainy d viz-a-vis leaf-spot disease incidence* in ginger by *Phyllosticta Z giberi*

	Temperature °C		Humidity (%)		Rainfall	No. of rainy	Disea incide
	Max.	Min.	F.N.	A.N.	(mm)	days	nce (%
June	29.6	23.4	90.1	83.3	1003.14	27.0	Traces
July	28.5	22.9	95.6	85.1	1084.30	29.9	10.05
August	28.5	23.1	94.3	84.3	763.20	28.3	19.00
September	29.2	22.6	93.0	77.0	239.00	17.3	22.55
October	31.3	23.2	95.0	70.3	99.10	10.3	21.72
November	31.9	22.1	91.3	64.0	115.15	9.0	18.99

* Mean of three years

When a concentrated culture filtrate of the fungus was applied to the leav water-soaked lesions were formed after 23 hrs at room temperature (27-30°C These were similar to those caused by the fungus on ginger leaves. It has be observed that the disease is severe when the crop is grown under exposed co ditions. The beating action of the rain is more severe under such conditions wh the rain falls straight without any interruption. However, when ginger is grow under partial shade, this direct drop is interrupted and thus the force of the dro reduced. This results in rain splashes of lesser intensity and thus decrease in t vertical or horizontal dispersal of conidia of *Phyllosticta*. Hence it is advisab to grow ginger under partial shade than in the open (Fig.10). Whenever perenni crops are not present, other crops like quick growing perennial, red gram, m be planted to intercept the wind and also to cast some shade on the crop.

Chemical control trials have been taken up on this organism by various workers chattopadhyay, 167; Brhma and Nambir (personal communication). Bordeaux mi ture 1% and captan 1% are both effective in checking the spread of the disea and also in reducing deterioration due to the disease.

A specific toxin is associated with the symptom production and tissue damage and can beeasily extracted from culture filtrate and concentrated. This can be used for screening ginger tissues that are tolerant/resistant to *Phyllosticta*. From such tissue, resisant plants can be regenerated and field tested (Kuruvinashetti, *1988* personal communication).

Table 8. Incidence of mosaic in different types of ginger during 1972-73 at Central Plantation Crops Research Institute, Kasaragod

S.No.	Type	Mosaic incidence (%)
1.	Gujarat II	13.30
2.	Peechi	15.56
3.	Wynad Manantody	26.67
4.	Rio-de-Janeiro	27.78
5.	Himachal Pradesh	31.67
6.	Sierra Leone	32.22
7.	Zacheerabad	33.33
8.	Gujarat I	33.33
9.	China	35.00
10.	Assam	38.89
11.	Wynad Local	38.89
12.	Vengara	40.00
13.	Tinlaidium	41.67
14.	Utter Pradesh	41.67
15.	Maran	41.11
16.	Thingpuri	42.22
17.	Jugijan	43.33
18.	Tura	45.00
19.	Burdwan	46.11
20.	Valluvanad	46.11
21.	Wynad Kunnamangalam	47.78
22.	Mysore	51.11
23.	Narasapattam	51.11
24.	Thodupuzha	51.67
25.	Ernad Manjeri	52.78
26.	Karakkal	53.89
27.	Jorhat	56.67
28.	Bajpai	62.22
29.	Ernad Chernad	64.17
30.	Nadia	65.56
31.	Poona	56.11

V. YELLOWS DISEASE

A. C.O.Fusarium species:

This was first described by Simmonds (1955) from Queensland. Later this w reported from Hawaii (Trujillo 1963) and India (Haware & Joshi1973b).The sp cies was designated as *Fusarium oxysporum* f. sp. zingiberi by Trujille (1963 Kumar (1977) described *Fusarium solani* to be causing wilt of ginger in Ka nataka State of India.

Symptoms of the disease manifest on the leaves as yellowing of the two ma gins of the lower leaves which gradually spreads, covering the entire leaf. Old leaves dry up first followed by the younger ones. Plants may thus show pr mature drooping, wilting, yellowing and drying of plants in patches or in who bed. Plants do not fall to the ground. The basal portions of the affected pla becomes soft and watery. The sheet can be easily pulled out from the mother rh zome. Stunting of plants is a common symptom. In the rhizomes, a cream to brow discoloration accompanied by shrivelling is seen. Central rot is prominent. Roo rot and rhizome formation is affected. In the final stages of decay, only the fibro tissue remains within the rhizome. A white cottony fungal growth may develo on the surface of stored rhizomes.

Haware & Joshi (1973b) reported that symptoms appeared on plants after tw months of planting inoculated rhizomes in sterilized soil. Microscopic examin tion of the infected plants revealed vascular discoloration after 25-30 days inoculation. Hyphae were present in the discolored vessels but none in the co tical tissue in the beginning. Later, cortical rot developed and inter-and intra-c lular mycelium was observed in the cortex also.

The disease perpetuates through rhizomes and infection lurks even in appa ently healthy rhizomes. Such rhizomes rot in storage and fail to germinate whe sown in the next season.

A temperataure range of 15-30°C is favourable for disease development, th optimum being 23-29°C accompanied by very high humidity and continuou presence of a free film of water (Sharma & Jain,1978b). Sharma & Jain (1978 while studying the colonising capacity of yellows disease pathogen using diffe ent soils, found that rhizosphere soil of healthy ginger showed higher suppressio as compared to that of diseased ginger. Agrawal *et al.*, (1974) observed that th medium incorporated with galled root (galls caused by *Meloidogyne* sp.) extra of gigenr supported better growth of the pathogen then healthy root extract mediu

The disease is both soil and seed-borne, Secondary spread takes place through irrigation water and by mechanical means. The fungus perennates as chlamydospores (Sharma & Jan, 1978).

1. Control

Simmonds (1958,1959) suggested seed treatament with mercural fungicides @ 2 lbs/40 gal of water for the control of this disease (Agrawal *et al.*, 1974). However, Haware & Joshi (1974b) and Sharma & Jain (1978a) suggested the control of the disease by soaking seed rhizomes in fungicidal suspension of 0.3% Dithane M-45 or Benlate or Bavistin for 2 hrs. followed by two (or more is necessary) drenchings @ 6 lit./sq. meter at 0.3% concentration at the rate of sowing and before 15th day. A third drenching was recommended if the disease persisted. Drenching of soil was recommended for infected areas only. Crop rotation is a desirable practice, as Sharma & Jain (1978a) suggested that continuous cropping in the same field helps in build up of the inoculum. In pot culture studies, biological control of the pathogen was obtained with *Bacillus subtilis, Memnoniella echinata* and *Aspergillus niger* (Sharma & Jain, 1978b). However, in sick soils, organic amendments like sawdust, oilcakes, maize meal and fallen leaved did not have any ameliorating effect *(Sharma & Jain,1978b).*

VI. THREAD BLIGHT

This was reported from Pattambi, Kerala by Sundaram in 1954. At first, small water-soaked lesions appear on the margin as well as on other parts of the leaf blade. Infected leaves lose turgidity, hang down and later get detached from the sheath. In advanced stages of attack, small brown scletion are formed on the lower surface of the same area. Infected portions turn white and papery on drying. Infection is restricted to leaf blades and does not proceed to the leaf sheath or other parts. All stages of leaf are attacked. Hyphae grow over the leaf and also enter in the leaf through stomata and spread intercellularly. Infection spreads rapidly during the monsoon season.

The fungus grows rapidly on different media. Mycelial growth consists of much branched hyphae 6-10 μ in thickness. In course of three to four days, chocolate-brown sclerotia with pubescent surface are abundantly formed in culture. These are generally larger than those formed on the host. The fungus was named *Pellicularia filamentosa* (Pat) Rogers by Sundaram (1954).

This disease is not of much significance and can be prevented by 1% Bordeaux mixture spray as prophylactic. This disease is also checked by Bavistin spray @ 2g/litre.

VII. HELMINTHOSPORIUM LEAF SPOT

This leaf spot was reported from Pusa (Bihar) by MC Rae in 1924a and subsequently by Mitra (1930,1931) and has very limited occurrence. The causal organism is Helminthosporium maydis (Nisik & Miyake) *(Cochliobolus of hosts heterostrophus)*. This organism is reported to have a wide ranges like Barley, Wheat and Oats.

The disease appears during the rainy months on the host in the form of small oval spots (4-5 mm x 2-3 mm in diameter), scattered on both surfaces of the leaves. The spots enlarge in size. Mature spot has a centrally dead straw-coloured area surrounded by a brown ring with a yellowish zone on the outside.

Conidiophores arise from the central dead area of the spot. Conidia on the host are straight to crescent-shaped with both ends rounded, brown, 3-9 soptate and 43-109 µ in length and 11.5-20 µ in breadth. This fungus grows best between 25 and 30°C. Thermal death point of the organism is between 63-64°C and growth is favoured by darkness. It can grow and sporulate on a variety of media. Optimum temperature for conidial development is 25°C. Sporculation begins at 7°C and is inhibited at 35°C. Conidia produced in culture media are predominantly lunar shaped. But they may be elliptical to cylindrical, curvature being influenced by medium. They are light olivaceous, dark brown or opaque with cells distinctly lighter. Conidia produced in culture media measure 30-115.5 µ x 11.5-20 µ (average 77 µ x 14 µ) 11-11 septate (average -6). Though the ginger isolate could not infect maize, Mitra (1931) considered it to be a strain of the maize pathogen. No work relating to the control of this disease is available on record.

VIII. COLLETOTRICHUM LEAF SPOT

This disease is of common occurrence in different parts of India. This was first described by Sundararaman (1922) from the Godavari District of Andhra Pradesh and the pathogen was then identified as *Vermicularia zingiberis* Sundar. Butler & Bisby (1931) named the pathogen as *Colletrotrichum zingiberis* (Sundar) Butler & Bisby. Other reports about this are Briton Jones (1933), Mc Rae (1924), Balagopal *et al.* (1975), Nema and Agrawal (1960), Park (1932) & Wallace and Wallace (1945).

Symptoms on the leaves and leaf sheaths manifest as small, round to oval, light yellow spots which gradually increase in size and often coalesce together to form large discolored areas. These areas often dry up at the centre, forming holes. In case of severe attack the entire leaf dries up. If the leaf tips are first attacked, the leaf bends and droops down. Infection is usually localized. The disease makes rapid progress during rains, under continued wet weather and high temperature. A change to a drier weather often checks the progress of the disease.

Fructifications appear in the centre of the lesions as minute dark dots which are arranged in irregular concentric rings. The hyphae are hyaline, sepate and vary in diameter from 2-8 µ. Acervuli are circular to oval, closely aggregated, black and 50-140 µ in diameter. Setae are numerous in the centre of the lesions. They are erect, dark brown, septate and 86-168 µ in length. Conidia are subfusoid, curved with rounded ends and measure 17.5 - 25 µ x 3.2 - 4.2 µ. Conidia germinate readily under suitable conditions by producing germ tubes which may form round to ovate to irregularly lobed appressoria at the end. They are dark olive in colour and become septate occasionally and each cell has a germ pore. This pathogen has been found to attack only ginger. It is not known as to how the fungus perennates in nature. Secondary spread is through conidia, dispersed by rain.

Removal and burning of affected plants and prophylactic spraying of 1% Bordeaux mixture during the growing season has been found to control the disease effectively. Two applications of Bordeaux mixture at an interval of 6 weeks during the growing season is necessary.

IX. OTHER FUNGAL LEAF SPOTS

Few other fungal leaf spots have also been reported on ginger which are of very limited occurrence. Not much information is available on them. These are *Phakospora* leaf-spot (Ramakrishnan,1956), *Cercospora* leaf-spot (Kar & Mandal, 1969; Sharma and Joshi,1976b), *Coniothyrium* leaf-spot (Anonymous,1938 Stevens & Atienza, 1931), *Piricularia* leaf-spot (Nishikada,1927; Rathaiah,1979) and *Septoria* leaf-spot (Turner,1971).

X. MOSAIC DISEASE OF GINGER

This virus disease has been recorded from Kasaragod, Kerala by Nambiar and Sarma (1975) and from Jabalpur by Sharma and Joshi (1977).

The first symptoms appear as chlorotic flecks. These may later develop into slender spindle-shaped chlorotic streaks. These coalesce to form large spots. With the advancement of the disease, these strips, together with intervening green areas form a mosaic pattern. Infected leaves are conspicuously yellow. They are neither malformed nor reduced in size. This causal organism appears to be systemic as the disease appears when diseased rhizomes are sown. Ganguli and Raychoudhuri (1971) reported this to be wheat streak mosaic virus which is sap-transmissible from wheat to ginger. Nambiar and Sarma (1974) reported that sap transmission from ginger to ginger, ginger to *Nicotiana tabacum* var. *Harrison Special, N.tabacum* var. *rustica, N. Tabacum* var. *Xanthii, N. glutinosa, Elettaria cardamomum, Curcuma longa* and *C. arometica* gave negative results. Hot water and hot air treatments of rhizomes from affected plants at 45°C and 50°C for 3 hrs., 6 hrs.,

and 12 hrs., did not alleviate symptoms. Observations on the natural inciden of the disease on various varieties and cultivars revealed that the type 'Poon showed maximum incidence (76.11%) while the type Gujarat II, showed mi mum (13.3%) (Table 8).

XI. BIG BUD

This disease is caused by the Tomato big bud organism (MLO) and was report from Queensland by Pegg *et al.*,1974,(Fig.11).

Affected plants cease to grow and leaves become bunched at the top of t stem. As the disease advances, plants turn yellow and die.

The pathogen has a wide host range and the disease is transmitted by leaf ho pers.

Since the occurrence is very limited, no special control measures have be tried. However, in seed production areas, affected plants should be removed a destroyed.

XII. NEMATODE DISEASES OF GINGER

Ginger has been found to be infected by a number of genera of nematodes inclu ing *Helicotylenchus, Hemicycliophora, Hopololaimus, Meloidogyne, Radopholu Rotylenechulus* and *Tylenchorhyncus*(Sundararaju *et al.*, 1980). However, par sitization by *Meloigogyne* sp. and *Radopholus* sp. has been worked out in deta

Root knot nematodes produce extensive galling of roots and stunting of plan Eggs of *Meloidogyne incognita* fail to survive the summer in the upper 10 c layer of soil under Kerala conditions (Nadakal,1963). Histopathological studi revealed that the target of infection was near the xylem pole (Shah & Raju, 197 and that the nematode induced the formation of giant cells and active divisi of cells surrounding the infected area. Hyperplasia of parenchyma cells w common in the infected rhizomes and roots and gall formation was conspicuo in adventitious roots.

Butler and Vilsoni (1975) reported heavy infestation of ginger by *Radopholu similis* in Fiji. Spread of the nematode had been attributed to infested planti materials. Nematodes penetrate the rhizomes and penetrate the tissues interc lularly (Vilsoni *et al.*,1976). Heavy infestation results in destruction of tissu and formation of channels or galleries within the rhizome. Infected ginger plan

xhibit stunting, chlorosis and sparse tillering.

In Fiji, for controlling root-knot nematodes, hot water treatment by steeping hizomes in water at 50°C for 10 minutes is recommended. Colbran (1972) in Australia assessed the efficacy of granular nematicides such as MOCAP, Nemcur, Temik and Vydate against *Meloidogyne javanica*, and Nemacur was found o be most effective. Rhizome yield went upto 15%. Split applications were found o be more efficient as against the single large doses added just prior to monsoon. olbran also found that using saw dust for mulching reduced disease incidence.

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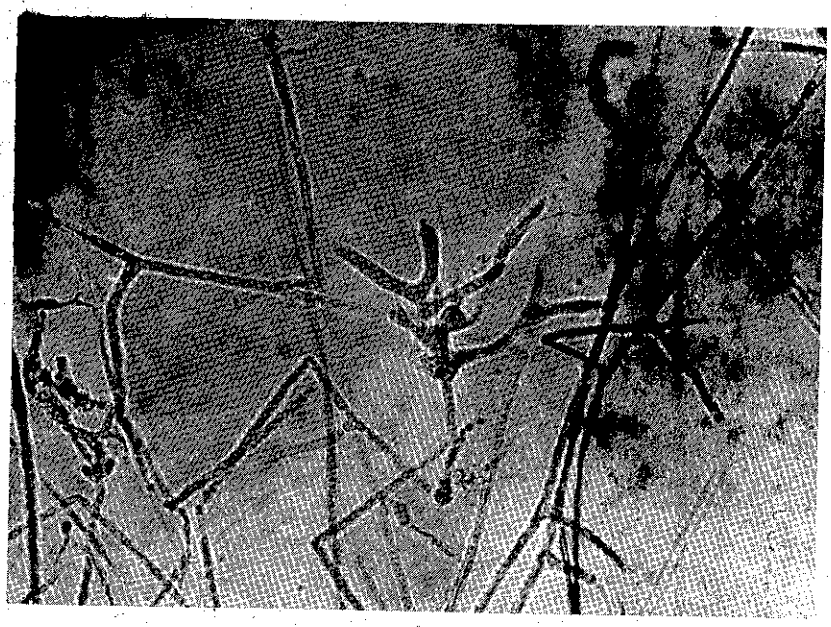

Fig. 1: *Pythium aphanidermatum* causing soft rot of ginger.

Courtesy (Central Plantation Crops Research Institute, Kasaragod 670 124.)

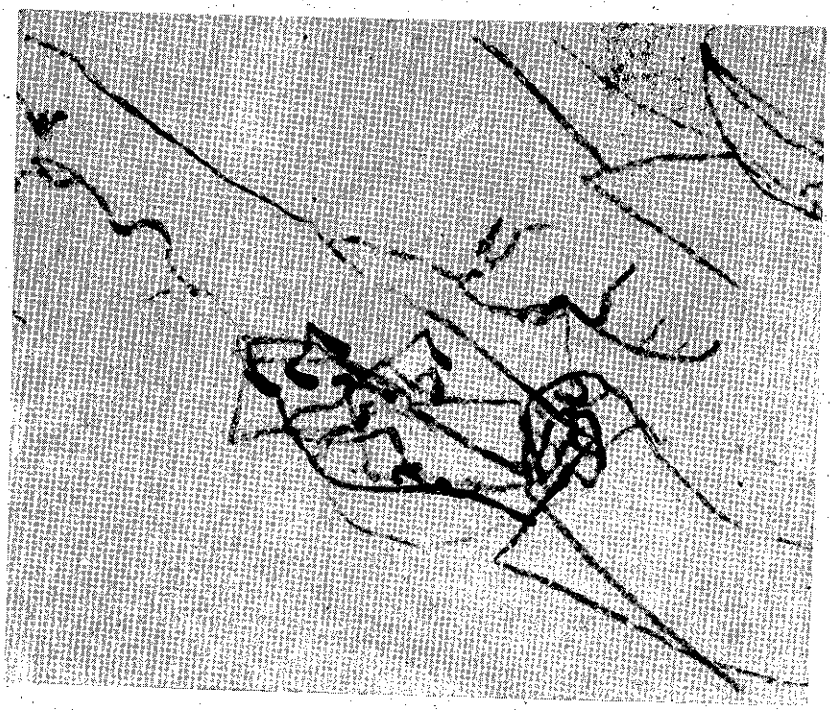

Fig. 2: Sporangial initiation in *P. aphanidermatum*

Courtesy (Central Plantation Crops Research Institute, Kasaragod 670 124.)

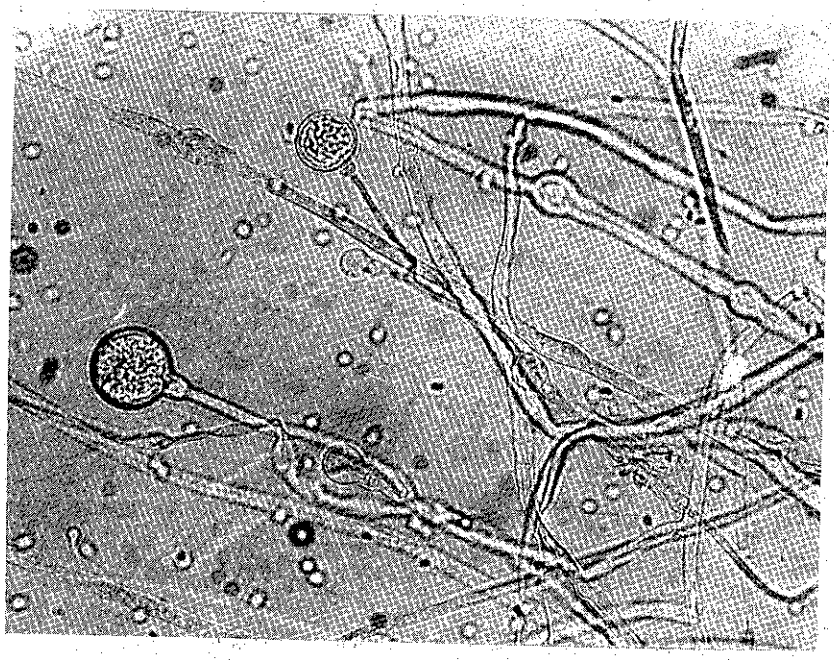

Fig. 3: Zoospore release

Courtesy (Central Plantation Crops Research Institute, Kasaragod 670 124.)

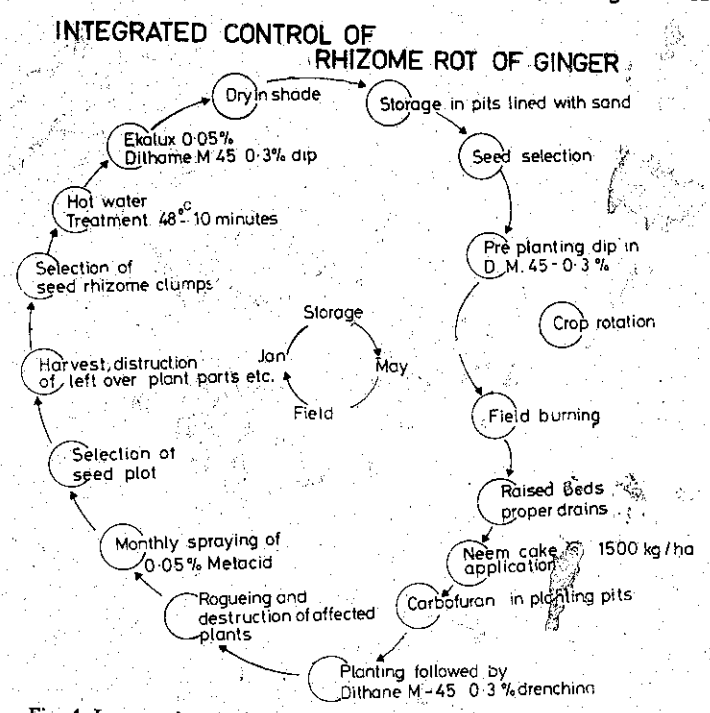

Fig. 4: Integrated control of rhizome rot of ginger

Courtesy (Central Plantation Crops Research Institute, Kasaragod 670 124.)

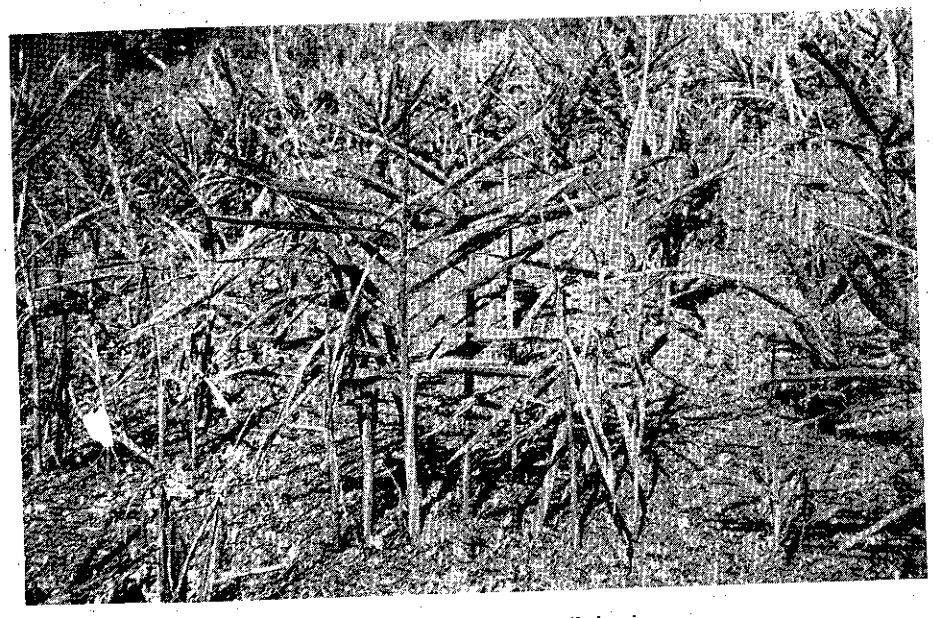

Fig. 5: Symptoms of bacterial wilt in ginger

Courtesy (KG Peg, Australia)
H = Healthy
I = Infected

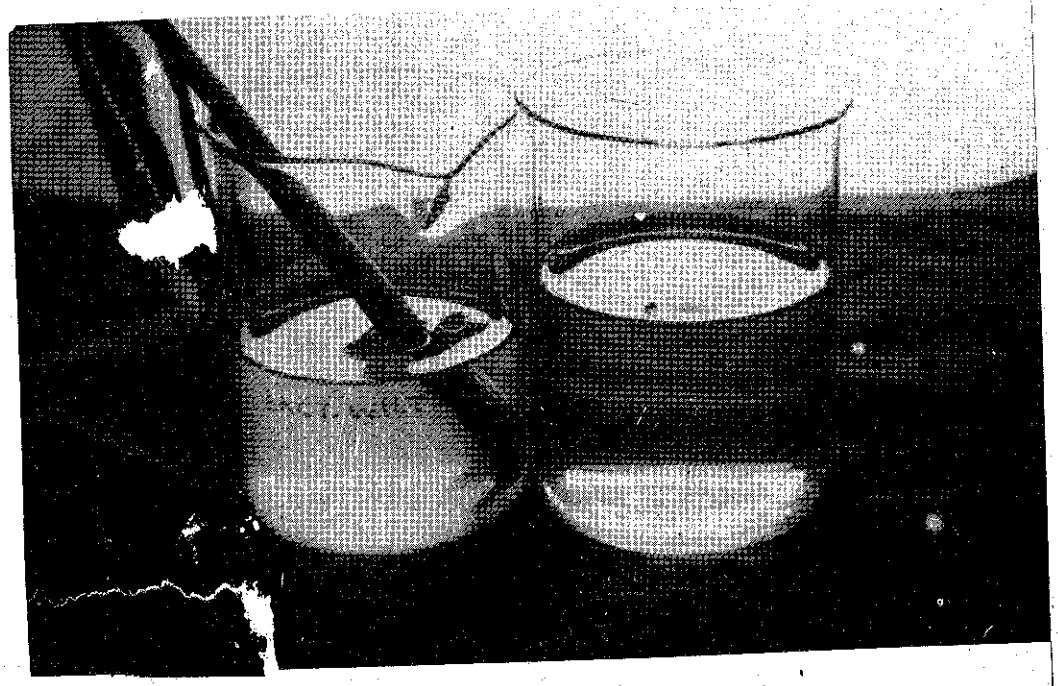

Fig. 6: Detection of bacterial wilt. Bacterial oze turns water milky

Courtesy (Central Plantation Crops Research Institute, Kasaragod 670 124.)

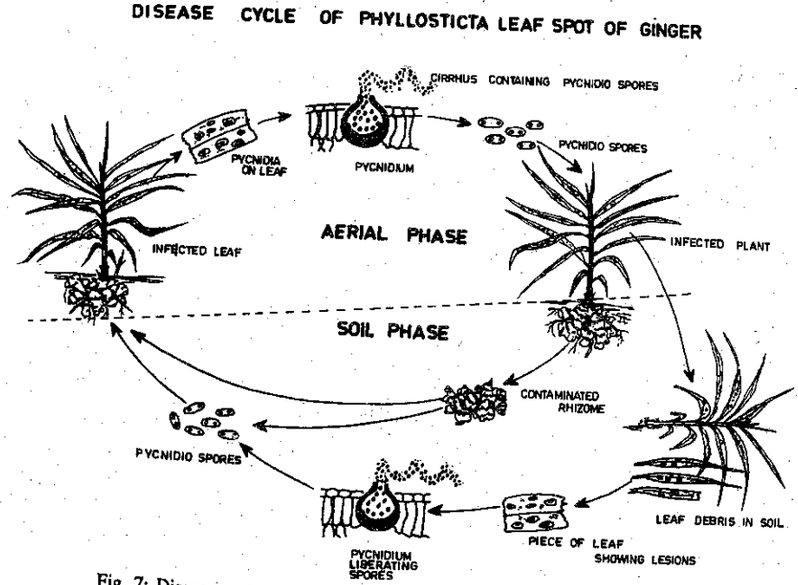

Fig. 7: Disease cycle of *Phyllosticta* leaf spot of ginger

Courtesy (Central Plantation Crops Research Institute, Kasaragod 670 124.)

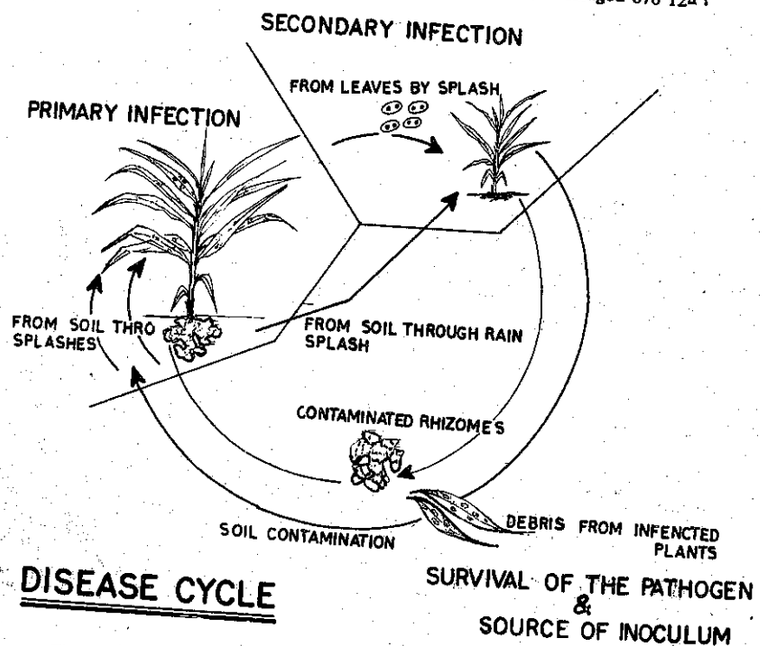

Fig. 8: Survival of the Pathogen and source of inoculum of *Phyllosticta zingiberi*

Courtesy (Central Plantation Crops Research Institute, Kasaragod 670 124.)

Fig. 9: Splash trap for collecting conidia of *Phyllosticta Zingiberi*

Courtesy (Central Plantation Crops Research Institute, Kasaragod 670 124.)

Fig. 10: Raising perennial red gram to reduce *Phyllosticta* infection in ginger

Courtesy (Central Plantation Crops Research Institute, Kasaragod 670 124.)

Fig. 11: Symptoms of big bud

Courtesy K. G Peg, Australia

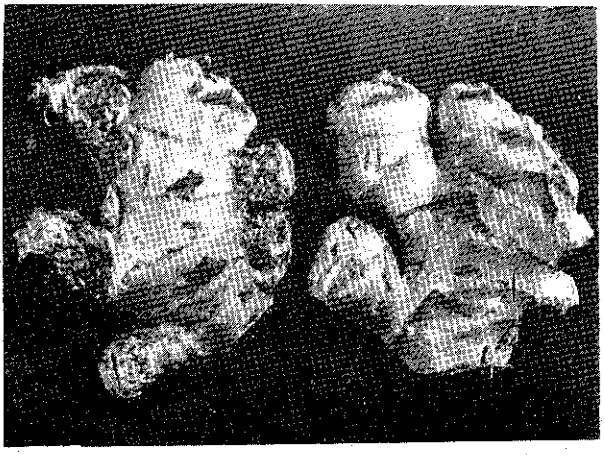

Fig. 12: Symptoms of root knot infection in ginger

Courtesy K G Leg, Australia

I = Infected

H = Healthy