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Rhizome Rot Diseases of Ginger and Turmeric

Y.R. SARMA

1. INTRODUCTION

Rhizome rot is the major disease problem affecting the two rhizomatous crops viz., ginger (*Zingiber officinale*) and turmeric (*Curcuma longa*), which belong to Zingiberaceae. Even though important foliar diseases do exist in these crops, rhizome rot is very important, in view of severe crop losses, it incurs in several parts of India where these crops are grown. The term rhizome rot is loosely used for all the diseases affecting the rhizome irrespective of the pathogens involved, since the ultimate result is the partial or total loss of rhizome. The pathogens involved decide the nature of damage and symptom expression. The major diseases identified are the soft rot resulting in wet rot caused by *Pythium* spp., yellows caused by *Fusarium oxysporum* f. sp. *zingiberi* causing vascular wilt and bacterial wilt caused by *Pseudomonas solanacearum*, Haywaard Biotype III. Besides, *Fusarium solani* causing wet rot and *Macrophomina phaseolina* causing dry rot have been reported.

Distribution of various pathogens associated with rhizome rot of ginger in different parts of India is given in Table 1. Because of differences in etiology in a given location, the disease control has been found to be of little success with uniform fungicidal treatments adopted.

It is also a coincidence that the pathogens involved are almost similar in both these crops and as such the disease management in general would be similar. However, for convenience, both are dealt separately. The problems have been reviewed earlier (Chattopadhyay, 1967; Sharma and Jain, 1977a; Joshi and Sharma, 1982; Iyer, 1987). This review presents the current status of these diseases in India.

The correct diagnosis of the disease in a given location and involvement of one or more than one pathogen should be clearly understood to plan effective integrated management measures which could be location specific.

Table 1 : Distribution of pathogens involved/associated with rhizome rot of ginger crop in India

Organism	Place	Reference
Pythium aphanidermatum Edson Fitz.	Pusa (Bihar)	Mitra and Subramanian (1928)
-do-	Kerala	Sarma *et al.* (1979)
-do-	Hyderabad (Andhra Pradesh)	Vaneduddin (1955)
-do-	Nagpur (Maharashtra)	Sahare and Asthana (1968)
-do-	Madhya Pradesh	Haware and Joshi (1974a)
P. butleri (Syn. *P. aphanidermatum*)	Malabar (Kerala)	Thomas (1938)
-do-	South Kanara (Karnantaka)	Thomas (1938)
P. gracile [(deBary) Schrent] (Syn. *P. aphanidermatum.*)	Bengal, Gujarat, Malabar (Kerala)	Butler (1907)
-do-	Assam	Sen (1930)
P. deliense Meurs.	Madhya Pradesh	Haware and Joshi (1974a)
P. myriotylum Drech.	Poona	Uppal (1940)
-do-	Bombay	Patel *et al.* (1949)
-do-	Nagpur	Sahare and Asthana (1968)
-do-	Kerala	Dake and Edison (1989)
P. pleroticum T.	Solan (Himachal Pradesh)	Sharma and Dohroo (1980)
P. vexans de Bary	Malabar (Wynad) (Kerala)	Ramakrishnan (1949)
P. ultimum	Himachal Pradesh	Dohroo *et al.* (1987)
Fusarium solani	Karnataka	Kumar (1977)
F. oxysporum f. sp. *zingiberi*	Madhya Pradesh	Haware and Joshi (1973b)
-do-	Rajasthan	Dohroo *et al.* (1988)
P. myriotylum and *F. solani*	Rajasthan	Mathur *et al.* (1984)
-do-	Rajasthan	Donee (1986)
P. solanacearum	Madras	Thomas (1941)
P. solanacearum Hayward Biotype III.	Kerala	Sarma *et al.* (1978)

2. RHIZOME ROT OF GINGER (*ZINGIBER OFFICINALE* ROSC.)

2.1 Soft Rot

The disease is caused by various *Pythium* spp. and prevalent in all states of India where ginger is grown and the problem still eludes solution. However, the *Pythium* spp. involved in different geographical regions appear to be different and are often involved with other biotic agents.

2.1.1 *Crop Losses*

Precise crop loss figures due to this disease are lacking. Crop losses vary from place to place. Approximately, losses to the tune of more than 50 and 80 per cent have

been reported (Butler, 1918; Joshi and Sharma, 1982). If the disease occurs in early stages of crop growth, it would result in total loss of rhizomes of affected clump and partial, if affected at late stages of crop growth.

2.1.2 *Symptoms*

Pythium spp. often infect immature and undifferentiated parts of the host plant. Both pre-emergence and post-emergence rhizome rot are noticed. Roots, rhizomes, emerging sprouts and pseudostems are all prone to infection depending on the stage of their maturity. Recent studies carried out at National Research Centre for Spices (NRCS), Calicut have shown that, apart from infection of young sprouts, pseudostem and rhizomes, root infection is equally important. About forty to fifty days after sowing, the emerging sprouts and also the plants in early stages of crop growth (up to four months) are infected. The emergence of fresh tillers being gradual, with the availability of tender suscept tissues, the infection continues upto three to four months after sowing.

When the undifferentiated sprout is infected, water soaked lesions appear at the emerging base which gradually enlarge, and the whole sprout turns pale yellowish and withers off leading to total death. Subsequent sprouts emerging from the same rhizome are often infected. Later infection spreads down to rhizome. In well differentiated clumps, infection starts at the collar region of pseudostem as water soaked area which generally spreads both upwards and downwards. Foliar yellowing is clearly seen starting from the margins from the lower most leaf and progresses upwards. As the infection progresses gradually to the inner region of pseudostem, intensity of foliar yellowing increases. Later, the infection spreads to the rhizome. The affected tissues rot, emiting foul smell and the pseudostems come off with a gentle pull. In early stages, the root infection often reaches the germinating sprouts leading to the rhizome rot. In a recent study carried out to study the role of root infection in soft rot of ginger, it was found that pseudostem infection was more when inoculum was placed at the surface. It decreased with increase in depth of inoculum source (Anandaraj and Sarma, unpublished). In a mature plant, it results in foliar yellowing and stunted growth of the plants (Fig. 2).

2.1.3 *Causal Organism*

About six *Pythium* spp., viz., *P. aphanidermatum* Edson Fitz (Mitra and Subramanian, 1928; Vaheduddin, 1955; Sahare and Asthana, 1968; Haware and Joshi, 1974a; Sarma *et al.*, 1979). *P. butleri* Subram (Thomas, 1938) (reported as syonymous with *P. aphanidermatum*) (Butler and Bisby, 1931), *P. deliense* Meurs (Haware and Joshi, 1974a) *P. gracile* (de Bary), Schrenk (Butler, 1907) and Sen (1930) reported synonymous with *P. aphanidermatum* (Butler and Bisby, 1931), *P. myriotylum* Drechsler (Uppal, 1940; Patel *et al.*, 1949; Sahare and Asthana, 1968), *P. pleroticum* (Dohroo and Sharma, 1985). *P. ultimum* (Dohroo *et al.*, 1987) and *P. vexans* de Bary (Ramakrishnan, 1949) have been reported to cause soft rot in different parts of India. The most commonly encountered are *P. aphanidermatum* and *P. myriotylum*.

2.1.4 *Epidemiology*

The disease is both seed-borne (McRae, 1911: Thomas, 1938; Mundkar, 1949) and soil-borne. In Kerala, which contributes a major share of ginger production in the country, where the crop is rainfed, the disease occurs during July-September, coinciding with south-west monsoon. High soil moisture and low temperature (25-28°C) prevailing during this period are highly conducive for the disease development. Disease incidence is more under ill-drained conditions. Once disease starts, it spreads gradually to the adjacent clumps mostly through soil water, both under rainfed and irrigated conditions. In general, the disease is less in hilly slopes probably because of better drainage.

In a comparative study, ginger planted both in ridge method and flat bed method, it was found that once disease starts, it spreads irrespective of the method of planting (Anon., 1979). The disease, though known as seed-borne, critical studies are lacking whether it is externally seed-borne or internally seed-borne and their contribution to disease incidence. The seed recovery from the stored, apparently healthy seed rhizomes vary. Recovery of 60 per cent from apparently healthy rhizomes decreased to 18 per cent when the original seed lot was mixed with 10 per cent of infected rhizomes (Anon., 1984). The nature of survival of the fungus in the seed rhizomes is not clear. However, scales harbouring the oospores in seed rhizomes has been reported (Thomas, 1938). *Pythium* sp. being soil dweller, is known to perennate in the soil in the form of oospores.

The positive isolation of *Pythium* from sick soils even after one year's fallow period indicated its long perennation (Anon., 1984). Being a soil-borne pathogen, continuous cultivation of ginger would result in soil inoculum build up in a given locality and shifting the site for cultivation/crop rotation as practised in Kerala would result in less disease incidence. When shifting cultivation is adopted, infected/contaminated seed rhizomes would serve as the primary source of inoculum.

The involvement of the pathogen in a given locality might be due to its adaptability to a agroclimatic situation. *Pythium vexans* reported from Wynad, Kerala, a high altitude (1170 m) region, had optimum temperature requirement of 28°C and did not grow beyond 34°C in contrast to *P. aphanidermatum* and *P. myriotylum*, where the optimum temperature for growth was 34°C and maximum being more than 40°C (Middleton, 1943; Ramakrishnan, 1949). Involvement of *P. myriotylum* and *F. solani* in rhizome rot of ginger is reported from Rajasthan (Dorjee, 1986). Similarly, *Pythium* sp. and *F. solani* have been reported causing rhizome rot in Gujarat (Chauhan and Patel, 1990). In a pilot survey carried out in Kerala during 1984 and 1985, incidence of soft rot caused by both *P. aphanidermatum* and *P. myriotylum* has been reported in addition to *F. oxysporum, F. solani* and *P. solanacearum* (Dake and Edison, 1988, 1989). The problem becomes more complex in combined infections, involving both fungus and bacterium.

2.1.5 *Role of Associated Organisms*

Detailed investigation on the role of consistent association of dipteran maggots *Mimegralla coeruleifrons* and *Eumerus* sp. with infected rhizomes revealed no role for

them in disease etiology and were considered as saprophytic colonisers, since they were not found to infect healthy rhizomes (Koya, 1988; Premkumar *et al.*, 1982; Radke and Borle, 1984). However, isolation of *Pythium* sp. from the foreguts of the field collected adult flies of *M. coeruleifrons* suggested their possible role in disease dissemination (Iyer *et al.*, 1981). However, this requires further studies.

An association of plant parasitic nematodes viz., *Meloidogyne incognita* (Nadakka and Thomas, 1964) *Pratylenchus coffeae* and their role in rhizome rot needs investigations. Increased rhizome rot incidence was reported in association with nematodes (Dohroo *et al.*, 1987). In the rhizome rot complex of ginger in Himachal Pradesh, *P. ultimum*, *F. equeseti* (Corda), *M. incognita* and *P. coffeae* have been implicated (Dohroo *et al.*, 1987). In a study on interaction of *P. myriotylum* with *M. incognita*, fungal antagonism to nematodes was observed (Lanjewar and Shukla, 1985; Aruna Parihar Yadav, 1987).

2.1.6 *Disease Management*

The disease, being both seed and soil-borne, the problem of disease management involves measures that effectively suppress/reduce the disease incidence through cultural, biological and chemical control methods (Dake *et al.*, 1988). Disease resistance in the available germplasm types in India at present is very low or absent.

2.1.6.1 *Cultural* : *Seed selection and storage*—Selection of healthy seeds from the disease free gardens and storing them in ideal condition is an essential pre-requisite. Seed storage practice consisting of seed treatment with Dithane M-45 (0.3%) solution for 30 min, air drying, storing in pits lined with sand, in thatched sheds or rooms where the temperature do not exceed 28-30°C ensured optimum recovery (Dake *et al.*, 1989; Mishra and Iyer, 1981). Precise methods of detection of seed-borne inoculum are yet to be standardised.

Seed treatment with aerated steam for 1 h at 40°C gave 91 per cent germination but, with increase in temperature, there was a drastic decrease in germination (Anon., 1988). Further studies are warranted to utilise the aerated steam for seed disinfection. Tissue culture methods of multiplication of ginger available at present (Kulkarni *et al.*, 1987) should be utilised to generate disease-free seed rhizomes.

2.1.6.2 *Time of sowing* : Young sprouts being highly susceptible, conducive environmental conditions like high soil moisture and low temperature would result in high disease incidence during July-August. By early sowing, the pseudostem will become hardy and less vulnerable to infection during critical periods (July-August) of infection. Studies carried at Calicut in Kerala showed that by early planting during May or early June resulted in less disease incidence (Anon., 1984). However, the period of sowing would vary for different agro-climatic zones.

2.1.6.3 *Drainage* : Better drainage would reduce the chances of infection, since stagnation leads to high inoculum build up and also rapid spread of the disease through soil water.

2.1.6.4 *Crop rotation* : This is another method practised to reduce the inoculum build up of soil-borne pathogens and is practised in Kerala. Detailed studies on this are warranted to suggest a suitable crop rotation that reduces the inoculum build up and consequent disease incidence.

2.1.6.5 *Soil management*: Soil amendments with neem cake at 2 t/ha reduced the disease incidence by 2 per cent, increased yield by 1.78 tonnes and also increased availability of nutrients viz., organic carbon, phosphorus, potassium and calcium. Both pongamia and neem cake as soil amendments showed reduced disease incidence compared to FYM treated plots (Sadanandan and Iyer, 1986). Disease incidence was highest (26.3%) in plots treated with lime + fertilizer compared to lime and fertilizer application alone and check plots with disease incidence of 20.3, 12.8 and 7.7 per cent, respectively (Sadanandan *et al.*, 1988). In another study involving surface soil burning and non-burning before planting, the former showed decreased disease incidence, increased yield and availability of nutrients (Sadanandan *et al.*, 1988). Soil amendment with woodsaw dust reduced rhizome rot incidence (Dataram, 1988). Soil amendment with oil cakes, viz., castor, groundnut, sesamum, margosa and coconut and also saw dust with and without urea tried for their effects on soft rot incidence in a *Pythium* sick plots showed that coconut cake was the best amendment that reduced the disease and increased the yields (Rajan and Singh, 1973). Application of soil amendments is known to improve the structure of soil and increase the microbiological population suppressive to pathogen.

Soil solarisation of *Pythium* sick plot prior to planting suppressed weed growth, improved the germination and yield and reduced disease incidence (Anon., 1990). Further studies are warranted for its feasibility for large scale application (Figs. 1 2 & 3).

2.1.6.6 *Chemical control* : This consisted of seed treatment with fungicides to check the seed borne inoculum and soil drench to check the soil borne inoculum. The sensitivity of the pathogen propagules to the concentration, quantity and time of exposure to the fungicide decides the efficacy of treatment. When the inoculum is internally seed borne, use of contact fungicide to eliminate the pathogens will be of little use. However, critical studies are warranted on the nature of seed borne inoculum in ginger.

Several contact fungicides used both as a seed treatment and soil drenches showed variable disease control which reflected on reduced pre- and post-emergence rot and increased yields. The variability of disease suppression might be due to inoculum levels of the plot, the frequency of application of fungicide in relation to inoculum potential, rate of degradation/leaching-off of the fungicide depending on amounts of rain fall and above all the seed borne and soil borne nature of the disease.

Seed treatment with 0.25 per cent Ceresan for 30 minutes was found effective in checking rhizome rot (Thomas, 1940). Seed treatment and soil drenching with Bordeaux mixture (2:2:50) was reported effective (Bhagawat, 1961). Pre-sowing soil drenching with Bordeaux mixture (4:4:50), 0.35 per cent prenox and Dithane D-78 and subsequently at three weeks intervals reduced rhizome rot caused by *P. aphanidermatum* and *P.*

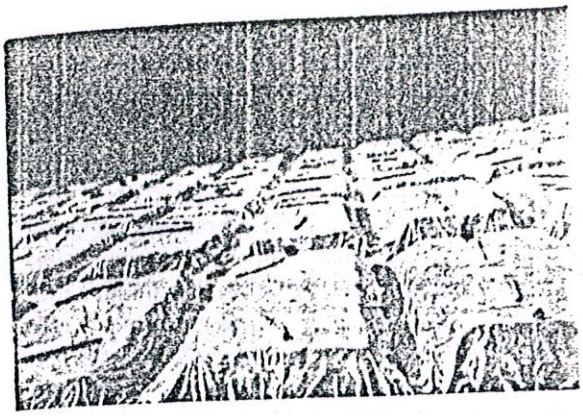

Fig. 1 : Soil solarisation

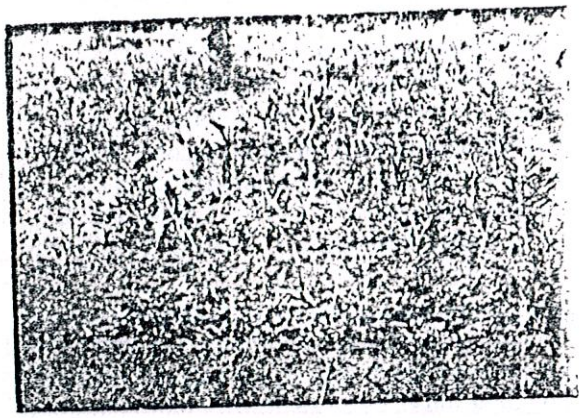

Fig. 2 : Disease in non-solarised plots

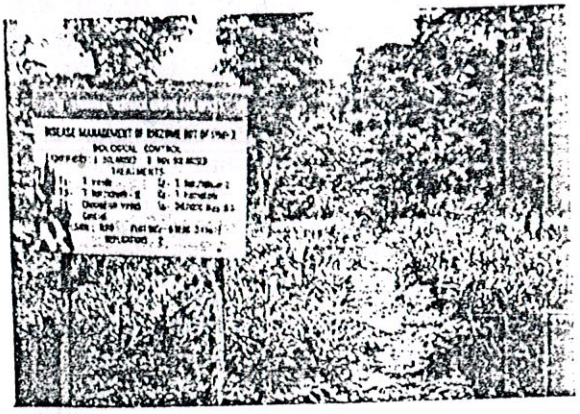

Fig. 3 : Crop-stand in solarised plots treated with biocontrol agent *Trichoderma harzianum*.

myriotylum (Sahare and Asthana, 1962). Seed dip in 0.2 per cent solution of Dithane M-45 and daconil was found effective in reducing rhizome rot caused by *P. pleroticum* (Sharma and Dohroo, 1982). In a comparative study of ridge method and raised bed method of planting, with and without dolomite application, superimposed by seed treatment, and soil drenching with Aureofungin, Dithane M-45, captafol and methoxy ethyl mercuric chloride (MEMC), it was found that only fungicidal treatments were significantly superior over control and MEMC was found to be the best (Sarma *et al.*, 1979). In a pot culture study, seed treatment and soil drenching with dexon was found to be effective in controlling soft rot of ginger (Sarma *et al.*, 1979). Studies carried out over several years at Central Plantation Crops Research Institute, Kasaragod and National Research Centre for Spices, Calicut showed greater consistency of captafol and Dithane M-45 as seed treatment and as a soil drench in controlling soft rot caused by *P. aphanidermatum*. Efficacy of Dithane M-45 in checking rhizome rot was further established (Koshy *et al.*, 1988b). Seed dip with 0.1 per cent Bavistin was found effective in controlling rhizome rot caused by *Pythium* and *Fusarium* (Mathur *et al.*, 1984).

The efficacy of seed treatment with captafol in reducing the post-emergence rot and increased yield was reported (Koshy Abraham *et al*, 1988a; Mathur *et al.*, 1984). In a recent study to check rhizome rot and bacterial wilt, seed treatments with captan, captafol, Dithane M-45 and hot water treatment at 42°C for 30 minutes was superimposed by 200 ppm streptocycline were evaluated. All the treatments significantly reduced pre-emergence rot. However, captan was found more effective. It did not have any depressive effect on bacterial wilt (Manmohandas *et al.*, 1990).

Systemic fungicides specific to oomycetes might be helpful even to check internally seed borne infection. Five systemic fungicides, viz., fosetyl Al, metalaxyl, oxadixyl, propamocarb and ethazole were evaluated against rhizome rot caused by *P. aphanidermatum* both as seed treatments and soil drenches. Metalaxyl formulations viz., Ridomil 5 G granules and Apron 35 WS gave the best control (Ramachandran *et al.*, 1989b). The *in vitro* sensitivity of the fungicides tested showed LD 90 values as 934.10, 9.5, 179.6, 305.56 and 1.14 µg/ml respectively (Ramachandran *et al.*, 1989a). In a comparative study with six non-systemic and four systemic fungicides as seed treatments tested for their effects on rhizome rot caused by *P. aphanidermatum*, Dithane M-45, difolaton (captafol), ziride, captan and metalaxyl reduced infection, increased germination and yield (Thakore *et al.*, 1988). Efficacy of metalaxyl in checking soft rot caused by *P. myriotylum* has been reported (Rathaiah, 1987).

An integrated disease management trial involving cultural practices, viz., burning and non-burning of the surface soil, early and late planting as main plots, application of organics, viz., FYM and neem cake and metacid as sub-plots and four fungicides as soil drenches as sub-sub-plots, it was found that burning the soil surface, early planting (1st May) together with metacid and Bordeaux mixture gave lower soft rot and increased yields. However, they were not statistically significant (Sadanandan *et al.*, 1988).

2.1.6.7 *Biocontrol* : The studies on biocontrol of rhizome rot dates back to studies of Thomas (1939) wherein he suggested the use of *Trichoderma lignorum* against *Pythium* in the dual cultures, since the former increased the acidity of the medium which was unfavourable to the growth of *Pythium*. The biocontrol potential of *Trichoderma* in rhizome rot of ginger is receiving attention in recent times (Bharadwaj and Gupta, 1987; Bharadwaj *et al.*, 1988). *T. viridae* in various combinations with wood saw dust, showed appreciable reduction in rhizome rot incidence (Dataram, 1989). In a comparative study of biocontrol efficacy of *T. viridae* and *T. harzianum* alone and in combination with Ridomil MZ 72 WP, it was found that *T. viridae* was superior in disease suppresion and was more effective in combination with Ridomil MZ 72 WP indicating its compatibility (Sarma *et al.*, unpublished). The delivery system of these biocontrol agents for large scale field application are yet to be standardised. *P. acanthophoron*, a hyperparasite to *P. myriotylum* and *Fusarium solani* (Lodha and Webster, 1990) reported recently, opens up new possibility of biocontrol of rhizome rot ginger. Isolation of this fungus was also reported from Kerala (Anon., 1985). Efficacy of *T. viridae*, *T. harzianum* and *P. acanthophoron* along with wood raw dust in supperessing rhizome rot under the field conditions was established at Udaipur, Rajasthan. Similarly at Calicut, Kerala *T. harzianum* and *T. namatum* were found effective in suppressing rhizome rot (Anon., 1993). The association of *Glomus* spp. and other VAM (vesicular arbuscular mycorrhizae) with root system and their suppressive effects on rhizome rot need indepth study.

2.1.6.8 *Disease resistance* : None of the available cultivars seems to have high degree of resistance even though variety Maran (Indrasenan and Pailly, 1974) and Nadia and Narasapattom (Balagopal *et al.*, 1975) have been reported as tolerant and moderately resistant. Of the 33 cultivars consisting of both indigenous and exotic, screened in the field and also under artificial inoculation conditions showed variability and inconsistent reaction during three years period. None of them were found resistant (Sarma *et al.*, 1979). Isolation of callus cultures of ginger insensitive to culture filtrate of *P. aphanidermatum* have been regenerated (Kulkarni *et al.*, 1987) and are being evaluated for their field reaction. Biotechnological approaches in inducing disease resistance through somaclonal variation in ginger has been stressed (Sarma and Ramadasan, 1990). Toxins of *P. aphanidermatum* have been reported to be glycoproteins (Rao, 1986) and need to be utilised for *in vitro* screening of callus and cell cultures to locate toxin insensitive cultures which can further be tested for disease resistance

2.2 Yellows Disease

The disease was first reported from Madhya Pradesh (Agarwal, 1972; Haware and Joshi, 1973) and later from Himachal Pradesh (Donroo *et al.*, 1988) and also from Kerala (Dake and Edison, 1988, 1989). In Himachal Pradesh, this is a major limiting factor in ginger production.

2.2.1 *Crop Losses*

Systematic crop loss estimates are lacking for this disease. In Kerala, a pilot survey for disease incidence showed that out of 195 gardens visited, 8.84 per cent gave

positive *Fusarium* sp. isolation (which includes *F. oxysporum*) (Dake and Edison, 1988, 1989). During 1984-87, about 40 per cent losses have been reported from Shillai, Rajgarh, Ronhat and Sirmur areas of Himachal Pradesh (Dohroo *et al.*, 1988). Ginger production of 1,27,000 tonnes as dry ginger from 52,460 ha in Himachal Pradesh dwindled to 850 tonnes from 2,360 ha during 1984-87 due to severe disease incidence (Venkataramani, 1989).

2.2.2 *Symptoms*

The infected plants exhibit foliar yellowing which starts from the lower leaves and progress upwards. The affected plants wilt and dry up but do not fall on the ground in contrast to soft rot and bacterial wilt affected ginger. Affected rhizomes become soft and watery. The pseudostem comes off from mother rhizome with a gentle pull. Affected rhizomes show a creamy discolouration of the vascular system and cortical rot. The infected plants appear stunted with root rot and rhizome formation is affected. They show varying degrees of decay.

2.2.3 *Causal Organism*

Fusarium oxysporum f. sp. *zingiberi* is the causal agent identified (Haware and Joshi, 1973b). Independent infection by *F. solani* (Kumar, 1977) and also combined infections of *F. solani* and *Pythium* spp. have been reported (Dorjee, 1986, Chauhan and Patel, 1990).

2.2.4 *Epidemiology*

The disease occurs in patches with plants with yellows symptom surrounded by green plants. The disease is soil and seed borne and secondary spread is through soil water. The infected but apparently normal rhizomes serve as the primary source of inoculum and chlamydospores serve as the perennating propagules (Sharma and Jain, 1978b).

Disease development was found favourable at a temperature range of 15-38°C. The infection increases at optimum temperatures of 23-29°C with a high relative humidity of 87-95 per cent and presence of thin film of water (Sharma and Jain, 1978a). Two months old ginger plants inoculated with *F. oxysporum* showed vascular discolouration in 25-30 days after inoculation followed by the presence of hyphae in the vessels which later spread to the cortical regions with inter- and intra-cellular hyphae (Haware and Joshi, 1973b).

Combined infections of *F. oxysporum*, *M. incognita* and *Pratylenchus coffeae* have been reported from Solan, Himachal Pradesh (Anon., 1993). Extracts from the root-knot affected ginger roots incorporated into the medium increased growth of the fungus (Agarwal *et al.*, 1972, 1974). This indicated the possibility of greater susceptibility of ginger infected by *M. incognita* to *F. oxysporum*. Soils from rhizosphere of diseased

ginger showed lesser suppression of the fungus compared to the rhizosphere soils of healthy ginger (Sharma, 1977). *Fusarium* being a soil dweller, it perennates in the soil for several years which renders disease control less effective.

2.2.5 *Cultural Control*

Being a soil-borne pathogen, raising ginger crop in the same field every year should be avoided to reduce the chances of inoculum build up (Sharma and Jain, 1978a).

2.2.6 *Biocontrol*

Biocontrol of *F. oxysporum* with *Bacillus subtilis*, *Memnoniella echinata* and *Asperigillus niger* was found effective in pot culture. *B. subtilis*, Strain II reduced disease incidence from 58.3 to 8.3 per cent and increased yield (Sharma and Jain, 1978b). Isolation and field testing of antagonists and hyperparasites of *F. oxysporum* and their field evaluation need to be intensified. Application of organic amendments like saw dust, oil cakes, maize meal and dried leaves of different plants species to wilt sick soils did not have any disease suppressive effects (Sharma and Jain 1978b). Suppression of plant parasitic nematodes and Fusarium yellows has been reported with soil amends of neem cake and pine needles. These two amendments increased native *Trichoderma* and *Gliocladium* population in Himachal Pradesh (Anon., 1993)

2.2.7 *Chemical Control*

Soaking the seed rhizomes in the fungicide solution/suspension of Dithane M-45 (0.3%) or benalate for two hours followed by two soil drenchings (6 l/sq m at 0.3%), one at the time of sowing and the second by 15th day of sowing was recommended (Haware and Joshi, 1974b; Sharma and Jain, 1978a). Seed dip with bavistin (0.1%), Dithane M-45 (0.3%) + bavistin and bavistin + Dithane M-45 + sumithion (0.1%) for 1 h were found to be superior to other treatments in disease suppression. However, seed treatment with bavistin (0.1%) was found to be cost effective (Dohroo *et al.*, 1988).

2.2.8 *Disease Resistance*

Published information on disease resistance to yellows disease is not available. Standardisation of a screening technique to identify resistance has been reported (Sharma and Dohroo, 1989) and there is an urgent need to test all the available ginger germplasm in India for their reaction to yellows disease.

The foregoing account brings out the current status of the yellows disease. The disease management strategies would be on the similar lines of the soft rot and bacterial wilt of ginger and calls for an intensification of investigations on disease resistance, biocontrol, crop rotation and the feasibility of soil solarisation on the disease suppression.

Apart from these three major disease problems causing rhizome rot of standing ginger crop, basal rot caused by *Sclerotium rolfsii* (Haware *et al.*, 1973a) and dry rot

caused by *Macrophomina phaseolina* (Sarma and Nambiar, 1974) have also been reported, which need close watch in future.

2.3 Bacterial Wilt

The disease is becoming increasingly serious particularly in Wynad district of Kerala. The disease was first reported from Madras state (Thomas, 1941). Later, it was reported from Kerala (Sarma *et al.*, 1978; James Mathew *et al.*, 1979), Bihar (Ojha *et al.*, 1985) and Himachal Pradesh (Dohroo, 1991).

2.3.1 *Crop Losses*

Precise crop loss figures are not available. Once disease ciled, the spread would be fast and incidence as high as (50-60 per cent) was recorded resulting in severe loss of affected clumps.

2.3.2 *Symptoms*

The earliest symptoms appear as water soaked linear streaks on the collar region of the pseudostems followed by yellow to bronze colouration of the margins of the lower most leaves which gradually progresses upwards. Later, the leaves become flaccid exhibiting wilt symptoms and intense foliar yellowing. The affected pseudostems at the base would be slimy to touch and come off with a gentle pull. A small piece of the affected tissue kept in water gives off milky white bacterial oozes. The affected plants droop and dry.

2.3.3 *Causal Organism*

Pseudomonas solanacearum (Smith) Smith, Biotype III of Hayward, has been identified as the causal agent of the disease (Sarma *et al.*, 1978). Biotype iv also has been isolated and identified from Kerala (Anon., 1994).

2.3.4 *Epidemiology*

The disease is typically soil and seed borne. It is generally noticed during June-July and would be maximum during August-September coinciding with south-west monsoon ensuring high soil moisture and relative humidity, and low temperature (Sarma *et al.*, 1978). The disease spread is along the gradient and spreads through soil water. Water splashes might result in spread against the gradient also (Dake *et al.*, 1988). Patnogenicity tests conducted showed that infected soil as well as affected rhizomes could induce the disease (Sarma *et al.*, 1978) indicating their potential as inoculum source under field conditions. Mixed infection of bacterial wilt and soft rot (*Pythium* spp.) have been reported (Dake and Edison, 1989) and would be more complex for disease control. The pathogen survived for two seasons in soil under natural conditions (Indrasenan *et al.*, 1981). *M. incognita* infestation two weeks earlier to bacterial inoculation increased

wilt incidence indicating its ability to predispose ginger plants to bacterial wilt (Samuel and Mathew, 1983).

Association of *M. coeruleifrons* was noticed occasionally. *P. solanacearum* was isolated from field collected maggots from infected rhizomes and also release of such maggots could induce the disease in potted ginger plants (Premkumar *et al.*, 1982). Their role in disease spread under field conditions needs investigations. Detailed studies on epidemiology are warranted.

2.3.5 *Disease Management*

In general, disease management practices are similar to rhizome rot of ginger and turmeric.

Since the disease being a seed-borne, selection of healthy seed becomes imperative. Provision of good drainage and removal of infected plants from the garden immediately, in early stages, would check the disease spread. *Eupatorium odoratum* and *Ageratum conizoides* were found infected with *P. solanacearum* in bacterial wilt affected ginger plot (Anon., 1989). This indicated that they might serve as collateral hosts of bacterial wilt pathogen.

2.3.6 *Chemical Control*

Soil drenching with agrimycin (1000 ppm), streptocycline (1000 ppm), 1 per cent Bordeaux mixture and application of bleaching powder did not check the disease once disease appeared (Sarma *et al.*, 1978). However, seed treatment with 200 ppm stretptocycline prior to planting delayed the disease development but could not check the disease (Anon., 1988). Emisan 6 (an orangomercurial fungicide) + plantomycin (0.05%) as seed treatment for 30 minutes and a plantomycin spray after 30 days and followed by two sprays at 15 days intervals proved to be most effective. Plantomycin alone and in combination with blitox was equally effective (Ojha *et al.*, 1986). In view of the predisposal of *M. incognita* infection of ginger to bacterial wilt, it becomes essential to check nematode infections with suitable nematicide where such combined infections are noticed. Beds treated with bleaching powder (25 g/3 × 1 m bed) sown with seed rhizomes treated with streptocycline at 200, 500 and 1000 ppm for 30 minutes did not check wilt (Anon., 1989).

2.3.7 *Disease Resistance*

Of the 30 types of ginger evaluated under field conditions, none found resistant. However, they showed variable reaction (Indrasenan *et al.*, 1982).

Early detection of seed contamination through serological techniques, biocontrol methods using *P. flourescens* and other antagonists and inducing disease resistance through somaclonal variation would be priorities since chemical control would be impractical.

A water soluble glycopeptide with two fractions has been reported from *P. solanacearum* (Gowda and Rai, 1980) and this could be utilised for *in vitro* screening of callus and cell culture to identify resistant or toxin insensitive cell lines.

2.4 Storage Rots

Storage of ginger becomes important both for seed purpose and also for their use throughout the year. Storage conditions influence considerably the deterioration/ infection of rhizomes in addition to the already existing seed-borne contaminations either externally or internally. Storage conditions vary from place to place. Rhizomes stored in a mud plastered pit at 20°C showed minimum rotting (Sharma and Joshi, 1979). The storage losses due to deterioration caused by storage fungi, mostly saprophytes, are substantial. Contaminated seed rhizomes serve as the primary source of inoculum and subsequent crop losses. Seed-borne infection to the tune of 87 per cent has been reported (Dohroo, 1989).

Some of the organisms associated with storage rot are *Acremonium kiliense*, *A. strictum*, *Alternaria alternata*, *Aspergillus flavus*, *A. niger*, *Memnoniella echinata*, *Pseudospapuiospora kendrickii* (Sharma and Jain, 1977b), *Geotrichum* sp. (Mishra and Rath, 1988), *F. solani* and *F. moniliforme* (Rath *et al.*, 1978), *F. roseum*, *Sclerotium rolfsii* (Mehrotra, 1952), *Pythium pleroticum* and *F. equeseti* (Sharma and Dohroo, 1982), and also a bacterium (Anon., 1974). Of the 29 fungi reported, the association with ginger in storage, four fungi, viz., *F. oxysporum*, *P. deliense*, *P. myriotylum* and *Pseudospapulospora kendrickii* were found internally in the rhizomes (Sharma and Jain, 1977b) causing deterioration. Incidentally, both *F. oxysporum* and *P. myriotylum* are known pathogens of ginger.

Storage rots are referred to by names depending on the colour and symptoms they produce. They are red rot caused by *Nectria inventa* (Sharma and Joshi, 1976a), black rot caused by *Memnoniella echinata* (Sharma and Joshi, 1976b), grey rot caused by *Trichurus spiralis* (Sharma and Joshi, 1976b), dry rot caused by *Diplodia natalensis* (Wilson and Balagopal, 1971) and *Macrophomina phaseolina* (Sarma and Nambiar, 1974).

Seed treatments with fungicides has been reported to reduce the storage losses. Seed treatment with Dithane M-45 (0.3%) for 30 minutes and storing rhizomes in pots lined with sand in the low thatched sheds at 28-30°C gave good recovery of the rhizome stored for about five months (Dake *et al.*, 1989). Seed treatment with aureofungin (0.02%) and benelate (0.2%) were found effective (Haware *et al.*, 1973). Kitazin (0.1%), bavistin (0.3%) and Dithane M-45 (0.3%) were found effective in controlling storage rot (Sharma and Jain, 1978a). Seed treatment with carbendazim was found effective because of its persistence for a long time (Arora *et al.*, 1977). Emisan 6, Blitox 50 and mercuric chloride as seed treatment for 30 minutes were effective in controlling storage rot caused by *P. plerocticum*. For rot caused by *F. equeseti*, Dithane M-45, Emisan 6 and blitox were found effective (Sharma and Dohroo, 1982). Antracol (0.25%), fycop (0.3%) and Blitox

50 (0.3%) as seed dip for 30 minutes were found effective in controlling storage rots (Dohroo and Sharma, 1983).

Thus seed storage and fungicidal treatments to reduce the storage losses and to minimise/eliminate the seed-borne infections are of greater relevance for ginger.

3. RHIZOME ROT OF TURMERIC (*CURCUMA LONGA*)

3.1 Rhizome Rot

Both *C. longa* and *C. aromatica* are generally referred to as turmeric and are affected by rhizome rot disease.

Rhizome rot of turmeric is similar to soft rot of ginger in all respects causing partial or total loss of the rhizome (Figs. 4 & 5). The disease was first reported from South India viz., Krishna district of Andhra Pradesh, Tiruchirapally and Coimbatore (Tamil Nadu) of erstwhile Madras State (Ramakrishnan and Soumini, 1954). This is also known from Telangana areas of Andhra Pradesh (Rao and Rao, 1988) the etiology of which is yet to be determined. It is also known from Kasaragod area of Kerala (Anon., 1975) and Assam region (Rathaiah, 1982b).

3.1.1 *Crop Losses*

Precise crop loss figures, though not available, losses to the tune of 50 per cent and above have been reported in some parts of Telangana and farmers resort to distress harvest to salvage the remaining crop once the disease starts appearing (Anon., 1988).

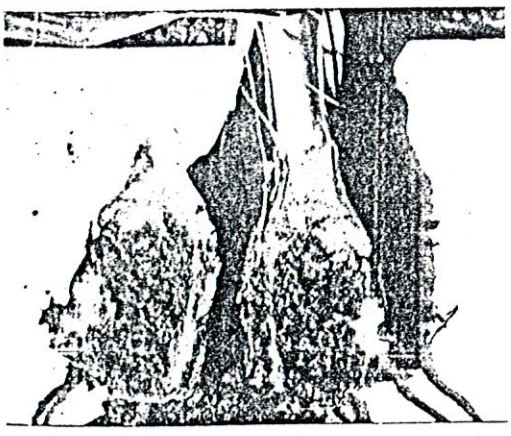

Fig. 4 : Rhizome rot in Mother rhizomes.

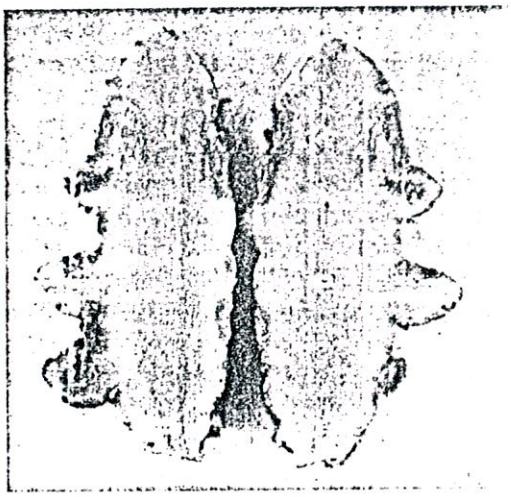

Fig. 5 : Rhizome rot in finger rhizomes.

3.1.2 *Symptoms*

The infected plants show yellowing of leaves starting from lower leaves which gradually spread to the upper regions of the plant. The margins of the yellowing leaves turn necrotic and start drying from the margins inwards resulting in partial or complete blighting of leaves. Water soaked to dark brown lesions appear on the pseudostems at the base which enlarge rapidly resulting in drying up. In Assam region, the affected pseudostems break away with a pull and the affected tillers topple off. The affected plants show varying degrees of root rot. The infection spreads from roots to rhizome causing soft rot (Fig. 4). Infection is also noticed from tips of rhizomes spreading inwards. The affected plants become stunted with foliar yellowing. The affected rhizomes show varying degrees of brown shades in contrast to the bright orange colour of healthy turmeric. In advanced stages, the rotten rhizomes emit foul smell.

3.1.3 *Causal Organism*

Pythium graminicolum Subram has been reported as the causal agent in erstwhile Madras State, South India (Ramakrishnan and Soumini, 1954). *P. myriotylum* from Assam (Rathaiah, 1982b) and *Pythium* sp. from Kerala (Anon., 1975) have been reported as pathogens of rhizome rot of turmeric. Association of *Pythium* sp.and *Fusarium* sp. with rhizome rot in Telangana area has been reported (Anon., 1988).

3.1.4 *Epidemiology*

Detailed epidemiological studies have not been carried out and are warranted to plan effective control measures. Turmeric is grown both as a pure and also as an intercrop along with maize, red gram and chillies. The effect of crop combination on disease incidence and intensity needs investigation.

In Telangana area of Andhra Pradesh, this crop is grown in red sandy, loamy soil (Chalka), sandy soils and black cotton soils. The disease incidence was noticed in all types of soils wheather ill-drained or well drained (Rao and Rao, 1988).

The disease is soil-borne and seed-borne (Chattopadhyay, 1967; Rathaiah, 1982b), and occurs at random and spreads contiguously to adjacent clumps. In artificial inoculation studies, *P. graminicolum* could induce root rot in a week and death of two months old plants within 18 days. The fungus was also found pathogenic to seedlings of sorghum, maize, barley, oats, arrowroot and cotton, and could not infect ginger (Ramakrishnan and Soumini, 1954). In Telangana area, the disease is generally noticed when the crop is about three months old. Where it is intercropped with maize, the symptom expression would be sudden immediately after the maize harvest (Anon., 1988) and this might be due to sudden exposure of the crop from the partial shade. The root rot, likely depletion of soil moisture, physical disturbance and change in the microclimate might be the factors affecting the symptoms expression. Application of heavy doses of tank silt, compost and FYM, the consequent increased soil moisture levels and their effects on disease incidence need to be understood.

3.1.5 *Role of Associated Organisms*

In Telangana area of Andhra Pradesh, association of maggots of *M. coeruleifrons* with disease affected rhizomes was noticed to varying degrees. However their absence in diseased rhizomes was also noticed. As such, studies are warranted to understand the role of maggots in disease etiology. *M. coeruleifrons* has been reported as pest of turmeric in Kerala (Jacob 1986) and also in Maharashtra (Ghorpade *et al.*, 1983). In rhizome rot of ginger, maggots of *M. coeruleifrons* did not have any role in disease etiology (Koya, 1988). The association of *Colobata albimana* with rhizome rot of turmeric (Nair, 1978) also needs in depth study, though this has been reported as a pest by itself (Venkateswara Rao and Subbarami Reddy, 1988). Root-knot infestation in turmeric was noticed in Telangana where rhizome rot incidence is severe. The role of *M. incognita* on root infection by fungal pathogens needs investigation to plan an effective integrated disease management.

3.1.6 *Disease Management*

The problem being similar to soft rot of ginger, the strategies of disease management in general are similar.

3.1.6.1 *Seed selection and storage* : The disease being seed-borne selection of healthy seed from disease-free gardens becomes imperative since precise methods of detection of seed-borne inoculum are yet to be standardised. There is a need to test the efficacy of hot water and aerated steam on elimination of seed-borne inoculum. It is also essential to multiply the nucleus seed material through tissue culture (Kuruvina Shetty *et al.*, 1992) to avoid seed-borne inoculum.

3.1.6.2 *Phytosanitation* : Removal and burning of the infected clumps from the field and drenching the soil with cheshunt compound or ceresan wet (0.1%) was recommended to check this disease (Ramakrishnan and Soumini, 1954).

3.1.6.3 *Soil amendments* : Information on the effect of inorganic and organic amendments on the diseases incidence is lacking though majority of the farmers apply high doses of tank silt and FYM. Application of 125-250 kg of superphosphate did not have any effect on disease incidence. However, the survival of the fungus was affected by application of one per cent urea to the infested soil. Urea did have depressive effect on the fungal growth (Chattopadhyay, 1967) and future studies are needed to test its efficacy on the disease suppression.

3.1.6.4 *Crop rotation* : In Telangana, where turmeric is generally grown as intercrop with maize and chillies, a restricted crop rotation is followed and might help in reducing the build up of soil-borne inoculum. Biocontrol programmes as suggested for soft rot of ginger need to be undertaken.

3.1.6.5 *Chemical control* : Soil drenching with cheshunt compound or ceresan wet (1%) 5.5 l/sq.m. was suggested for the control of *P. graminicolum* (Ramakrishnan and Soumini,

1954 . Seed dip in ridomil (0.25%) for 40 minutes and soil drenching (0.01%) was found superior to ceresan wet treatment in the control of rhizome rot caused by *P. myriotylum* (Rathaiah, 1982a). The rhizome yield was 17.4 kg/plot (2 rows of 4 m each) in ridomil treated plots compared to 11.74 and 11.95 kg in ceresan treated and untreated control respectively, thereby indicating the superiority of ridomil.

3.1.6.6 *Disease resistance* : Cultivars Mydukur, Tekurpet and Duggirala were found susceptible whereas Ca-69 of *C. aromatica* and cultivar Shillong of *C. longa* were found resistant to *P. myriotylum* (Rathaiah, 1982). Newly released turmeric selections from National Research Centre for Spices, Calicut, viz., PCT 13, PCT 14 have been reported to be free from disease in Telangana areas (Rao and Rao, 1992) and their reaction to the pathogen should be tested by artificial inoculation. Seed set reported in turmeric should be exploited for variation for disease resistance in turmeric. Similarly, somaclonal variation through tissue culture need be induced for variation for disease resistance.

Compared to ginger, the investigations carried out on rhizome rot of turmeric are meagre. Detailed surveys on crop loss and indepth investigation on the etiology and epidemiology of the disease to develop suitable strategies of disease management are needed. Early detection of seed-borne inoculum, nature of survival of the pathogen, effect of soil solarisation, organic amendments crop rotation on the disease suppression need detailed investigations. Based on the causal agents involved, suitable fungicidal seed protection can be standardised. There is a need to check several germplasm materials now available at various centres for disease resistance.

3.2 Dry Rot

The disease is becoming increasingly important in Kerala. The disease causes root rot and rhizome rot resulting typical dry rot of rhizomes from October onwards. The affected rhizomes appear soft and shrunken to start with, later dry up and becomes hard. Foliar yellowing and drying up of foliage which are the normal symptoms of maturity of crop during October-November would be indistinguishable from the symptoms of the disease affected clumps. When infected rhizomes are cut open, the infected zones typically appear as dull brown and dark. *Rhizoctonia bataticola* is associated with the disease (Sarma, unpublished).

3.3 Brown Rot

This was first reported from Kerala (Sarma *et al.*, 1974) in *C. aromatica* and was noticed in freshly harvested rhizomes indicating its natural occurrence during the crop season. The disease affected plants were stunted with poor root development. The infected rhizomes appear dull coloured, later become deep grey to dark brown, less turgid, light and wrinkled exhibiting dry rot symptoms. The necrotic lesions in the rhizome start from margins and progress inwards involving a major portion of rhizome. *Pratylenchus* sp. and *Fusarium* sp., a nematode-fungal complex, could induce the disease.

3.4 Bacterial Wilt

In addition, bacterial wilt of turmeric caused by *Pseudomonas solanacearum* was also recorded (Sarma *et al.*, unpublished) on *C. longa* in Kerala during 1978 and subsequently. At present this is not a serious disease.

3.5 Storage Rots

Storage rot of turmeric caused by *Sclerotium rolfsii* was reported in Duggirala of Guntur district of Andhra Pradesh. Seed dip with bavistin was found effective in checking the rot of seed rhizomes and ensuring better germination (Reddy and Rao 1973). Ten fungi isolated from the diseased rhizomes collected from Delhi market were pathogenic with knife injury technique. *A. flavus*, *A. niger*, *M. phaseolina*, *P. aphanidermatum*, *S. rolfsii* were pathogenic even without injury. *F. oxysporum*, *C. cladosporioides*, *F. moniliforme* and *Drechslera rostrata* were pathogenic with pinprick except *R. solani* (Kumar and Roy, 1990).

Of the ten organisms associated with storage rot of turmeric, viz., *Aspergillus flavus*, *A. niger*, *A. tamarii*, *Cladosporium cladosporioides*, *Cephalosporium acremonium*, *Dreschlera tetramera*, *F. culmorum*, *F. nivale*, *F. oxysporum* and *M. phaseolina* all were found pathogenic with wound inoculation, whereas *M. phaseolina* and *C. cladosporioides* were more virulent and could infect without any injury (Sharma and Roy, 1984, 1986). In Kerala, storage rot of turmeric is associated with *R. bataticola* and *Fusarium* sp. *C. longa*, *C. aromatica* and *C. amada* were found often infected with *R. bataticola* (Sarma, unpublished). Studies are warranted on better storage condition and effective seed treatment that could reduce storage losses.

4. FUTURE PRIORITIES ON RHIZOME ROT OF GINGER AND TURMERIC

4.1 Etiology

Since the etiology of the disease in several places appear to be complex, detailed etiological studies should be undertaken to identify primary or major pathogens and also other biotic agents that accelerate disease development. This information is essential to evolve effective disease management strategies.

4.2 Disease Resistance

Evolving disease resistant clones against major pathogens of ginger and turmeric viz., *Pythium* spp. is of high priority. Absence of seed set in ginger and, to some extent, in turmeric limits variability of these crops. The tissue culture techniques available at present for the multiplication of these crops have to be exploited to induce somaclonal variation. To screen the callus or cell cultures for their insensitivity to toxic metabolites of the pathogens is the first step and should be followed by regeneration of plantlets to test the clones obtained, for their reaction with the actual pathogens. This becomes essential since none of the pathogens of these crops produce host-specific toxins.

Further, efforts should be to look for multiple disease resistance coupled with quality and productivity.

4.3 Seed Certification

Production of disease-free nucleus seed through tissue culture would be ideal. Until such time, precise detection of seed-borne inoculum should be evolved to select disease-free seed material so that seed certification could be introduced.

4.4 Seed Treatments

Ideal seed treatments with both systemic and protectant agro-chemicals need to be standardised. Efforts should be to look for agro-chemicals compatible with biocontrol agents so that chemical and biocontrol agents could be integrated.

4.5 Biocontrol

Isolation, identification of potential biocontrol agents including VAM that could effectively check the rhizome rot pathogens and developing suitable techniques for large scale multiplication should receive immediate attention. Besides identification of the suitable organic amendments that could reduce the pathogen population and boost up the vigour and productivity of the crop is a priority

4.6 Cultural Practices

Feasibility of utilising soil solarisation as a practice needs to be tested. Suitable agronomic practices to regulate the soil moisture levels and to identify ideal crop rotation systems that could suppress the disease need to be standardised, and also the biotic agents that accentuates the disease. The information is essential to evolve disease management strategies

A multipronged integrated disease management system involving phytosanitation, cultural and biological methods of control coupled with disease resistance would be the practical solution to check the crop losses of these crops.

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