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Diseases infecting ginger (*Zingiber officinale* Roscoe): A review

Gupta Meenu* and Manisha Kaushal[1]

Department of Vegetable Science,
Dr YS Parmar University of Horticulture and Forestry, Nauni, Solan-173230, India.

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ABSTRACT

Ginger (*Zingiber officinale* Roscoe) is an important spice crop in India, which is also one of the leading producer and exporter of ginger in the world. During cultivation, the crop is severely infected by various diseases of them soft rot, yellows, Phyllosticta leaf spot, storage rot, bacterial wilt, mosaic, chlorotic fleck are important. These diseases reduce the potential yields drastically. The geographical distribution, losses, symptoms, causal organism, disease cycle, epidemiology and host resistance, cultural, biological, chemical and integrated management of above mentioned diseses have been discussed in the present paper.

Key words: Bacterial wilt, *Phyllosticta* leaf spot, Soft rot, Storage rot, Viral diseases, Yellows.

Ginger (*Zingiber officinale* Roscoe) is an important spice crop in India, the leading producer and exporter of ginger in the world. The productivity of ginger in India is 40,903MT with annual income of Rs. 44.04 crores in the year 2010-2011 (Dohroo *et al*., 2012). In India, major ginger growing states are Kerala, Sikkim, Meghalaya, West Bengal, Orissa, Tamil Nadu, Karnataka, Andhra Pradesh, Maharashtra and Himachal Pradesh. During cultivation, the crop is severely affected by various diseases of fungal, bacterial and viral origin and reduce its potential yields drastically reduced.

1. SOFT ROT

In warm and humid conditions, it may assume serious proportions and cause significant losses. It is prevalent in almost all ginger growing areas of the world. The disease was first recorded during the year 1907 from Surat, Gujrat, India (Butler, 1907).

Symptoms: Ginger crop is affected by this disease throughout the growing period. Almost all parts of the plant including sprouts, roots, developing rhizome and collar region of the pseudostem are vulnerable to infection.

Symptoms of soft rot first appear on above ground parts at the collar region in the form of watery, brown lesions. These lesions then enlarge and coalesce, causing the stem to rot and collapse (Dohroo, 2005). On the leaves, the initial symptoms caused by the basal infection appear as yellowing of the tips of older leaves first with the chlorosis gradually moving downward along the margin involving the rest of the leaf blade and, eventually, the leaf sheath. As on older leaves progress, younger leaves start developing a similar symptom progression until the entire plant dies (ISPS, 2005). Once it happens, diseased stems can be easily dislodged because of little structural integrity between and rhizomes. Rhizomes from diseased plants appear brown, water soaked, soft and rotten, and will decay gradually (Dohroo, 2005).

Causal Organism: Several species of *Pythium* have been reported to cause soft rot disease in different parts of the world.

Pythium spp. are fungal-like microorganisms belonging to the family *Pythiaceae* in the order *Peronosporales* of the phylum *Oomycota*, a member of the kingdom Stramenopila (Webster and Weber, 2007).

The mycelium of *P. aphanidermatum* is colourless, sometimes lustrous and occasionally slightly yellowish due to abundant oospores or hyphal swellings or grayish lilac (Dohroo *et al*., 2012). The main hyphae are up to 10 µm wide. Sporangia consisting of terminal complexes of swollen hyphal branches of varying length and up to 20 µm wide. Oogonia terminal, globose, smooth, 20-25 µm in diameter. Anthredia mostly intercalary, sometimes, broadly sac shaped, 10-14 µm wide, 2/ oogonium, monoclinous or diclinous, oospores aplerotic (18-22 µm) in diameter, wall 1-2 µm thick. Zoospores are released either by a pore developed at the tip of sporangium or by bursting of vesicle. Sporangia are never detached from the hypha. The oospores are smooth walled, plerotic (oospore wall fused with oogonial wall) and spherical in shape measuring 12.0-20.0 µm in diameter (Dohroo, 1982).

For confirming the identity of the pathogen by molecular techniques, Wang *et al*. (2003) established a booster PCR method for detection of *P. myriotylum* using a specific primer selected from rDNA ITS1 region coupled with universal primer ITS2. It was successfully applied to

*Corresponding author's e-mail: meenugupta1@gmail.com
[1]Department of Food Science and Technology, Dr YS Parmar University of Horticulture and Forestry, Nauni, Solan (HP) 173230, India.

Table 1: Reports of *Pythium* spp. associated with ginger with identification based on morphological and/or molecular techniques and pathogenicity on ginger.

Species	Countries recorded	Species confirmed by
Pythium aphanidermatum (Edson) Fitz	Bangladesh	M, MT, P
	China	M, MT, P
	India	M, MT, P
	Japan	M, P
P. deliense Meurs	India	M, MT, P
P. graminicola Subram	Australia	M, P
	Fiji	M, MT
	Hawaii	M, P
	India	M, P
P. myriotylum Drechsler	Australia	M, MT, P
	Fiji	M, MT, P
	India	M, MT, P
	Korea	M, P
	Taiwan	M, P
P. spinosum Sawada	Australia	M, P
	Japan	M, P
P. splendens Braun	India	M, MT, P
	Malaysia	M, P
P. ultimum Trow	India	M, MT, P
	Japan	M, P
P. vexans de Bary	Fiji	M, MT
	India	M, MT, P
P. zingiberis Takahashi	Japan	M, P
	Korea	M, P

M: Morphology, MT: Molecular technique, P: Pathogenicity confirmed by Koch's postulates.
(Le *et al*., 2014)

the detection of *P. myriotylum* in naturally infected ginger rhizomes but not from DNA of ginger rhizomes collected from field without target fungus. A simple technique for producing oospores in *P. myriotylum*, causing soft rot of ginger has been demonstrated by Yella *et al.* (2006).

Disease cycle and epidemiology: There are two ways by which the disease is carried over and perpetuated, firstly through diseased rhizomes as oospores in scales (Thomas, 1938) and secondly through oospores in soil. *Pythium* species are capable of saprophytic survival in plant debris. The infected plant debris remaining in the field forms an important source of primary inoculum. Such plant parts may contain large number of perennating oospores.

The disease is both seed and soil borne. The wet soil conditions, high soil moisture and soil temperature are the most important factors influencing the development of this disease. Severity of disease is more in areas where rainfall is high or rhizomes are planted in heavy clay soil and poor drainage. The optimum temperature for germination of *P. aphanidermatum* and *P. myriotylum* is about 34°C (maximum is 40°C) and for *P. pleroticum* is 25-30°C. A warm and humid climate predisposes the plant to infection at sprouting stage, because of its tender and succulent tissues (Dake, 1995).

MANAGEMENT

Soft rot is considered as a complex disease problem. Various available methods should be combined to obtain satisfactory control of this devastating disease.

Cultural practices: Cultural practices including seed selection, crop rotation, tillage, organic amendment, drainage and quarantine are commonly employed on ginger fields to control soft rot and limit the spread of *Pythium* spp. in fields that are unaffected (Le *et al*., 2014). Infected rhizomes are important sources of perennation and spread of the disease. The best method to manage the disease is the use of disease free rhizomes during planting (Dohroo, 1993). Harvey and Lawrence (2008) believed that crop rotations could change *Pythium* spp. populations, suggesting that each crop would be associated with particular *Pythium* spp. and potential inoculum could be reduced to some extent by crop rotation. Rames *et al.* (2013) also concluded that species richness of fungal and bacterial soil populations were significantly greater in plots with a 4-year program of summer and winter crop rotations or a continuous growth of pasture grass (*Digitaria eriantha* sub sp. *pentzii*), where all the green biomass was returned to the fields before the ginger was grown, than in plots treated with fumigant or left as bare fallow.

Soil amendments alter the soil reaction, change the spectrum of soil microflora and thus affect the pathogens existing in soil (Dohroo, 1993; Dohroo and Pathania, 1997). Reduction in soft rot incidence after addition of soil amendments like oil seed cakes, neem cake and other organic matter from various plants in the field was recorded (Sadanandan and Iyer, 1986; Thakore *et al.*, 1987). Integration of neem seed powder and punarnava (*Boerhavia diffusa*) leaves, instead of poultry manure, into soil at the time of land preparation also reduced soft rot intensity in ginger by up to 89% in comparison to the untreated control (Gupta *et al*., 2013). Amendment of organic matter, including poultry manure and sawdust (200 t/ha) enriched diversity of soil microbial communities in these ginger fields (Rames *et al*., 2013). These practices also increased soil carbon levels and water infiltration rates which supported growth and yield of ginger and helped to suppress soft rot on ginger (Smith *et al*., 2011; Stirling *et al.,* 2012). Neem seed cake was found most effective with least average mortality of 20.3 per cent followed by poultry manure (22.7 %) while evaluating organic and inorganic amendments to manage rhizome rot in pot culture studies (Kadam *et al*., 2014).

Kumar *et al*. (2012) also found that *Schima wallichii* and *Datura* spp. were the best mulches with regard to suppressing soft rot caused by *P. aphanidermatum* with the level of disease incidence at 12 and 14%, respectively, compared to 48% in a non-mulched treatment. Smith and Abbas (2011) suggested that good water drainage was important in soft rot management because of zoospores production by *Pythium* spp. which were able to swim and spread in free water.

Biological management: *Trichoderma* spp. are the most widely used biocontrol agents for control of soft rot of ginger. Non-volatile and volatile compounds produced by *T. viride* could inhibit the growth of *P. myriotylum* recovered from infected ginger by 70 and 100%, respectively when assessed *in vitro* (Rathore *et al*., 1992). In addition, *T. harzianum* and *T. saturnisporum* also showed strong antagonism against *P. splendens in vitro* (Shanmugam *et al*., 2013a). Along with *Trichoderma* spp., several rhizobacteria were also antagonistic and showed significant inhibition of *P. myriotylum* growth in dual culture assays (Bhai *et al*., 2005). Dohroo *et al*. (2012) found that growth of *P. aphanidermatum* on PDA amended with onion and garlic extracts at 5% and 7.5% (v/v), respectively, was completely inhibited by poisoned food technique. Fresh and stored cow urine also strongly inhibited the growth of *P. aphanidermatum* at a concentration of 20% (v/v) by using the similar technique (Rakesh *et al.,* 2013). Use of *Jeevatu*, particularly *Jeevatu* based organic liquid manure, was found to play a vital role in soft rot control and no further spread of the disease was observed after the application of *Jeevatu* based organic liquid manure in the field in Lalitpur district of Nepal (Poudyal, 2012).

Ram *et al*. (2000) found that the percent of soft rot on *Trichoderma.* spp. coated ginger was 2-3 times less than that of the untreated control. Suppression of soft rot was even better, reducing soft rot incidence up to four times, once seed ginger was first surface disinfested with 1% HOCl for 5 min and soaked in a suspension of *Trichoderma* spp. talc-based formulation (6 x 10^7 CFU/L) and followed by three applications of talc-based formulation (3 x 10^6 CFU/g) to the soil at 15 day intervals from the time of planting (Shanmugam *et al.,* 2013b). An application of *T. harzianum* + *Glomus mosseae* + fluorescent Pseudomonad strain G4 limited infection of *P. splendens* to 10% compared with 30, 43, and 50% infection in treatments with *T. harzianum*, *G. mosseae*, and G4 as single treatments, respectively (Gupta *et al.*, 2010). Moreover, antagonists also play a role as plant growth promoters. Growth and yield of ginger growing in soil with antagonistic agents were better compared to soil without biocontrol agents application (Bhai *et al.,* 2005; Gupta *et al.,* 2010; Shanmugam *et al*., 2013a).

A mixture of *Burkholderia cepacia* + *T. harzianum* recorded a maximum rhizome production efficiency of 84% with reduction of soft rot incidence of 79.7% under polyhouse conditions. Such reduction of soft rot incidence was attributed to the increase in products of defense gene chitinase and which might be involved in disease suppression. In field experiments, the same mixture reduced the rhizome rot to 49.3% and an increased rhizome yield with an average increase of 60.0% over the untreated control (Shanmugam *et al*., 2013b). Praveen and Sharma (2014) screened crude extracts of 20 plant species against *P. aphanidermatum in vitro* and found that *Jacaranda mimosifolia, Moringa olifera* gave the best inhibitory activity of 27.7%.

Host resistance: Developing a *Pythium* resistant variety would be ideal for effective soft rot disease management. Indrasenan and Paily (1974) reported that Maran cultivar was resistant against soft rot caused by *P. aphanidermatum.* Setty *et al.* (1995a) evaluated eighteen ginger cultivars against soft rot (*Pythium* sp.) under field conditions and none was found resistant, however, cultivars Supraba and Himachal Pradesh demonstrated less than 3 per cent disease incidence. Senapati and Sugata (2005) screened 134 ginger varieties available in Koraput, Orissa, India and found one resistant cultivar and eight others with moderate resistance. Kavita and Thomas (2008) found that the accessions of *Zingiber zerumbet* were the most suitable candidates for donating soft rot resistance to cultivated ginger. Bhai *et al.* (2013) screened 650 ginger accessions and showed that only 7 percent of the accessions were having the relative resistance to the pathogen.

Chemical management: *Pythium* spp. can survive in the soil for a long period once introduced (Hoppe, 1966). Therefore, the management of soft rot becomes difficult. Chemicals such as mancozeb, ziram, guazatine, propineb

and copper oxychloride effectively controlled soft rot when used as 30 minutes dip treatments for rhizomes (Dohroo and Sharma, 1986; Thakore *et al.*, 1988). An experiment conducted on a naturally infested field with *P. aphanidermatum* in Raigarh, India, showed that seed dipping applications with Ridomil MZ at a rate of 1.25 g/L could increase survival of rhizomes by about 30 percent in comparison to hot water treatment at 51°C for 30 min (Singh, 2011). Seed coating with Fytolan (copper oxychloride) 0.2 percent + Ridomil 500 ppm + Bavistin (carbendazim) 0.2 percent+ Thimet could keep ginger rhizomes free from soft rot in a pot trial (Rajan *et al*., 2002). Similarly, Smith and Abbas (2011) found that fungicides such as metalaxyl, Ridomil, Maxam XL (fludioxonil) and Proplant (propyl carbamate hydrochloride) applied as a seed treatment could give significantly better control of soft rot caused by *P. myriotylum* than sole carbendzim seed treatment in a pot trial.

Different chemicals have also been tested by various workers as soil drench against soft rot of ginger. Soft rot was reduced by zineb, captafol, methyl bromide, mercuric chloride, thiram, phenyl mercury acetate, copper oxide and mancozeb (Doshi and Mathur, 1987). Dohroo *et al.* (1984) found metalaxyl application quite effective for the control of rhizome rot. Dipping of seed one day before planting and soil drenching with a mixture of metalaxyl plus captafol 3 months after planting controlled soft rot of ginger (Rathaia, 1987). Fosetyl-Al, metalaxyl, oxadixyl, propamocarb and ethazole (epidiazole) were also evaluated against *P. aphanidermatum.* Metalaxyl formulations (Ridomil 5G and Apron 35 WS) gave best control of the disease when used as soil and seed treatments (Ramachandran *et al*., 1989). Srivastava (1994) managed soft rot (*P. aphanidermatum*) in Sikkim effectively by drenching the soil with zineb or mancozeb following rhizome treatment with carbendazim and incorporating Thiodan dust into the soil to control insect invasion. Nath (1993) suggested planting of ginger under shade after treating with 1 per cent formaldehyde. Treated rhizomes grown under shade had 19.4 per cent incidence of soft rot (*P. myriotylum*) as compared to 41.3 per cent incidence in treated rhizomes grown without shade.

Integrated management: Management of soft rot is difficult by following a single approach because it does not work effectively to suppress the pathogens under field conditions. Therefore, an integrated approach was suggested by Smith and Abbas (2011) which was mainly based on cultural practices and a strict quarantine procedure to manage the disease, while Mathur *et al.* (2002) found that soil solarization and application of fungicides could effectively minimize the incidence of soft rot caused by *P. myriotylum*. Rhizome treatments with Ridomil MZ (metalaxyl + mancozeb) at 6.25 g/L in addition to soil drench with Thimet (Phorate) and Ridomil MZ at 10 L/3x1 m plot at 60 days after sowing gave the best control of *P. myriotylum* on an experimental ginger field in southern Rajasthan, India. The treatments performed even better in solarized plots achieved by sealing thick transparent polythene film over the soil surface for 20 days. Lokesh *et al.* (2012) suggested that seed ginger could be solarized at 47°C under 200 mm polyethylene sheet for 30 min for *Pythium* spp. disinfestations. However, longer periods (up to 4 weeks) of soil solarization caused significant reduction in the populations of *Pythium* spp. and a lower disease incidence in solarized plots (Deadman *et al*., 2006).

Dohroo and Gupta (2014) reported combined applications of bioagents more effective in reducing the disease than the individual treatments. *T. harzianum*+ *P. fluorescens* + *B. subtilis* gave minimum disease incidence on rhizomes (8.64 %) as well as on tillers (12.50 %). Copper oxychloride rhizome treatment effectively suppressed the disease development (5.16%) at 150 days after planting in the field followed by neem extract rhizome treatment (Lalfakawma *et al*., 2014). A two years field study indicated that rhizome treatment in hot water at 47°C for 30 min and soil application of *T. harzianum* @ 2.5 kg/ 50 kg FYM/ha, followed by three drenching of mancozeb @ 0.25% were most effective in limiting the incidence of soft rot on ginger besides having their significant response in improving the growth and yield (Dohroo *et al*., 2015).

2. YELLOWS

Yellows disease is one of the serious problems of ginger because it has become more widely spread wherever warm and humid environmental conditions prevail. It was first described by Simmonds (1955) from Queensland.

Symptoms: Yellowing of the margins of the lower leaves is the initial symptom which gradually spreads, covering the entire leaves. Older leaves dry up initially followed by the younger ones. Plants may show a premature drooping, wilting, yellowing and drying in patches or in whole bed. Plants may show stunting. In rhizomes, a cream to brown discolouration accompanied by shriveling is commonly seen. Central core rot is also prominent. Rotting of roots is common and the rhizome formation is affected. In final stages of decay, only the fibrous tissues remain within the rhizomes. A white cottony fungal growth may develop on the surface of stored rhizomes. In dry rot, though the leaves become pale but no soft rot of collar region is observed. Such plants cannot be easily pulled out. Mycelial growth in the form of white, peach or buff coloured cushions can be seen on the surface of rhizomes (Dohroo, 1982).

Causal organism: The disease is caused by *Fusarium oxysporum* Schlechtend ex Fr. f.sp. *zingiberi* Truzillo (Yang *et al.*, 1988). Other species of *Fusarium* such as *F. solani* (Mart.) Sacc., *F. equiseti* (Corda) Sacc. and some unidentified *Fusarium* spp. were also reported to be associated with ginger rhizomes. Sharma and Dohroo (1990) reported *F.*

oxysporum as the major cause of yellows from Himachal Pradesh. The second most frequently isolated species was *F. solani* (Dohroo, 1987; Chauhan and Patel, 1990), however, *F. moniliforme* Sheld., *F. graminearum* Achwabe and *F. equiseti* (Sharma and Dohroo, 1980; Bhardwaj *et al.*, 1988b; Dohroo, 1987) were also found associated with the diseased plants. Isolates of *F. oxysporum* f. sp. *zingiberi* differed in their aggressiveness (Dohroo and Sharma, 1992b).

F. equiseti (Forda) Sacc. produces microconidia and macroconidia. Microconidia are aseptate measuring 6.5 – 10.0 x 3.0 – 4.0 µm in size. Macroconidia are 1 to 3 septate, sickle to spindle shaped measuring 23.5- 33.0 x 3.0- 4.0 µm in size. Sporodochial formation is rare (Dohroo, 1982). Ramteke and Kamble (2011) investigated the effect of carbon and nitrogen sources, temperature, pH levels and light spectra on mycelial growth of benomyl sensitive and resistant isolates of *F. solani* (Mart.) Sacc. Sucrose was found to be the best source of carbon, whereas calcium nitrate was the best source of nitrogen. The fungus grew at temperatures ranging from 10 to 35ºC with optimum growth at 25ºC while no growth was observed at 5ºC and 40ºC. The most suitable pH level for the growth was 4.5.

Pappallardo *et al.* (2009) analysed genetic variation among 29 isolates of *F. oxysporum* f.sp. *zingiberi* using DNA amplification fingerprinting (DAF). Within these isolates, three haplotypes were identified based on 17 polymorphic bands generated with five primers. Two groups showed very little genetic variation (98.6% similarity), whereas the third single isolate was quite distinct in terms of its molecular profile (77.2% similarity). Shanmugan *et al*. (2013b) studied genetic variability of 32 *Fusarium* isolates from diseased ginger rhizomes from Western Himalayas in India. They were analyzed by the unweighted pair group method with arithmetic averaging using randomly amplified polymorphic DNA amplicons. Of two major clusters formed, one was dominated by *F. oxysporum* and the other by *F. solani*. Morphological, cultural, pathological and molecular variability among *F. oxysporum* f.sp. *zingiberi* isolates were studied by Gupta *et al*. (2014). Molecular variability revealed 0 to 80% variation among nineteen isolates and they were grouped into two different major groups each comprising of ten and nine isolates, respectively.

Disease cycle and epidemiology: The seasonal carryover of fungus inoculum takes place through infected rhizomes and soil. The fungus survives in soil as chlamydospores which may remain viable for many years in the field. The fungus spreads through infected seed rhizomes and about 87% of field infection is due to infected rhizomes (Dohroo, 1989a). The secondary spread of the disease can also take place through irrigation water and by mechanical means.

For the development of yellows disease, a temperature range of 15 to 30°C is favourable (the optimum being 23-29°C) accompanied by very high humidity and continuous presence of free water (Sharma and Jain, 1978). Maximum disease incidence occurred when soil temperature ranged from 24 to 25°C and the soil moisture from 25 to 30 per cent (Sharma and Dohroo, 1989).

MANAGEMENT

Cultural practices: The main mode of disease spread is through contaminated rhizomes. Therefore, the selection of healthy seed rhizomes has been found an effective control measure for the disease (Rana, 1991; Dohroo, 1993). Smith *et al*. (2011) found greatest (74.2 t/ha) rhizome yield and minimal (7.0%) losses to pathogens in the pasture lay that had been cultivated prior to ginger planting. Stirling *et al*. (2012) reported that organic inputs, tillage and rotation practices did not influence yellows disease. Results of bioassays were too inconsistent to draw firm conclusions, even soil management practices had little impact on disease severity. Sharma *et al*. (2012) observed plant spacing of 25 × 30 cm, seed rhizome size of 50 to 75 g to be optimum for better crop return and lower disease incidence.

Chemical management: Efficacy of a variety of chemicals has been evaluated for the management of this disease by different workers and they have found very promising effect of different chemicals against the disease (Singh and Gomez, 2001; Singh *et al.,* 2004; Meena and Mathur, 2005; Usman, 2006; Stirling *et al.*, 2006). Systemic and contact fungicides like Bavistin 50WP, Ridomil Gold MZ-72 and contact fungicides like Captan, Dithane M-45, copper oxychloride and Bordeaux mixture etc. were reported effective against the disease (Sagar, 2006; Hasnat *et al*., 2014).

Biological management: Applications of *Trichoderma* spp. are very effective biological mean for plant disease management especially for the soil borne pathogens. Growth of *F. oxysporum* f.sp. *zingiberi* was inhibited effectively by *T. harzianum and Gliocladium virens* (Sharma and Dohroo, 1991), *T. viride* and *T. harzianum* (Khatso and Ao, 2013), *T. viride* (Amreen and Kumar, 2013). Besides *Trichoderma* spp., the efficiency of six *Streptomyces* species was tested against *F. oxysporum* f.sp. *zingiberi* by Manasa *et al.* (2013) following dual culture method and agar well diffusion method. In both the assays, marked inhibitory activity was observed in case of *S.* species SSC-MB-02.

Application of a mixture of *Bacillus cepacia* + *T. harzianum*, recorded an increased rhizome production and decreased incidence of yellows in a polyhouse. In field experiments, the said mixture reduced yellows and increased rhizome yield of 45.9 percent and 60.0 percent, respectively, over control (Shanmugam *et al*., 2013a). Talc-based formulations of the plant growth promoting rhizobacteria strain XXBC-TN (*Bacillus subtilis*) and a mixture of S2BC-1 (*B. subtilis*) with TEPF-Sungal (*Burkholderia cepacia*), known to inhibit *F. oxysporum* and *F. solani*, were developed for rhizome dressing and soil application in ginger fields. The strain mixture recorded the maximum rhizome

production (85.2%) with fewer yellows and reduced rhizome rot incidences (87.8% and 88.4%) over the control in a polyhouse. This was associated with an increase in the defense enzymes chitinase, β-1,3-glucanase, and polyphenol oxidase. Furthermore, the strain mixture treatment promoted plant growth and enhanced rhizome production by 45.8%. In field experiments, the PGPR strain mixture reduced yellows and rhizome rot incidences by about 50.5%, which was comparable to that of carbendazim and mancozeb fungicide mixture (Shanmugam *et al*., 2013b).

Among 14 plant extract tested against *F. solani,* maximum inhibition of mycelial growth was noticed in *Ferula asafeotida* powder extract (68.51%) followed by *Ocimum* leaf extract (60.16%) (Sagar *et al*., 2007). Alcoholic leaf extracts of *Swietenia macrophylla* King, *Azadirachta indica* A. Juss., *Hyptis suaveolens* (L.) Poit., *Polyalthia longifolia* (Sonn.) Thw., *Boerhaavia repens* L. var. *diffusa* (L.) Hook. and *Tithonia diversifolia* A. Gray had 100 percent control against both sensitive and resistant isolates of *F. solani* at 25 percent concentration (Ramteke and Kamble, 2011).

Host resistance: By using resistant or less susceptible cultivars of ginger the disease can be managed to a great extent. Cultivars like SG 666 (Dohroo, 1989b) and Kerala local (Rana and Arya, 1991) have been reported to show fewer incidences of yellows under field trials in Himachal Pradesh. Priya and Subramanian (2008) reported the presence of a resistance (R) gene of CC–NBS–LRR class of plant resistance genes. Both direct PCR amplification from genomic DNA as well as cDNAs, yielded a 0.6 kb DNA sequence indicating the absence of an intron. Sequence analysis of the PCR amplicon obtained from the genomic DNA showed very high homology to R-genes and this R-gene is present in only resistant varieties. Sharma *et al*. (2012) reported cultivar "Majauley" to be moderately susceptible while none was found to be tolerant against rhizome rot and wilt disease complex of ginger.

Integrated management: Dohroo (1995) suggested an integrated approach to combat the yellows disease of ginger which included treatment of seed rhizomes with mancozeb and carbendazim and use of biocontrol agents *T. harzianum, T. hamatum and G. virens* as seed treatment and soil application. Hasnat *et al.* (2014) observed the lowest disease incidence (27.78%) in Ridomil Gold which was statistically similar with the plots which were applied with poultry waste, Bavistin 50WP, Dithane M-45 and saw dust at 240 days after planting.

3. PHYLLOSTICTA LEAF SPOT

Phyllosticta leaf spot disease is becoming increasingly important in many of the states due to severe leaf rot and blight it causes. The disease was first reported from Godavari district of Andhra Pradesh and Malabar area of Kerala, erstwhile Madras state (Ramakrishnan, 1942).

Symptoms: Initial symptoms of the disease are small oval to elongated spots on the leaves, measuring 1-10 mm x 0.5 - 4 mm. Later on, the spots show white papery centre and dark brown margins with a yellowish halo surrounding it (Ramakrishnan, 1942). The spots increase in size and coalesce to form larger lesions. The affected leaves become shredded and may suffer extensive desiccation. Symptoms appear first on younger leaves. As the plants put forth fresh leaves, these get infected subsequently.

Causal organism: Phyllosticta leaf spot is caused by *Phyllosticta zingiberi* T.S. Ramakr. The fungus forms amphigynous, subglobose, dark brown ostiolate pycnidia on the host measuring 78 to 150 µm in diameter. On standard media, the fungus forms pycnidia having 100-270 µm diameter bearing hyaline, unicellular, oblong, big guttulate spores measuring 3.7 to 7.4 x 1.2 to 2.5 µm (Ramakrishnan, 1942).

Disease cycle and epidemiology: The infected debris or seed serves as primary inoculum for the disease. In leaf, pycnidiospores and mycelia remain viable for 14 months under laboratory conditions (Brahma and Nambiar, 1982). Pycnidia survive in the leaf debris throughout the summer having temperature range of 30 to 35°C. The pycnidiospores remain viable in soil even at 25 cm depth for 6 months. The optimum temperature range for mycelial growth of *Phyllosticta* was 25.0 to 27.5°C with maximum and minimum to be 32.5 and 10.0°C, respectively. At 5 and 35°C complete inhibition of mycelial growth was observed (Cerezine *et al*., 1995).

The extent of dispersal of causal fungus depends upon the intensity of precipitation. Higher intensity of rain accompanied by wind seems to exert greater impact on target leaf so that spores are splashed to greater distances resulting in liberation of greater amount of spores and increasing disease incidence. The disease begins to appear towards the end of June. During this period, the temperature varies between 23.4 to 29.6°C and relative humidity is between 83.3 to 90.2 per cent. Later in July when the number of rainy days and total rainfall increase, the disease aggravates and spreads very fast (Brahma and Nambiar, 1984). Sood and Dohroo (2005) reported the influence of environmental factors *viz*., air temperature, relative humidity and rainfall on the disease development to an extent of 85.5 per cent. Ginger plants up to the age of 6 to 7 months are susceptible to the disease and two weeks old leaves are most susceptible. It was observed that temperature range of 23 to 28°C with intermittent rain favoured disease development. Disease incidence was found to be less when ginger is grown under partial shade or as intercrop in coconut gardens (Senapati *et al*., 2012). Continuous cultivation of ginger in the same field builds up higher concentrations of incoculum and early infection of the plant reduce the vigor leading to reduction in the rhizome yield (Singh, 2015).

MANAGEMENT

Cultural practices: Shade plays an important role in reducing the severity of Phyllosticta leaf spot. Farmers of the Sikkim state observed that partial shading of mandarian orange provided a favourable environment for growth of ginger and disease intensity remained often less as compared to that of open cultivation (Patiram Upadhaya *et al*., 1995). The disease severity and sun burn was statistically lower in heavy shade in comparison to open sun grown ginger. However, considering all the parameters viz; reduction in Phyllosticta leaf spot and sun burn of leaves, increased the number of tillers per clump and yield, growing of ginger in partial shade may be recommended to avoid the fungicidal spray for controlling Phyllosticta leaf spot and consequently avoiding fungicidal pollution (Singh *et al*., 2004).

Chemical management: Sprayings of Bordeaux mixture, zineb and maneb have been reported effective in checking the disease (Sohi *et al.*, 1973). Twelve sprays or mixture of benomyl (0.1%), mancozeb (0.2%) and soluble boron (0.1%) and iprodione (0.2%) alone were most effective in reducing the disease (Grech and Frean, 1988). In Brazil, Cerezine *et al.* (1995) found highest reduction in the disease progress with chlorothalonil. One spray of carbendazim (0.15%) and two sprays of mancozeb (0.25%) gave good protection against the disease and resulted in higher yield under pot culture experiment (Verma and Vyas, 1981). Sood and Dohroo (2005) found that rhizome treatment as well as foliar sprays with Bordeaux mixture (1%), Companion (0.2%), Indofil M-45 (0.25%), Unilax (0.2%) and Baycor (0.05%) were effective in checking the disease severity, however, Bordeaux mixture and Companion effectively increased the rhizome yield of ginger.

Host resistance: None of the 18 cultivars tested in Karnataka were resistant to *Phyllosticta* leaf spot (Setty *et al.*, 1995b). However, the cultivars Narasapatom, Tura, Nadia, Tetraploid and Thingpani were classed as moderately resistant with a disease index less than 5 per cent. In Himachal Pradesh, none of the tested material of ginger was rated resistant to *P. zingiberi,* however, 8 lines showed moderate resistance (Dohroo *et al.*, 1986b). Different workers obtained variable results and none of the tested cultivars showed high degree of resistance (Dohroo *et al.,* 1986b; Rao *et al.,* 1995). Nageshwar R*ao et al.* (1995) screened 100 accessions of ginger for their reaction and tolerance to leaf spot under field conditions and of them, 11 accessions were found tolerant and a further 42 were moderately tolerant. Senapati *et al*. (2012) found that PGS-16, PGS-17 and Anamica as moderately resistant out of 135 ginger cultivars tested.

4. STORAGE ROTS

Ginger rhizomes are stored for seed and commercial purpose in different types of storage structures. During storage, rhizomes are attacked by number of fungi and bacteria.

Causal organism: During storage, different fungi have been found associated with the ginger rhizomes, which result in rotting and decaying of the rhizomes (Dohroo, 1993). These fungi include *F. oxysporum* Schlechtend ex Fr., *P. deliense* Meurs and *P. myriotylum* Drechs. (Sharma and Jain, 1977), *Geotrichum candidum* Link (Mishra and Rath, 1989), *Aspergillus flavus* Link ex. Fr. (Geeta and Reddy, 1990), *Cladosporium lennissimum, Gliocladium roseum* Bainer, *Graphium album* (Corda) Sacc., *Mucor racemosus* Fresen., *Stachybotrys sansevieriae, Thanatephorus cucumeris* (Frenk) donk and *Verticillium chlamydosporium* Goddard (Dohroo and Sharma, 1992a). *Pythium ultimum, Fusarium oxysporum* and *Verticillium chlamydosporium* were found associated with storage rot of ginger. The disease was noticed in storage pits from January, which reached its maximum intensity in April at 15.5°C temperature and 67.5 per cent relative humidity (Dohroo, 2001). *Penicillium brevicompactum* was the predominant species isolated from 85% of the rhizomes displaying visible mold growth (Overy and Frisvad, 2005).

Moreira *et al.* (2013) reported positive pathogenicity tests for *Acremonium murorum, Acrostalagmus luteo-albus, Fusarium* sp., *F. oxysporum, Lasiodiplodia theobromae* and *Sclerotium rolfsii* associated with the post-harvest rot of ginger rhizomes in the Serrana region of Espírito Santo, Brazil.

MANAGEMENT

Cultural practices: Storage of rhizomes under cooled conditions may prolong storability by reducing weight loss and sprouting but may result in higher pathogen incidence than storage at room temperature. Packing of rhizomes in PVC film also reduces weight loss but increases incidence of fungal infection (Lana *et al.*, 1993). Ram and Thakore (2009) showed that storage rot could be effectively minimized by dipping rhizomes of ginger in an extract of *Allium sativum* @ (10% w/v) or in a combined suspension of *P. fluorescens* and *T. harzianum* @ 0.5% for 30 min before storage. Jadhav *et al*. (2013) reported that storage rot could be effectively reduced by dipping ginger rhizome in garlic extract @ 20% w/v for 30 minutes before storage.

Chemical management: Okwuowulu and Nnodu (1988) suggested pre-storage chemical treatments of ginger rhizomes with benomyl (750 ppm) and/or gibberellic acid (150 ppm) to reduce the incidence of storage rots. Dipping of rhizomes in imazalil or prochloraz at 0.8 g a.i. per litre and then storing at 10°C gave good protection against the infection with fungi such as *Botryodiplodia, Aspergillus, Diplodia, Fusarium, Rhizoctonia* and *Pythium* in storage (Grech and Swarts, 1990). A combined application of mancozeb and carbendazim to ginger rhizomes controlled storage rot of ginger (Dohroo *et al.*, 1986a; Dohroo and Malhotra, 1995). Under stored conditions, application of 0.3% Ridomil MZ resulted in the lowest incidence of disease

(Singh *et al.*, 2004). Steeping of rhizomes in carbendazim (0.1%) for 60 minutes before storage also controlled storage rots and reduced the disease incidence from 71.4 to 18.2 per cent (Sharma and Dohroo, 1991).

Under storage conditions, postharvest dipping of aureofungin (0.02%) and Benomyl (0.2%) provided better control of the disease (Haware *et al.*, 1973). Disease incidence under storage was reduced from 71.4 to 18.2 per cent by steeping the rhizomes in carbendazim (0.1%) for sixty minutes (Sharma and Dohroo, 1991).

Amongst various fungicides used for dipping the rhizomes before storage, mancozeb is known to persist longer than carbendazim (Sharma *et al.*, 1992). Mancozeb residues were observed even after 120 days of storage. However, the health risk in carbendazim treated rhizomes is low as compared to mancozeb if they are consumed after peeling. Dohroo (2001) reported that pre-storage treatment with Topsin-M and Bavistin each at 0.2 per cent concentration for 60 minutes reduced incidence of storage rot, loss in rhizome weight, surface shriveling, sprouting of ginger and increased the recovery of the rhizomes.

5. BACTERIAL WILT

Bacterial wilt is widespread and exceedingly destructive in several countries. Bacterial wilt has been reported for the first time from Malabar region in the Madras presidency in 1941 by Thomas. In India, this disease occurred since middle of the century but Mathew *et al.* reported it in 1979 from Kerala.

Symptoms: On the collar region, water soaked patches or linear streaks appear. These symptoms are followed by yellow to bronze colouration of margins of the lower-most leaves which gradually progresses upwards. At later stages, the leaves become flaccid with intense yellowish bronze colour and droop ultimately exhibiting typical wilt symptoms. In the infected plants, leaf sheaths look yellowish to dull green. The leaves roll up and the whole plant dries up finally. The pseudostems can be easily separated with a gentle pull and can be broken off at the base. At advanced stage the pseudostem appears slimy. The plants which are infested by the disease stand persistently and do not collapse. If the affected rhizomes are pressed, a milky bacterial exudate oozes out. When infected tissues are steeped in clear water for a while, the water turns cloudy and milky.

Causal organism: The disease is caused by *Ralstonia solanacearum* (Smith) Yabuuchi *et al.* Three biotypes of *R. solanacearum* have been described and biotype III causes the wilt in India (Dake *et al.*, 1988). However, Pegg *et al.* (1974) reported that biotype III of the bacterium caused slow wilt whereas biotype IV caused rapid wilting and death of infected plants. *R. solanacearum* biotype III was found restricted only to ginger and its common weeds whereas biotype IV had a wide host range including tomato, potato, *Zinnia elegans, Capsicum frutescens, Physalis peruviana* and eggplant. Biotype II of *R. solanacearum* was also isolated from potato plants (Hayward *et al.*, 1967).

In Indonesia, the race 1 of biovar III of *R. solanacearum* was considered as the cause of ginger wilt (Mulya *et al.*, 1990). Dohroo (1991) reported occurrence of bacterial wilt of ginger in Himachal Pradesh. The wilt incidence increased when abundant nematodes were there in the ginger soil (Samuel and Mathew, 1986). The host range of *R. solanacearum* race 4 was restricted to edible ginger (Nelson, 2013). Fourteen species of ginger belonging to Zingiberaceae and Costaceae were evaluated for susceptibility to *Ralstonia solanacearum* (Rs) race 4 (ginger strains) by several methods of inoculation, including tests to simulate natural infection. Twelve of 14 species tested were highly susceptible to all strains of Rs race 4 upon stem inoculation, and susceptible plants wilted within 21 days (Paret *et al*., 2008).

The genetic diversity of *R. solanacearum* strains isolated from ginger growing on Hawaii island was determined by analysis of amplified fragment length polymorphisms (AFLPs). *R. solanacearum* strains from ginger in Hawaii island showed a high degree of similarity at 0.853 and strains from ginger in Hawaii were genetically distinct from local strains from tomato (race 1) and heliconia (race 2) (Yu *et al*., 2003). Kumar and Sarma (2004) detected Isolates of *R. solanacearum* of ginger with NCM-ELISA and biovars on the basis of membrane protein pattern on SDS-PAGE and biovar specific protein from *R. solanacearum* could be isolated. *R. solanacearum* was detected by PCR from rhizomes and soil (Kumar and Anandaraj, 2006; Kumar and Abraham, 2008) and using real time PCR from rhizomes (Thammakijjawat *et al*., 2006).

Disease cycle and epidemiology: *R. solanacearum* spreads by infested soil adhering to hands, boots, tools, vehicle tires, and field equipment; in water from irrigation or rainfall; and by infected ginger rhizomes (Janse, 1996). This bacterium infects ginger roots and rhizomes through openings where lateral roots emerge or through wounds caused by handling, parasitic insects, or root-knot nematodes (Swanson *et al*., 2005). The pathogen survives in soils within infected plant debris and as free-living bacteria. Ginger crops can be completely lost to the disease in heavily infested soils (Nelson, 2013).

MANAGEMENT

Cultural practices: Currently, the bacterial wilt management depends on selection of disease free seed rhizomes, rhizome treatment by hot air or hot water or rhizome solarization, periodical rouging of infected plants and crop rotation with non-host plants to reduce the disease causing potential of soil. Indrasenan *et al.* (1981) suggested selection of healthy

seed rhizomes, eradication of weeds and adoption of an effective crop rotation as control measures for the disease. Almost all the cultivars were susceptible to wilt pathogen (Indrasenan *et al.*, 1982). Tsang and Shintaku (1998) found that ginger rhizome when exposed to heat for 30 min at 50°C (122°F) and 45 min at 49°C (120°F), respectively, bacteria were eliminated. Exposing ginger seed pieces to hot air at 75% RH until their center temperatures attained 49°C (120°F) for 30 and 60 min and 50°C (122°F) for 30 min, resulted in minimal injury to the hosts. More than 87% of the seed pieces germinated without adverse effect on growth.

Chemical management: Treatment of seed rhizomes with emisan plus plantomycin for 30 minutes followed by three sprayings, first at 30 days after planting and others at an interval of 15 days, also gave good control of the disease (Ojha *et al.*, 1986). Sinha *et al*. (2000) evaluated five antibiotics *in vitro* and *in vivo* against bacterial wilt of ginger. Streptomycin and streptopenicillin were superior over the other antibiotics against the pathogen under both conditions.

Biological management: *Bacillus subtilis* strain 1JN2, *Myroides odoratimimus* 3YW8, *B. amyloliquefaciens* 5YN8, and *Stenotrophomonas maltophilia* 2JW6 showed biocontrol efficacies greater than 50% in greenhouse against *bacterial* wilt of ginger (Yang *et al*., 2012).

6. MOSAIC

The symptoms appear as yellowish and dark green mosaic pattern on leaves. The affected plants show stunting.

The virus causing mosaic in ginger has spherical particles with diameter of 23 to 38 µm. It showed positive serological reaction with antiserum of cucumber mosaic virus (CMV). The virus is known to be transmitted by sap to different plants known to be hosts of CMV (Su, 1980). Nambiar and Sarma (1974a) 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 cardamom, Curcuma longa* and *C. aromatica* gave negative results.

7. CHLOROTIC FLECK

The chlorotic fleck virus was characterized and described by Thomas (1986). He detected the virus in ginger imported from Australia and a number of countries. The geographical distribution of the virus was uncertain, and thought to include India, Malaysia and Mauritius.

The ginger chlorotic fleck virus (GCFV) has isometric particles about 30 nm diameter, with a sedimentation coefficient of 111s and readily purified from infected ginger leaf tissue. The purified preparations contained a major species of single stranded RNA MW 1.5 x 10^6 and major coat protein MW 29 x 10^3. At pH 7, the particles formed a single zone in both cesium chloride and cesium sulphate gradients, with buoyant densities of 1.355 g/cm^3 (fixed virus) and 1.297 g/cm^3 (unfixed virus), respectively. The virus particles migrated as two electrophoretic components and were liable when treated with 10mM EDTA, 1M NaCl, 10 mM tris pH 8.25 or when negatively stained with potassium phosphotungstate.

The virus was transmitted by *Myzus persicae, Pentalonia nigronervosa, Rhopalosiphum maydis* or *R. padi*. Possible affinities of GCFV with the subemovirus group was also described by Thomas (1986).

8. MINOR DISEASES

Some diseases of minor importance have also been reported on ginger like Cercospora leaf spot caused by *Cercospora zingibericola* (Kar and Mandal, 1969), anthracnose caused by *Colletotrichum zingiberis* (Nema and Aggarwal, 1960), *Pyricularia* leaf spot caused by *Pyricularia zingiberi* (Rathaiah, 1979), basal rot caused by *Sclerotium rolfsii* (Mehrotra, 1952; Haware and Joshi, 1973) and *Septoria* leaf spot caused by *Septoria zingiberis* (Sundaram, 1961).

CONCLUSION

Ginger occupies an important place in spices throughout the world and India is one of the leading producer and exporter of ginger in the world. However, during cultivation this crop is infected by a myriad of diseases caused by different fungal, bacterial and viral pathogens which reduce the potential yields drastically. For reducing the losses caused by these pathogens, a sound knowledge of occurrence, distribution, symptoms, biology, perpetuation, transmission and epidemiological factors is required. Further, an insight into management practices such as cultural practices, host resistance, biological, chemical management and an integration of these practices is needed. Therefore, an effort has been made to review geographical distribution, losses, symptoms, causal organism, disease cycle, epidemiology, host resistance, cultural, biological, chemical and integrated disease management strategies of diseases infecting ginger.

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