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Chapter

Diseases of Ginger

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Abstract

Ginger is one of the earliest known oriental spices grown for its edible rhizome, which is widely used as a fresh vegetable, spice, and as a popular folk medicine. Ginger crop is being affected by insect pests, and pathogenic and non-pathogenic diseases cause production constraints. Severely, various pathogenic diseases of viral, bacterial, fungal, and nematode origin reduce its potential yields drastically. Among the various diseases, soft rot, yellows, *Phyllosticta* leaf spot, storage rot, bacterial wilt, mosaic, and chlorotic fleck are important. The present chapter includes the symptoms, causative agent, disease cycle, epidemiology and host resistance, cultural, biological, chemical, and integrated management of these diseases.

Keywords: ginger, soft rot, yellows, leaf spot, wilt, mosaic, chlorotic fleck

1. Introduction

India is considered as a '*magical land of spices*' with diverse variety of spices. Ginger (*Zingiber officinale* Roscoe) is earliest known oriental spices, belonging to the family, *Zingiberaceae*. Though entire plant is refreshingly aromatic, the underground rhizomes of this crop are valued as spice. It is one of the commonly consumed dietary condiments in the world and has high medicinal properties.

Ginger is being cultivated in the various parts of the world. The total production of ginger in the world is 1683 thousand tons with the total acreage of 310.43 thousand ha [1]. China, India, Nepal and Thailand are the major producers of ginger in the world. India is the leading producer and exporter of ginger in the world. Annually, India produces 385.33 thousand tons of ginger [1].

The production of ginger is being affected by biotic and abiotic factors. Biotic factors include virus, bacteria, fungus and nematodes [2, 3]. Among the biotic factors, bacteria are most important, causes wilt and soft rot. Fungus is the next major pathogen causes rhizome rot, soft rot, Sclerotium rot and yellows disease. Nematode produces root knot disease and virus's causes mosaic and chlorotic fleck in ginger plants reduce the yield of the rhizome. The ginger is also affected by various insects like *Conogethes punctiferalis*, *Aspidiella hartii*, rhizome scale, rhizome fly and thrips. Abiotic factor causes sunburn (due to high light intensity) and lime-induced chlorosis (due to excessive liming in soil) in the ginger crop.

It is imperative to know the symptoms of the disease, the organism responsible and the protection measures. This chapter emphasizes the importance of diseases of ginger by including the symptoms, causative agent, epidemiology and protection from these diseases.

2. Viral disease

2.1 Mosaic

2.1.1 Symptoms

The symptoms appear with yellowish and dark-green mosaic on leaves of ginger in the early stage and stunted of leaves and rhizomes at the late stage of infection. Infection of this virus on ginger causes severe reduction of rhizome yield.

2.1.2 Causative agent

Virus isolated from affected ginger plants by So [4] named the virus as Ginger mosaic virus. The virus infected ginger possesses spherical particles with a diameter of 23–38 nm. The purified virus particle showed positive to serological reaction for cucumber mosaic virus (CMV) antiserum. Serological relationship and electron microscopic observation of this virus conclusively suggested that ginger mosaic virus could be the CMV group. To support this, last year (2018), a group from Malaysia identified that the mosaic disease in ginger was due to the CMV which is based on the partial nucleic acid sequence of coat protein (GenBank: MH355647.1).

2.1.3 Epidemiology

Virus produces mosaic disease in 18 cultivars of ginger and 23 other plant species [4]. The virus is transmitted through the sap to different plants which are believed to the hosts of CMV [4]. Nambiar and Sarma [5] failed to absorb the sap transmission from ginger to ginger, ginger to *Nicotiana tabacum* var. Harrison special, *N. tabacum* var. xanthi, *N. tabacum* var. rustica, *N. glutinosa*, *Elettaria cardamomum*, *Curcuma longa*, and *C. aromatica*. Assessing the concentration of virus in various parts of ginger revealed that leaves and flower possess higher concentration of virus than rhizome, stem and other parts of the plant.

2.1.4 Transmission

The virus is transmitted by insect vectors such as *Myzus persicae*, *M. certus*, *M. humuli*, *Macrosiphum euphorbiae* and *Rhopalosiphum insertum*. *Myzus persicae* and *M. certus* are comparatively more efficient vectors for this virus.

2.1.5 Protection

Hot-water and hot-air treatments of affected rhizomes at 45 and 50°C for 3, 6, and 12 h does not alleviate symptoms [5]. Ginger mosaic virus in standard extract is inactivated with 10 min exposure at 60°C [4].

2.2 Chlorotic fleck

Thomas [6] from Australia reported the chlorotic fleck in ginger is because of ginger chlorotic fleck virus (GCFV). From Australia, this virus is distributed to a number of countries which import ginger from Australia.

2.2.1 Symptoms

The leaves of infected plants show chlorotic flecks, 1–10 mm long on the centered and parallel to the veins (**Figure 1**). Symptoms started appearing in the young

leaves at the 3–4 weeks of infection and subsequently to other leaves. No obvious symptoms occur on the rhizomes.

2.2.2 Causative agent

Chlorotic fleck in ginger is due to ginger chlorotic fleck virus (GCFV). It can be readily purified from the virus ginger leaves through ultracentrifugation with a sedimentation coefficient of 111 s. The purified virus is an isometric nature with size of 28–33 nm. Virus particle contains a major coat protein with molecular weight of 29 kDa and single stranded RNA with molecular weight of 1.5 × 106 Da.

2.2.3 Epidemiology

It differs from mosaic virus in particle properties, host range and serology. Many properties namely size of particles, possession of ssRNA, salt-labile nature of particles and a limited host range of this virus are similar to sobemovirus group [7] but it, serologically unrelated to several sobemoviruses including lucerne transient streak virus, cocksfoot mottle virus, sowbane mosaic virus, Solanum nodiflorum mottle virus, southern bean mosaic virus, velvet tobacco mottle virus and turnip rosette virus.

Figure 1.
Chlorotic fleck symptoms on ginger leaf (source: https://www.plantwise.org).

2.2.4 Transmission

GCFV is mechanically transmitted only to ginger but not like ginger mosaic virus, transmitted by *Myzus persicae, Pentalonia nigronervosa* and *Rhopalosiphum maidis* or *R. padi*.

2.2.5 Protection

The viral diseases of ginger are controlled in tissue cultures by heating at 50°C for 5 min [8].

2.3 Viruses infecting flowering ginger

Flowering ginger is a member of the Alpinia genus of *Zingiberales* order of *Zingiberaceae* family. Banana bract mosaic virus (BBrMV) (Potyvirus genus of the family *Potyviridae*), Canna yellow mottle virus (CaYMV) and Banana streak virus (BSV) of Badnavirus genus of the *Caulimoviridae* family are reported to infect flowering ginger [9].

3. Bacterial diseases

3.1 Bacteria wilt

Bacterial wilt of ginger is the most serious rhizome-borne diseases. It is also soil and seed-borne disease. It is widespread and exceedingly destructive for the ginger grows in tropical, subtropical and warm temperate regions of the world. Bacterium causes rapid wilt in ginger within 5–10 days of the infection [10]. The severity of the disease is occurred due to the rapid spread of the pathogen happens during the favorable environment conditions like high rain fall and warm weather.

3.1.1 Symptoms

Initially, water soaked patches or linear streaks appear at the collar region of the pseudo stem and then progresses both upwards and downwards. The pseudo stems from the infected plant can be easily separated with a gentle pull and can be broken off at the base. Mild drooping and curling of leaf margins of lower leaf is the first prominent symptom occurred after the infection, then the infection spread upwards later. Yellowing starts from the lower-most leaves which gradually progresses upwards. In the advanced stage, infected ginger exhibit intense yellowish and wilting symptoms (**Figure 2**). Dark streaks are observed in the vascular tissues of the affected pseudo stems. In the infected plants, leaf sheaths look yellowish to dull green. The leaves roll up and the whole plant dries up, finally. The plants which are infested by the disease stand persistently and do not collapse. The base of the infected pseudo stem and the rhizome emit foul smell. The affected pseudo stem and rhizome extrudes milky ooze from the vascular strands while they pressed gently. Milky bacterial exudates ooze out from the cut end (**Figure 2**).

3.1.2 Causative agent

Ralstonia solanacearum Yabuuchi causes bacterial wild disease in ginger. It is considered as the second most important destructive bacterial pathogens identified

Figure 2.
Yellowing of leaves and oozing of rhizome due to bacteria wilt disease (source: Vikaspedia).

to date. Four biotypes of *R. solanacearum* have been identified so far. Biotype III from India causes the wilt in ginger [11]. Biotype III of the bacterium cause slow wilt and biotype IV causes rapid wilting and death [12]. Biotype III is restricted to ginger plant and its weeds whereas biotype IV infects a wide host range including potato, tomato, eggplant, *Capsicum frutescens*, *Zinnia elegans* and *Physalis peruviana*. Biotype II is reported to infect only potato plants [13]. In Indonesia, the race 1 of biovar III is responsible for bacterial wilt in ginger [14]. Nematodes in the soil increase the incidence of wilt in ginger [15]. Nelson [16] observed that the host range of race 4 of biovar III of *R. solanacearum* is restricted to edible ginger. Twelve of 14 species of ginger belonging to Zingiberaceae and Costaceae are highly susceptible to all the strains of race 4 and susceptible plants wilted within 21 days [17].

Yu et al. [18] studied the genetic diversity of *R. solanacearum* strains collected from ginger plants growing in Hawaii island with amplified fragment length polymorphisms (AFLPs) showed a high degree of similarity between the strains with the value of 0.853. Strains from ginger in Hawaii showed less similarity for the strains from tomato (race 1) and heliconia (race 2) [18]. Kumar and Sarma [10] have characterized the isolates of *R. solanacearum* on the basis of their membrane protein pattern on SDS PAGE and serological detection (NCM-ELISA) using *R. solanacearum* specific antibodies. Detection of *R. solanacearum* from rhizomes and soil through PCR was developed for bacterial wilt [19–21]. Shan et al. [22] reported the complete genome sequence of *R. solanacearum* SD54, a race 4 biovar 4 (R4B4) strain from a diseased ginger plant.

3.1.3 Transmission

R. solanacearum is a soil-borne disease spreads from the soil by adhering to hands, boots, tools, vehicle tires and field equipment; through water during irrigation and rainfall; and through infected ginger rhizomes [23]. From the soil, this bacterium enters into the ginger plant through the roots and rhizomes *via* the openings where lateral roots emerge or through wounds caused while handling and by root-knot nematodes or parasitic insects [24]. The bacteria survive in the infected plant debris and as free-living in soil. Ginger crops on this infested soil severely affected and completely lost to the disease [16].

3.1.4 Protection

Difficulties are associated with controlling this pathogen due to its endophytic nature, survive in deep soil, travel along water, and its relationship with weeds. The available physical, chemical, biological, methods and cultural practices to manage this pathogen are discussed below.

3.1.4.1 Physical methods

Physical control methods like solarization and hot water treatment have proved to be effective against the pathogen. Rhizome solarization for 2–4 h on ginger seeds reduce the bacterial wilt (90–100%) at 120th day of planting, and further with discontinuous microwaving (10-s pulses) at 45°C reduces the wilt by 100% [25]. Tsang and Shintaku [26] reported that bacterial wilt pathogen is eliminated when the rhizome is exposed to heat for 30 min at 50°C and 45 min at 49°C, respectively. Exposing ginger seed pieces to hot air at 75% RH until their temperatures attained 49°C for 30 and 60 min and 50°C for 30 min, resulted in minimal injury to the hosts. More than 87% of the seed pieces germinated without adverse effect on growth.

3.1.4.2 Chemical methods

Treating seed rhizomes with emisan in addition to plantomycin for 30 min followed by three sprayings, first at 30 days after planting and others at an interval of 15 days, gave good protection against wilt disease [27]. Streptocyclin (20 g/100 l water) treatment on seed and drenching the soil with 0.2% copper oxychloride, protect the seed from the bacteria infection. Sinha et al. [28] observed that streptomycin and streptopenicillin are superior over the other antibiotics against the bacterial wilt pathogen.

3.1.4.3 Biological methods

Bacillus subtilis strain 1JN2, *Myroides odoratimimus* 3YW8, *B. amyloliquefaciens* 5YN8, and *Stenotrophomonas maltophilia* 2JW6 are used as biocontrol showed efficacies greater than 50% against bacterial wilt of ginger [29].

3.1.4.4 Cultural practices

The effective management of ginger against the pathogen is depends up on the various factors. Selecting the disease free seeds, sowing the disease free seed on disease free land based on previous history, following 4–5 years of crop rotation with non-solanaceous plants, planting on raised beds (help to avoid water stagnation during rainy season), giving thick mulching (to avoid weed growth and to conserve soil moisture), reduces the disease causing potential of the soil. Indrasenan et al. [30] suggested selection of healthy seed rhizomes, eradication of weeds and adoption of an effective crop rotation as control measures for the disease.

3.2 Bacterial soft rot

This is not considered as a major problem in ginger, but periodic outbreaks occur when ginger is planted in waterlogged soil [12]. The disease is more prevalent in rhizomes that have formed deep in the ground. The sections closer to the surface are generally healthy. Disease is not found in well-drained soils.

3.2.1 Symptoms

The disease causes a gradual softening of the rhizome tissue accompanied by an offensive odor.

3.2.2 Causative agent

Erwinia chrysanthemi is the only *Erwinia* species responsible soft-rot of ginger. High temperatures, saturated soils and injury during seed preparation all tend to exacerbate the disease.

3.2.3 Disease cycle

Ginger can have the bacteria either from the infected seed, or from direct inoculation, through wounds or natural openings. The bacteria started to feed liquids released from injured cells and multiply. Bacteria secrete pectolytic enzymes degrade and break the cells providing more food for the bacteria. Often the epidermis is left unscathed, keeping the rotten flesh contained within until a crack allows the ooze to leak out and infect others around it. The bacteria from the harvested infected plant to others placed with it and also through the insects.

3.2.4 Prevention

The most effective way to prevent this disease is simply keeping sanitary growing practices. It includes removing all plant debris from storage ware houses and disinfecting walls and floors with either formaldehyde or copper sulfate between harvests, maintaining low humidity and temperature of the storage facility with an adequate ventilation system. It also by planting in well-drained soils, rotating susceptible plants with non-susceptible plants.

4. Fungal diseases

Ginger plantation is majorly affected by deuteromycetous group of fungi cause variable symptoms [31]. In India, fungal diseases reduce the potential yield to a greater extent in field, storage and market and may cause losses of even more than 50% [32].

4.1 Soft rot/rhizome rot

Soft rot found in all the ginger growing countries, reported as the most dangerous and destructive disease of ginger which can reduce the production by 50–90%. Disease cause significant losses during warm and humid conditions. Butler on 1907, recorded this disease first time in Surat district of Gujarat, India [33].

4.1.1 Symptoms

This disease is prevalent in ginger crop throughout the growing period. Sprouts, roots, developing rhizome and collar region of the pseudo stem are highly prone to infection. Symptoms first appear on the aerial parts of the plant. Pathogen form watery and brown lesions in the collar region of the pseudo stem. Later the lesion enlarges, coalesce and cause the stem to rot and collapse [34]. In the old leaves, initially, yellowing (chlorosis) symptoms appear in the tips, which

then spread downward along the margin involving the rest of the leaf blade and, eventually, the leaf sheath. Later, chlorosis from the older leaves progress to younger leaves start developing a similar symptom progression until the entire plant dies [35] (**Figure 3**). The appearance of lesion in pseudo stem and chlorosis in the leaf indirectly show the sign of rhizome rot. Due to the infection, rhizomes appear soft, brown, water soaked, rotten, and decay gradually [34] (**Figure 3**). It is not like bacterial rots, the soft rot caused by fungus does not produce offensive odors.

4.1.2 Causative agent

Eleven species of *Pythium* are responsible for the soft rot diseases in ginger are identified so far. Among the 11 species, *P. myriotylum* and *P. aphanidermatum* cause severe damage in warm climates. *Fusarium* is another fungus reported to cause soft rot of ginger. Among different species of *Fusarium*, *F. oxysporum* causes decaying of ginger rhizomes [36].

Booster PCR method to detect *P. myriotylum* from the infected ginger rhizomes has been established [37]. Yella *et al.* [38] has developed a simple technique to produce oospores in *P. myriotylum*. Le et al. [39] developed PCR-RFLP based detection of isolates of *Pythium* by studying the genetic variation between *P. myriotylum*.

4.1.3 Disease cycle and epidemiology

It is seed and soil borne disease carried by two ways: (1) Through diseased rhizomes scales [40] and (2) through soil as oospores. *Pythium* survive in plant debris as perennating oospores, an important source for infections. The favorable conditions like wet soil conditions, high soil moisture and soil temperature influence the development of the oospore [41]. Severity of disease is influenced by high rainfall and when rhizomes planted in heavy clay soil with poor drainage. Temperature, 34°C is optimum for the germination of *P. aphanidermatum* and *P. myriotylum* spores.

4.1.4 Protection

Soft rot is a complex disease problem and various methods should be applied to combat the disease.

Figure 3.
Yellowing of leaves and rotting of rhizome due to soft rot disease (source: Vikaspedia).

4.1.4.1 Physical methods

Using disease-free seeds is an essential step to prevent the contamination of *Pythium* [41]. The various strategies like seed protection (biologically, physically or in combination), seed disinfestations (to control spores on the seed surface), seed disinfection (to eliminate pathogens living inside the cells) such as chemical infusion protect the seed from the infection as well as improve the germination of seeds [42]. In order to protect the plant from the persistence nature of the pathogen in soil, crop rotation with rice and corn (tolerant to pathogens of ginger) can be done to minimize the recurrence of pathogen in the next harvest [42–44]. Using soil with higher clay content and lower pH, suppress *P. zingiberum* and *F. oxysporum* [45]. Soil solarization with biological control methods is beneficial to plant growth and antagonistic to many pathogens like fungi, bacteria, nematodes, and arthropods. Phytosanitation is needed when the symptoms of soft rot appear in the ginger plantations.

4.1.4.2 Chemical methods

Pythium spp. is soil borne pathogen survive in the soil for a long period [46]. It is very difficult to control this. Treating the rhizomes with mancozeb, ziram, guazatine, propineb and copper oxychloride for 30 min effectively control this pathogen [47, 48]. Treating seed with Ridomil MZ (1.25 g/L) increase the survival of rhizomes (about 30%) in comparison to hot water treatment (at 51°C for 30 min) [49]. Rajan et al. [50] reported that rhizomes are protected from *Pythium* while treating the seed with Fytolan (copper oxychloride) (0.2%), Ridomil (500 ppm), Bavistin (carbendazim) (0.2%) and Thimet. Application of fungicides such as metalaxyl, Ridomil, Maxam XL (fludioxonil) and Proplant (propyl carbamate hydrochloride) on seed give significantly better result for *P. myriotylum* than carbendzim alone [51]. Soil drenching with Zineb, captafol, methyl bromide, mercuric chloride, thiram, phenyl mercury acetate, copper oxide and mancozeb protect the ginger crop from soft rot disease [52]. Application of Metalaxyl in soil is effective to control the soft rot [53]. Treating seed with a mixture of metalaxyl and captafol and treating the soil with the same chemicals after 3 months of planting controlled the disease [54]. Metalaxyl formulations (Ridomil 5G and Apron 35 WS) in soil and treating the seed gave very good protection against soft rot [55]. Srivastava [56] effectively managed *P. aphanidermatum* by drenching Zineb or Mancozeb in the soil after treating the rhizome with carbendazim. Treating with 1% formaldehyde and then planting the ginger under shade had lower incidence (19.4%) of soft rot as without shade (41.3%) [57].

4.1.4.3 Biological methods

Trichoderma spp. is the best biocontrol agents for soft rot. Non-volatile and volatile compounds from *T. viride* inhibit the growth of *P. myriotylum* [58]. Similarly, *T. harzianum* and *T. saturnisporum* also showed strong antagonism against *P. splendens* [59]. Several rhizobacteria were reported to possess strong antagonistic activity on *P. myriotylum* growth [60]. Growth of *P. aphanidermatum* on potato dextrose agar (PDA) was completely inhibited when it amended with onion and garlic extracts at 5 and 7.5% (v/v), respectively [61]. Rakesh et al. [62] showed inhibition of *P. aphanidermatum* with treatment of fresh and stored cow urine at a concentration of 20% (v/v). Application of *Boerhavia diffusa* leaves and seed powder of *Azadirachta indica* in the soil before the planting reduces the infection [63]. Adding neem cake in the soil also found to suppress *P. aphanidermatum* [64]. Application of *Jeevatu* based organic liquid manure in the field, control the soft rot and the spreading of

this disease [65]. Coating the seeds with *Trichoderma* spp. reduce the soft rot, 2–3 times less than that of the untreated control [66]. Reduction of soft rot occurred when seed was first disinfested with HOCl (1%) followed by soaking in *Trichoderma* spp. and followed by three applications of talc-based formulation (3×10^6 CFU/g) to the soil at 15 day intervals from the time of planting [67]. Treating with *T. harzianum*, *Glomus mosseae* and fluorescent Pseudomonad strain G4 together inhibit the infection up to 10% than treating individually with 30, 43, and 50% infection, respectively [68].

Treating with *Burkholderia cepacia* and *T. harzianum* showed production efficiency of 84% with reduction of disease incidence of 79.7% [67]. Extracts from *Jacaranda mimosifolia* and *Moringa oleifera* gave the best inhibitory effect of 27.7% against *P. aphanidermatum* [69].

4.1.4.4 Cultural practices

Cultural practices such as seed selection, crop rotation, organic amendment, tillage, drainage and quarantine practicing in ginger plantation not only control the disease but also limit the spread of *Pythium* spp. [70].

Planting the disease free rhizomes is the best method to manage the disease [71]. Harvey and Lawrence [72] believed that crop rotations reduce *Pythium* spp. populations. Rames et al. [73] found that fungal and bacterial populations were significantly greater in soil when pasture grass (*Digitaria eriantha* subsp. *pentzii*) grown continuously than the soil is treated with fumigant or left as bare fallow.

Addition of organic matter from various plants, oil seed cakes and neem cake, reduce the incidence of soft rot [74, 75]. Mixing organic matter with poultry manure and sawdust enriched the soil microbial populations in the ginger growing soils [73] and enhance the soil carbon levels and water infiltration rates, support the growth and yield of ginger [51, 76]. Kadam et al. [77] reported that neem seed cake with least average mortality (20.3%) followed by poultry manure (22.7). Kumar et al. [78] also reported that *Schima wallichii* and *Datura* spp. were the best mulches with respect to inhibit soft rot caused by *P. aphanidermatum*. Smith and Abbas [51] recommended proper water drainage in the ginger filed to inhibit the *Pythium* spp. zoospores germination and spreading.

4.1.4.5 Host resistance

Identifying *Pythium* resistant ginger is an ideal for effective soft rot disease management. Indrasenan and Paily [79] identified Maran cultivar resistant against *P. aphanidermatum*. Setty et al. [80] identified Supraba and Himachal Pradesh cultivars showing less than 3% soft rot incidence. Senapati and Sugata [81] found one resistant cultivar and eight others with moderate resistance while screening 134 ginger varieties available in Koraput, Orissa, India. Kavita and Thomas [82] reported that *Zingiber zerumbet* accession is suitable candidates to obtain soft rot resistance for ginger. Screening 650 ginger accessions by Bhai et al. [83] found that only 7% of the accessions have the relative resistance to the *Pythium* sp.

4.1.4.6 Integrated management

Following a single approach is not ideal way to a have soft rot resistant. It is an important to have a multiple approach. Smith and Abbas [51] focused on cultural practices with strict quarantine procedure to manage the disease. Soil solarization

with fungicides effectively reduces the *P. myriotylum* [84]. Treating rhizomes with Ridomil MZ (metalaxyl and mancozeb) and soil drenching with Thimet (Phorate) and Ridomil MZ after planting gave the best control of *P. myriotylum*. Seed solarization at 47°C for 30 min within a polyethylene sheet is ideal for *Pythium* sp. disinfestations [85]. Having soil solarization for longer periods reduces *Pythium* spp. populations with lower disease incidence [86]. Applications of bioagents such as *T. harzianum*, *P. fluorescens* and *B. subtilis* together reduce the disease than individually on the rhizomes (8.64%) as well as on tillers (12.50%) [87]. Rhizome treatment with copper oxychloride followed by neem extract, suppresses the soft rot disease [88]. Rhizome treatment in hot water at 47°C for 30 min and drenching of soil *T. harzianum*, followed by mancozeb are most effective in inhibiting *Pythium* sp. as well as improve the yield of ginger [89].

4.2 Yellows/wet rot

Yellows disease is serious problems of ginger causes stem and rhizome rot. It is wide spread and prevailed in warm and humid environmental conditions. It was first described by Simmonds [90] from Queensland. Later this disease was reported from Hawaii [91] and India [92].

4.2.1 Symptoms

Yellowing starts on the margins of the lower leaves which gradually spreads and cover the entire leaves. Later, the yellowing diffuse to older leaves. Old leaves dry first and then younger leaves. The affected plants wilt and dry up but do not fall on the ground in contrast to soft rot and bacterial wilt. Infected pseudo stem comes off from the rhizome with a gentle pull. Rhizomes become soft and watery with a creamy discoloration of the vascular system and cortical rot. Plants may show a premature drooping, wilting, yellowing, drying in patches or in whole bed and show stunting. Rotting of roots is common. Mycelial growth in the form of white, peach or buff colored cushions can be seen on the surface of rhizomes [93].

4.2.2 Causative agent

Fusarium oxysporum f.sp. *zingiberi* Trujillo is a causative agent for yellow disease [94]. Other species of *Fusarium* such as *F. solani* (Mart.) Sacc., *F. equiseti* (Corda) Sacc., *F. graminearum* Schwabe, *F. moniliforme* Sheld and some unidentified *Fusarium* spp. were also reported to be associated with yellow disease ([95–97]. *F. solani* is the second most important species [96, 98]. Isolates of *F. oxysporum* f. sp. *zingiberi* differed in their aggressiveness [99].

Genetic variation of *F. oxysporum* f.sp. *zingiberi* isolates with DNA amplification fingerprinting (DAF) segregated the isolates into three haplotypes based on 17 polymorphic bands generated with five primers [100]. Two haplotypes showed very little genetic variation (98.6% similarity), whereas the third haplotype 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. [101]. Molecular variability revealed 0 to 80% variation among 19

isolates and they were grouped into two different major groups each comprising of 10 and 9 isolates, respectively.

4.2.3 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 [102]. 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 15–30°C is favorable (the optimum being 23–29°C) with very high humidity and continuous presence of free water [103]. Maximum disease incidence occurred when soil temperature ranged from 24 to 25°C and the soil moisture from 25 to 30% [104].

4.2.4 Protection

4.2.4.1 Chemical methods

The various chemicals have been shown promising result against the pathogen [105–109]. So far fungicides like Bavistin 50WP, Ridomil Gold MZ-72, Captan, Dithane M-45, copper oxychloride and Bordeaux mixture are found to be effective against the disease [110, 111].

4.2.4.2 Biological methods

Microorganisms like *Trichoderma harzianum* and *Gliocladium virens* [112], *T. viride* and *T. harzianum* [113], *T. viride* [114] inhibits *F. oxysporum*. Among six *Streptomyces* species, SSC-MB-02 was effective against *F. oxysporum* [115]. Treatment of *Bacillus cepacia* and *T. harzianum*, increased the production of rhizome as well as protect the plant from yellow disease [59]. Talc-based formulations with rhizobacteria strain XXBC-TN (*Bacillus subtilis*) and a mixture of S2BC-1 (*B. subtilis*) and TEPF-Sungal (*Burkholderia cepacia*), inhibit *F. oxysporum* and *F. solani*. Treatment resulted in increased rhizome production with reduced yellows. Increased protection is due to increase in defense enzymes such as chitinase, β-1,3-glucanase and polyphenol oxidase and promote plant growth and rhizome [67].

Among 14 plant extracts, the plant extract of *Ferula asafoetida* (68.51%) has high protection against *Fusarium*, followed by *Ocimum* leaf extract (60.16%) [116]. Leaf extracts of *Swietenia macrophylla* King, *Azadirachta indica* A. Juss., *Hyptis suaveolens* (L.) Poit., *Polyalthia longifolia* (Sonn.) Thw., *Boerhavia repens* L. var. *diffusa* (L.) Hook. and *Tithonia diversifolia* A. Gray showed 100% resistance against *F. solani* [117]. Among the various nanoparticles synthesized from plants, a sulfur nanoparticle has high inhibitory effect on *F. oxysporum* [118]. Further, sulfur nanoparticles with fungicides like bavistin, ridomil gold, sunflex and streptocycline were evaluated for the inhibition of the fungus showed that sulfur nanoparticles with bavistin with high inhibition.

4.2.4.3 Cultural practices

The disease is spread mainly through contaminated rhizomes. Planting healthy seed rhizomes is the best way to avoid this disease [71, 119]. Applying organic manure, tillage and crop rotation reduces the yellows disease [76]. Planting the seed

rhizome (size of 50–75 g) with spacing of 25 × 30 cm is ideal for good yield and lower disease incidence [120].

4.2.4.4 Host resistance

Developing a *Fusarium* resistant variety would be ideal for effective yellows disease management. Two cultivars: SG 666 [121] and Kerala local [122] are reported to have disease resistant. The resistance is due to the presence of a resistance (R) gene of CC–NBS–LRR [123].

4.2.4.5 Integrated management

The integrated approach like treating the seed and well as in soil with fungicides mancozeb and carbendazim and biocontrol agents like *T. harzianum, T. hamatum and G. virens* reduces the incidence of disease [124]. Hasnat et al. [110] reported lowest yellows disease while treating with Ridomil Gold along with poultry waste, Bavistin 50WP, Dithane M-45 and saw dust in the soil at 240th days of planting.

4.3 Leaf spot

Leaf spot disease is becoming increasingly important in many places of India due to severe leaf rot and blight it causes. Ramakrishnan [125] reported this disease first time from Godavari district of Andhra Pradesh and Malabar area of Kerala.

4.3.1 Symptoms

On the young leaves, small spindle to oval to elongated spots size of 1–10 mm × 0.5–4 mm appears. Later, the spots developed as white papery center and dark brown margins surrounded by yellow halos [125]. The spots increase in size and coalesce to form larger lesions which lead to the reduction of effective photosynthetic area on the leaves. The affected leaves become shredded and may suffer extensive desiccation (**Figure 4**). Symptoms appear first on younger leaves. As the plants put forth fresh leaves, these get infected subsequently.

4.3.2 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 78–150 μm in diameter. On standard media, the fungus forms pycnidia having 100–270 μm diameter bearing hyaline, unicellular, oblong, big guttulate spores measuring 3.7–7.4 × 1.2–2.5 μm [125].

4.3.3 Disease cycle and epidemiology

Primarily, disease is spread from the debris of infected plants and from the infected seeds. Under the laboratory condition, pycnidiospores and mycelia of fungi alive for 14 months [126] and spores remain viable in soil even at 25 cm depth for 6 months. Pycnidia of *Phyllosticta* survive in the leaf debris even during the summer. Mycelium of *Phyllosticta* grows at 25.0–27.5°C with maximum and minimum of 32.5 and 10.0°C, respectively. The mycelial growth was inhibited at 5 and 35°C [127].

During the rainfall, the dispersal of spore occurred. Higher precipitation along with high wind give greater impact on dispersion of spores to many leaves which are

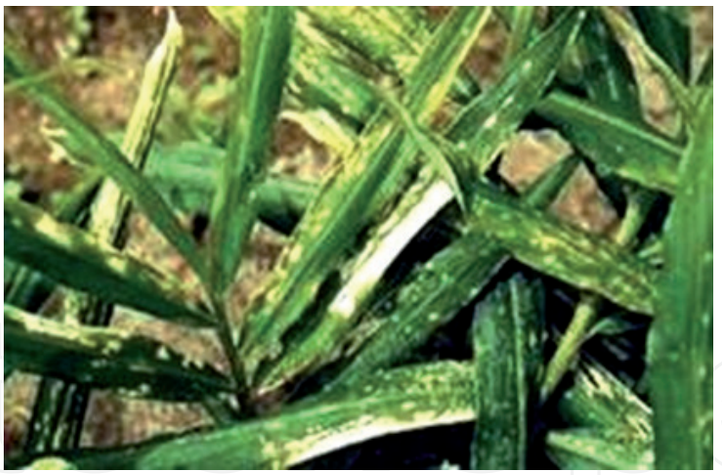

Figure 4.
Symptoms of spot on ginger leaf (source: Vikaspedia).

in longer distances [128]. Factors like air temperature, relative humidity and rainfall influence the incidence of disease to an extent of 85.5% [129]. Six to seven months old plants are prone to infection of *Phyllosticta* and 2 weeks old leaves are most susceptible. Temperature of 23–28°C with intermittent rain favored the occurrence of this disease. Senapati et al. [130] observed that incidence of leaf spot disease was less for the plants grown under partial shade or as intercrop in coconut. Incidence of disease is higher and yield of rhizome is reduced when the ginger is cultivated continuously without the rotation of the crop [131].

4.3.4 Protection

4.3.4.1 Chemical methods

Treating the plant with Bordeaux mixture, zineb and maneb are effective in treating the disease [132]. Grech and Frean [133] observed that spraying mixture of benomyl (0.1%), mancozeb (0.2%) and soluble boron (0.1%) and iprodione (0.2%) reduces the production of disease. Highest reduction of the incidence of disease is observed with spraying chlorothalonil [127]. Verma and Vyas [134] observed higher protection while spraying carbendazim (0.15%) and mancozeb (0.25%) and due to this higher yield also obtained. Increased yield of rhizome and decrease disease incidence was found while treating the rhizome and doing foliar spraying with Bordeaux mixture (1%), Companion (0.2%), Indofil M-45 (0.25%), Unilax (0.2%) and Baycor (0.05%) [129].

4.3.4.2 Cultural practices

Growing the crop under the partial shade reduce the severity of Phyllosticta leaf spot. Growing the ginger under the partial shade of mandarin orange increase the growth of the plant and reduce the disease intensity [135]. Reduction of leaf spot and sun burn on leaves occurred while growing plants under shade have increased the number of tillers per clump recommends the growing of ginger in partial shade to avoid the fungicidal spray [107].

4.3.4.3 Host resistance

None of the 18 cultivars tested in Karnataka, India were resistant to *Phyllosticta* leaf spot [136]. However, the cultivars Narasapatom, Tura, Nadia, Tetraploid and

Thingpani were classed as moderately resistant with a disease index less than 5%. In Himachal Pradesh, India none of the tested material of ginger was rated resistant to *P. zingiberi,* however, eight lines showed moderate resistance [137]. Different workers obtained variable results and none of the tested cultivars showed high degree of resistance [137, 138]. Nageshwar et al. [138] 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 further 42 were moderately tolerant. Senapati et al. [130] found that PGS-16, PGS-17 and Anamica as moderately resistant out of 135 ginger cultivars tested.

4.4 Storage rots

Post-harvest losses in ginger are a serious concern. The post-harvest losses are affected by various biotic and abiotic causes. Rhizomes are stored for seed and commercial purpose. During storage, rhizomes soft are affected by fungi [71] and bacteria.

4.4.1 Symptoms

Fungal mycelia discolored the surface of rhizome accompanied with dry rotting and decaying (**Figure 5**).

4.4.2 Causal organism

Fungus like *F. oxysporum* Schlechtend ex Fr., *P. deliense* Meurs and *P. myriotylum* Drechs. [139], *Geotrichum candidum* Link [140], *Aspergillus flavus* Link ex. Fr. [141], *Cladosporium tenuissimum, Gliocladium roseum* Bainer, *Graphium album* (Corda) Sacc., *Mucor racemosus* Fresen., *Stachybotrys sansevieriae, Thanatephorus cucumeris* (Frank) donk and *Verticillium chlamydosporium* Goddard [142]. *Pythium ultimum, Fusarium oxysporum* and *Verticillium chlamydosporium* are responsible for storage rot. 85% of the rhizomes possess the mold growth of *Penicillium brevicompactum* [143].

Pathogenicity test is available for *Acremonium murorum, Acrostalagmus luteoalbus, Fusarium* sp., *F. oxysporum, Lasiodiplodia theobromae* and *Sclerotium rolfsii* associated storage rot [36].

Figure 5.
Rotting of rhizome due to the infestation of F. oxysporum (source: pestnet.org).

4.4.3 Protection

4.4.3.1 Chemical methods

Incidence of storage rots is reduced while the rhizome is treated with benomyl (750 ppm) and/or gibberellic acid (150 ppm) before the storage [144]. Dipping the rhizomes with imazalil or prochloraz (0.8 g a.i/liter) and then storing at 10°C gave good protection against *Botryodiplodia, Aspergillus, Diplodia, Fusarium, Rhizoctonia* and *Pythium* [145]. Mancozeb and carbendazim together treatment on ginger rhizomes protect the rhizomes from the rot [146, 147]. Application of 0.3% Ridomil MZ during the storage condition causes the low infection [107]. Steeping of rhizomes in carbendazim (0.1%) for 60 min before storage also controlled storage rots and reduced the disease incidence from 71.4 to 18.2% [112].

Treating the rhizome with aureofungin (0.02%) and Benomyl (0.2%) before the Storage, control the disease [148]. Immersing the rhizome in carbendazim (0.1%) for 60 min reduce the disease from 71.4 to 18.2% [112].

Sharma et al. [149] reported that mancozeb fungicides compared to carbendazim is best chemical to protect the rhizome for the longer period from the fungus infection. The presence of Mancozeb was observed at 120th days of storage. But the health point of human, carbendazim treated rhizomes is safe than mancozeb treated rhizome Pre-storage treatment of rhizome with Topsin-M and Bavistin (each at 0.2% concentration for 60 min) reduce the appearance of disease on rhizome, increase the weight of rhizome, surface shriveling and sprouting of rhizomes [31].

4.4.3.2 Cultural practices

Storing the rhizomes in lower temperature avoid weight loss, increase the sprouting but with higher risk of infection when comparing with storage at room temperature. Rhizomes packed in PVC film preserve the weight but has high chance of disease [150]. Dipping the rhizomes in the *Allium sativum* extract or immersing in a suspension of *P. fluorescens* and *T. harzianum (*0.5% for 30 min) before storage, reduce disease incidence [151]. Similarly, dipping in garlic extract (20% w/v for 30 min) also reduces the disease incidence [152].

5. Nematodes causing disease in ginger

Meloidogyne sp., *Radopholus similis* and *Pratylenchus* sp. are major nematodes cause significant loss to ginger.

5.1 Root-knot

Disease causes 74% of reduction in rhizome weight. Nematode infections aggregate the fungus and bacterial infection.

5.1.1 Symptoms

Nematode feeds rhizomes, roots and base of the pseudo stems. In the root, it causes the swellings or knots. The symptoms of root knot are very similar to root gall. Irregular round galls and spindle-shaped enlargements appear on the tap and side roots. Diameter of gall is 3.3 cm. Infested plants show stunting, chlorosis and marginal necrosis of leaves. Roots are often stunted and deformed. Roots and rhizomes exhibit galling and rotting. Cortex of the rhizomes becomes lumpy and

cracked during the severe infection. During the infection process, female nematode attains the maturity and emerges from the gall by breaking the epidermis of the rhizome which gives corky in appearance for rhizome. Small, circular, water soaked, slightly brown lesions with mature females of the nematode (**Figure 6**) below the epidermis of the rhizomes are quite numerous in severely infected rhizomes. The lesions serve as entry points for bacteria and fungi, invade, extend the injury into other tissues and destroy the rhizomes.

5.1.2 Causative agent

It is caused by the plant parasitic nematode, *Meloidogyne* sp. They are sedentary, endoparasitic and produce gall. Due to this, crop is severely affected without destroying the rhizomes. Nematode does not produce galls in ginger rhizomes as it occurred in other plants but it invades immature tissues. The infective stage of the nematode is the second stage juveniles, which have lightly sclerotized cephalic framework.

5.1.3 Protection

Crop rotation with non-hosts such as graminaceous and a few antagonistic crops for 1 or 2 years reduces the nematode. Crop rotation with groundnut-mustard was effective in reducing the population of *M. incognita*. Decomposition of soil amendments (dry or green crop residues, oil cakes, meals, sawdust, FYM etc.) in the nematode infested field not only reduce the nematode but also alter the physical, chemical and biotic conditions of the soil which improve the plant growth. Application of nematicides like Thimet or Phorate (12–15 kg/ha) followed by light irrigation before the sowing reduce the incidence of nematode. Biocontrol agents like *Paecilomyces lilacinus*, *Pasteuria penetrans* and *Pseudomonas fluorescens* are effective against *M. incognita*.

5.2 Burrowing nematode

5.2.1 Symptoms

Infected plants exhibits stunting, reduced vigor and tillering. Top most leaves become chlorotic with scorched tips. Infected plants show yellow leaves with less

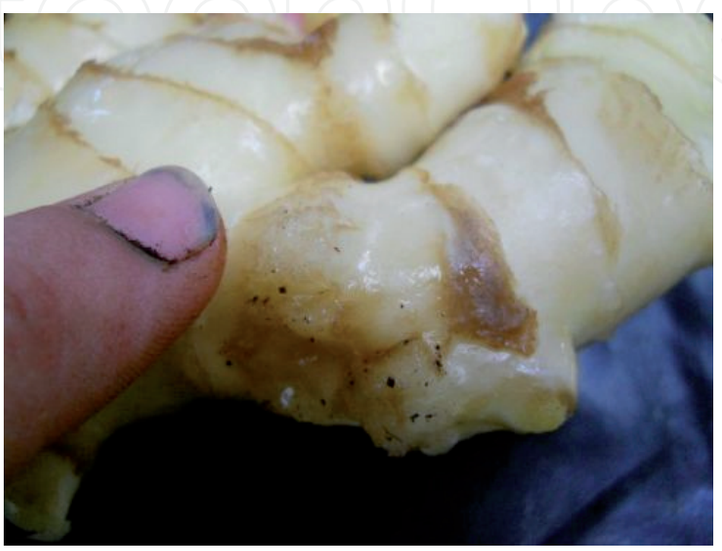

Figure 6.
Brown lesions on the root knot nematode infected rhizome (below) (source: http://www.eastbranchginger.com).

number of shoots and stunted growth. Infected rhizomes possess small, water-soaked shallow lesions which later turn brown. These small lesions merge and rot the rhizome.

5.2.2 Causative agent

The causative agent is Radopholus similis.

5.2.3 Protection

Crop rotation with taro and cassava, applying large qualities of poultry manure, dipping seed in hot water at 51°C for 10 min before sowing, reduce the infection of *Radopholus similis*. Chemical control is not recommended for the control of this disease.

5.3 Lesion nematodes

5.3.1 Symptoms

It affects roots and rhizome severely. It causes extensive damage to cortical tissues of root. Infestation of the nematodes causes yellowing of leaves and dry rot on rhizome. Dark brown necrotic lesion is observed in nematode infected rhizomes. The fingers are severely affected by this nematode.

5.3.2 Causative agent

Several species of *Pratylenchus* namely *P. brachyurus, P. coffeae, P. indicus* and *P. zeae* are infecting ginger.

6. Minor diseases

Some diseases of minor importance have also been reported on ginger like Cercospora leaf spot caused by *Cercospora zingibericola* [153], anthracnose caused by *Colletotrichum zingiberis* [154], *Pyricularia* leaf spot caused by *Pyricularia zingiberi* [155], basal rot caused by *Sclerotium rolfsii* [92, 156] and *Septoria* leaf spot caused by *Septoria zingiberis* [157].

7. Nonparasitic diseases

7.1 Sunburn

Young ginger plants are very susceptible to sunburn when the temperatures exceed 90°F due to high light intensity. Mild sunburn affects only the leaves, but acute sunburn damages the entire shoot [158]. Drought and lack of water may cause the same effects as sunburn.

7.2 Lime-induced chlorosis

Excessive liming or abundant coral sand in the soil may cause yellowing of the blades and poor growth.

8. Disease caused by arthropods

Insect involved in spreading the pathogens responsible for the diseases and also involved in damaging the foliage and rhizomes.

8.1 Shoot borer

8.1.1 Symptoms

The moth lay eggs on the growing bud, petiole or leaf of the young plants. Caterpillars bore through the central shoots, feed the growing buds resulting in withered and dried shoot referred to as "Dead Heart". The presence of a bore hole on the pseudo stem through which frass is extruded and withered and yellow central shoot is a characteristic symptom of pest infestation.

8.1.2 Causative pest

Shoot borer caused by *Dichocoris punctiferalis* (**Figure 7**).

8.1.3 Management

Ginger is protected from the shoot borer by collecting the entire emerged adult, destroying and by installing light trap for adult mass trapping, destroying infested plant and by chemical application of Metarhizium and treating with *Beauveria bassiana*.

8.2 White grub

8.2.1 Symptoms

It feeds the base of the pseudo stem, roots and newly formed rhizomes. Pest infestation leads to yellowing of the leaves. It make large hole in the rhizome and reduce its market value. The entire crop may be lost in severely infested plantations. The adults are dark brown beetles and measures about 2.5 mm x 1.5 mm in size. The grubs are creamy white and live in soil.

Figure 7.
Dichocoris punctiferalis cause shoot borer in the ginger (source: Ref. [159]).

8.2.2 Causative pest

White grub caused by *Holotrichia* spp. (**Figure 8**).

8.2.3 Management

Leaving the land fallow for 2 consecutive years reduce the pest population. Growing of resistant crops such as sunflower also checks the buildup of grub population. Sowing of trap crops such as sorghum, maize and onion reduce the white grub infestation**.** Application of *Beauveria bassiana* or *Metarhizium anisopliae* with vermicompost (5 g/kg) or drenching the soil with these entomopathogenic fungi (5 g/l) reduces the pest. Two sprays of neem oil 0.15 EC (1500 ppm) at 3 ml/l at 15 days interval is found to be effective.

8.3 Leaf roller

8.3.1 Symptoms

It is an olive green caterpillar with a distinct black head. It folds the leaves and stays inside the fold and defoliates the leaves from the tip and margins. When one portion is complete, it moves and makes another fold.

8.3.2 Causal organism

Leaf roller is caused by *Udaspes folus* (**Figure 9**).

8.3.3 Protection

Maintain the field sanitation and application of *Bacillus thuringiensis* (1–2 g/l) prevents the leaf roller.

8.4 Chinese rose beetle

The Chinese rose beetle, *Adoretus sinicus* (Burm.), (**Figure 10**) is pale reddish-brown and has nocturnal feeding habits. The damage on the foliage is characteristic, being peppered or shot with holes, or more or less skeletonized. The eggs, which are

Figure 8.
Holotrichia responsible for white grub in ginger (source: Ref. [159]).

Figure 9.
Udaspes folus causes leaf roller in ginger (source: Ref. [159]).

Figure 10.
Chinese rose beetle (source: Vikaspedia).

laid in the soil, hatch in about 4 days, and the entire life cycle from egg to adult takes about 6–7 weeks. The larvae apparently feed on decaying plant material; pupation occurs in the soil. During the daytime, adults are usually found hiding in loose soil or among dead leaves. This pest is prevalent in the field at low elevations [160].

8.4.1 Protection

Chinese rose beetles are repelled by bright light and so shining the plants with bright light deter them from feeding; covering young plants with e.g. floating row covers can help to protect plants until they are old enough to withstand attacks by the beetle.

8.5 Fullers rose beetle

The Fullers rose beetle, *Pantomorus godmani* (Crotch), feeds ginger leaves [160]. The adults are 7–9 mm long and more prevalent at higher elevations, apparently requiring cooler temperatures for survival.

8.6 Grasshoppers

Ginger leaves are occasionally damaged by grasshoppers. This occurs at times when there is a high population incidence of these insects [160].

8.7 Scavenger flies

These flies breed in decayed plant tissues of ginger. These include *Eumerus marginatus* (Grims) (syrphid fly), *Euxesta quadrivittata* (Macq.) (otitid scavenger fly) and sciarid gnat fly. The importance of these insects in ginger production remains to be determined. Syrphid fly may be a threat to ginger when the crop becomes dormant. Sometime, the eggs laid on the decaying flower stalk, develop into larva, reaches the rhizome and reduce the quality of rhizome [160].

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