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Management of Sclerotinia Sclerotiorum, Fungal Soil Borne Pathogen

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A B S T R A C T

Sclerotinia sclerotiorum (Lib.) de Bary is one of the most important and devastating soil borne pathogens with wide-spread distribution which perpetuates by formation of Sclerotia and remain viable upto 4-5 years in soil. It is one of the most omnipresent, non-specific, ubiquitous necrotrophic pathogen that attacks a wide range of cultivated and wild plant species including canola (oilseed rape), mustard, alfalfa, soybean, field-bean, lentil, field pea and sunflower. It results in damage of the plant tissue, followed by cell death and soft rot or white mold of the crop. *S. sclerotiorum* infect 64 plant families, 225 genera, 361 species, 22 other cultivars etc. with a total of 383 species of plant kingdom. It causes considerable damage in Pepper, Cauliflower, Turnip, Kiwi, Lettuce, French bean, Melon, Potato, Neem, Soybean, Sunflower, Pear, Carrot, Brinjal, Tomato etc. In the recent years, much emphasis has been given on biological control of plant diseases, which is also the recent live-wire of Integrated Disease Management (IDM) strategies. The efficiency of bio-control agent is increased to a great extent when integrated with reduced amount of fungicides as it can stress and weaken the pathogen and render their propagules more susceptible to subsequent attack by antagonist apart from reducing the probability of development of fungicide resistance.

Keywords: Integrated Disease Management, *Sclerotinia sclerotiorum,* Fungal, Soil Borne

Introduction

Sclerotinia sclerotiorum is omnipresent and has a very wide host range and causes economic losses in crops such as oilseeds, pulses, forage legumes, vegetables and ornamentals. There was severe yield loss due to the infection of Sclerotinia in vegetables such as lettuce, celery, potato and cabbage (Purdy, 1979). Average crop loss of dry bean due to *S. sclerotiorum* was 30 per cent with individual field loss of 92 per cent in Nebraska (Schwartz and Steadman, 1989). Crop loss of soybean in Canada during 2000 due to Sclerotinia stem rot was estimated as 0.9 per cent accounting for $7.2 million (Anderson and Tenuta, 2001). White mold of soybean is a devastating disease in Canada, northern US, Argentina and China. The annual loss in Canada was 6 million dollars (Simmonds et al., 2001). In central Manitoba, 76 per cent and 52 per cent of canola fields were affected during 2001 and 2002 respectively (Duncan, 2003). Yield loss in Manitoba and North Dakota due to Sclerotinia rot was around $16,768,955 during 2001 (Lamey et al., 2001). An annual loss of $15 million was realized by the sunflower producers in United States due to Sclerotinia infection (Sayler, 2003).

Three principal modes of infection by *S. sclerotiorum* have been identified, infection may result from infection at the

stem base from mycelia that develops from germinating Sclerotia (Purdy, 1958), germination and penetration of ascospores at wound sites (Abawi and Grogan, 1975) and the germination of ascospores on senescent flowers or leaves and organic matter in contact with the host (Newton and Sequeira, 1972). Sclerotinia infection on above ground plant parts is due to ascorporic inoculums resulting from carpogenic germination of Sclerotia (Cook et al., 1975). According to environmental conditions, Sclerotia either undergo myceliogenic or carpogenic germination. Myceliogenic germination results in the production of mycelial strands from Sclerotia that infect young stems directly (Agrios, 1997). Sclerotia of the fungus are of white cottony septate mycelium which later turn into black hard bodies and measure 2-12 mm, the average being 6 mm (Mehta et al., 1946; Saha and Singh, 1988). Sclerotia are composed of pseudoparenchyma in which interior cells are hyaline and the outer walls are thick dark brown in colour. Upon establishment the fungus deploys two main pathogenicity determinants, the secretion of oxalic acid and a battery of acidic lytic enzymes released by advancing mycelium (Abawi and Grogan, 1979; Adams and Ayers, 1979; Bolland and Hall, 1998).

Keates et al. (1998) reported that extracts of Sclerotia of *S. sclerotiorum* contained 2 active constituents, D-glyceropent-2-enono-1,4-lactone (D-erythroascorbic acid), 5- O-(alpha-D-galactopyranosyl)-D-glycero-pent-2-enono-1,4- lactone. Sharma et al.(2001) reported the activities of pectinases and pectin methylesterase enzymes in culture medium of *S. sclerotiorum* which were inhibited by phenolic compounds (m-coumaric, homovanillic and protocatechuic acid) present in the culture medium. By comparing with the retention time of the standards, phenolic compounds can be identified (Sarma et al., 2002). Singh et al. (2004) analyzed ethyl acetate fraction of exudate of *S. sclerotiorum* with the help of High Performance Liquid Chromatography (HPLC) and showed that it consisted of tannic, gallic, ferulic and cinnamic acids along with many other unidentified compounds and the exudate showed antifungal activity against some parasitic as well as saprophytic fungi. HPLC is a highly sensitive method for detection, identification and quantification of any chemical in a particular sample using ultraviolet and visible absorbance (Hanachi & Golkho, 2009).

Under favourable conditions, the Sclerotia germinate and cause infection to the host plants. Infection of susceptible host plant occurs either by mycelium from eruptive germination of Sclerotia in soil (Blodgett, 1946; Purdy, 1979). Infection of *S. sclerotiorum* may either be via Sclerotia or ascospores, the epidemiology of these two types of infection is different and the effects of weather on their incidence and development differs considerably (Abawi and Grogan, 1979). The optimum temperature range for sexual germination of *S. sclerotiorum* has been reported by several workers to be 10 to 25°C (Hawthorne, 1976; Saito, 1977; Willetts and Wrong, 1980). Temperature 20-25°C and R.H of 95-100 % found to be congenital for *S. sclerotiorum* to develop disease (Sharma and Sharma, 1985). The fungus grows best at 15-25°C but can also grow and produce Sclerotia between the range of 0-3°C showing its viability at almost every temperature during the cropping season (Saha and singh, 1988). Boland and Hall (1988) concluded that the development of the crop, reproduction of the pathogen and initiation of disease and optimum environmental factors are all interrelated. It is the development of a closed crop canopy that provides extended periods of high soil moisture and is a prerequisite for carpogenic germination and apothecial development. Generally, prolonged cool and wet conditions are the primary factors influencing disease development via ascospores.To germinate apothecia, Sclerotia requires moist soil conditions for 1 to 2 weeks, and cool temperatures from 11 to 20°C (Schwartz and Steadman, 1978). The carpogenic germination of apothecia from Sclerotia, the germination of ascospores, and severity of infection within host tissue is influenced by moisture and temperature (Schwartz and Steadman, 1989; Phillips, 1994). Singh and Tripathy (1997) found that initial infection in field initiates either with air-borne ascospores or soil-borne mycelium originating from Sclerotia left in the field depending on the available permissive conditions. Stipes are produced over a wide range of temperature (5 to 25°C) but those produced at below 10°C or above 20°C frequently fail to differentiate into apothecia or their growth remains abnormal. At low RH's (25%), ascospores were less efficient than mycelia in causing disease (Harikrishnan and Del Rio, 2006). The greatest number of apothecia were produced at 20°C with a high light intensity, apothecia at a high light intensity were larger in size than those produced at a low light intensity (Dael Desiree Visser, 2007).

S. sclerotiorum secretes multiple pathogenicity factors. Degradation of plant cell wall, its components and tissue maceration occur by the concerted action of several extracellular lytic enzymes. Oxalic Acid (OA) exerts a toxic effect on the host tissue by acidifying the immediate environment and by sequestering calcium in the middle lamellae leading to loss of plant tissue integrity (Bateman and Beer, 1965). Reduction in extracellular pH, activate the production of cell wall degrading enzymes (Marciano et al., 1983). In conjunction, plant cell wall-degrading enzymes, including cellulolytic and pectinolytic enzymes, cause maceration of plant tissues, necrosis followed by plant death (Collmer and Keen, 1986). Oxalic acid is a recognized virulence factor produced by several phytopathogenic fungi, including *S. sclerotiorum*, the causal agent of white mold and related diseases (Godoy et al., 1990). Effective pathogenesis by *S. sclerotiorum* requires the secretion of

pathogenicity factors like extracellular lytic enzymes such as cellulases, hemicellulases and pectinases (Riou et al., 1991), oxalic acid (Cessna et al., 2000), aspartyl protease (Poussereau et al., 2001) and acidic protease (Girard et al., 2004). These enzymes are highly active under the acidic conditions provided by oxalic acid and degrade the plant cell wall and tissues beneath it. Thus the release of an array of lytic enzymes and the oxalic acid from the growing mycelium is the pathogenicity factors that are required for the establishment of the host-parasite relationship.

The pathogen *S. sclerotiorum* perpetuates by formation of Sclerotia and remains viable in soil for 4-5 years. Sclerotia of S .sclerotiorum have been reported to survive for more than 4 years (Halkilathi, 1962). He found that, survival of Sclerotia on the soil surface depended on weather conditions in summer. Sclerotia survived well in dry summer but in wet summer high temperature favoured their destruction. Williams and western (1965), reported that increasing soil moisture accelerated degeneration of Sclerotia of *S. sclerotiorum*, but at high moisture levels that was offset by the formation of secondary Sclerotia. They noted that soil moisture above 30 per cent of the moisture holding capacity of the soil favour sclerotial survival. Cook et al. (1975) reported that survival of Sclerotia of *S. sclerotiorum* was affected at various depths, and both soil moisture and temperature. They found that survival of Sclerotia was not affected by placement, either on the soil surface or at the 5 cm depth in soil kept at 5°C for 3 months. Sclerotia survival in soil kept at 27°C with a percentage of water 8.4 was similar to that at the low and moderate moisture levels kept at 5°C. They further reported that nearly 75 per cent of the Sclerotia recorded after 3 years burial at 5, 12.5 and 20 cm below the soil surface germinated and formed apothecia. Merriman (1976), reported that burial of Sclerotia at 4 cm for 35 weeks reduced recovery of Sclerotia to zero in sandy clay loam and 50 per cent in sandy loam soil. At the soil surface, recovery was reduced by 55 per cent in sandy clay and by 10 per cent in sandy loam soil. Less than 50 per cent of Sclerotia recovered were viable. Adams and ayers (1979) reported that soil biological component as the major factor determining the survival of Sclerotia in soil and also found that approximately 90 per cent of the life cycle of the fungus are spent in soil in this quiescent viable state.

According to Willetts and Wrong (1980), sclerotium formation generally occurs after host tissues are colonized by mycelium and are broken down by cell wall degradating enzymes and secretion of oxalic acid. Liu and Sun (1984) demonstrated that Sclerotia were lysed often 20-30 days in flooded soil, but survived well in dry soil. They suggested that, the microflora in flooded soil may be responsible for sclerotial lysis. When the soil moisture survived better at 80 per cent moisture. Although there are several reports on the survival period of Sclerotia, a multiplicity of factors influences their survival in nature (Coley-Smith and Cooke, 1971; Punja and Levesque, 1996). Punja (1986) reported that, the accumulation of staling compounds, injury to mycelium, presence of various chemicals and nutrient deprivation influences sclerotium formation. Tu (1988) worked on white mold-infected beans (Phaseolus vulgaris L.) and observed that *S. sclerotiorum* survived in infected seeds as dormant mycelium. He found that, the survival rate was 85-89 per cent and it did not change appreciably over a 3 years period. Further noted that, when the infected seeds were sown in sand or soil, 88-100 per cent failed to germinate.

Management Strategies

Chemical Management of *S. sclerotiorum*

A few chemicals are known to give effective control against sheath blight pathogen (Roy and Saikia, 1976). Sharma and Leu (1980) found that spraying 0.02 per cent Benlate controlled the stalk rot of cauliflower caused by *S. sclerotiorum*. Control of white rot of seed cauliflower caused by *S. sclerotiorum* was attempted with chemicals like Brassicol, Ziram, Caolin, Benlate MBC, Difolatan-80 and Mercuric chloride (Singh and Gangopadhyay, 1984). Pommer and Lorenz (1987) demonstrated high protective activity of Di-carboximide fungicides against *S. sclerotiorum* infection on lettuce, capsicum, beans, rapeseed etc. Raj (1991) and Singh (1994) reported that among the various diseases, Sclerotinia rot incited by *S. sclerotiorum* causes serious damage in terms of stand, quality and yield of French bean. Considering the soil-borne and polyphagous nature of the pathogen, management of the disease is still far from satisfaction. Nevertheless, several attempts to control the disease have been made by screening both systemic and non-systemic fungicides. Guha et al. (1998) also found that soil drenching with Bavistin (Carbendazim) at 0.1 per cent solution was most effective in management of stem rot of chickpea and rajma.

Of the mixtures of the fungicides Thiram and Azoxystrobin that were tested using an in vitro mycelial growth assay, the 1:4 ratio provided the greatest inhibition of *S. sclerotiorum*. When tested against nine isolates, the 1:4 mixture resulted in a mean synergy ratio of 2.31, indicating synergistic inhibition. Mycelial respiration was inhibited for about 2 h by Azoxystrobin alone but for 48 h by the mixture of Thiram and Azoxystrobin have been reported (Yabing et al., 2012).Yabing et al. (2013) found that phenylpyrrole fungicide, fludioxonil had a strong inhibition on mycelia growth of *S. sclerotiorum*. After fludioxonil treatment, cell membrane permeability, glycerol content, POD and PAL activity increased markedly, but oxalate and EPS content significantly decreased. The protective and curative test of fludioxonil suggested that protective effect was better than curative either on leaves or on stems of oilseed rape.

Benzothiostrobin, a novel Strobilurin fungicide had good control efficiency against *S. sclerotiorum*, protective activity was better than curative activity which strongly inhibited mycelial respiration within 12 h (Congying Xu, 2014).

Cultural Management of *S. sclerotiorum*

Cultivation of non-host crops to *S. sclerotiorum* resulted in the reduction of inoculums load (Adams and Ayers, 1979). But the pathogen has more than 400 plant species as its host for survival, three to four years of crop rotation did not reduce the incidence of stem rot of canola (Morrall and Dueck, 1982). A minimum of 5 year rotation of two non-host crops of *S. sclerotiorum* is essential to decrease the infection level by the pathogen (Gulya, 1997). Integration of zero-tillage with crop rotation will reduce the risk of the crop from the attack of the necrotrophic pathogen *S. sclerotiorum*. The sclerotial bodies are seen near the top 2-3 cm of soil and they deteriorate faster by the attack of mycoparasites that dwell in the top soil (Tu, 1986). Carpogenic germination of the Sclerotia occurs in the upper 5 cm soil profile (Kurle, 2001). But if the soil is ploughed the resting structures are buried deeper in the soil and have the capability to survive for several years. A significant negative relationship was found between sclerotial viability and depth of burial, and between sclerotial viability and populations of colonizing bacteria under zero tillage condition (Duncan, 2003). Thus, the inoculums load of the Sclerotia could be reduced well through zero tillage and there by infection of host plants by the pathogen could be minimized. Ghasolia and Shivpuri (2005) reported that production of apothecia was higher when Sclerotia were placed at soil surface and decreased significantly with the increasing depth of burial which indicated that deep ploughing of the infested fields may be a useful practice to reduce inoculums of the pathogen.

Biological Management of *S. sclerotiorum*

Antagonistic activity of Gliocladium spp. against *S. sclerotiorum*. Several species of Gliocladium are parasitic on a wide variety of plant pathogens including *S. sclerotiorum* (McCredie and Sivasithampara, 1985; Phillips, 1986; Mukhopadhyay, 1995; Phookan and Chaliha, 1997; Ao, 1999). Mode of parasitism of plant pathogens by Gliocladium spp. is shown to be through necrotrophic mycoparasitism or antibiosis (Howell and Stipanovic, 1979). Tu (1980) reported that G. virens is a mycoparasite which can parasitize both mycelia and sclerotia of *S. sclerotiorum*. He further described the inhibition of carpogenic germination of Sclerotia of *S. sclerotiorum*.

Papavizas (1985) described the production of toxic metabolites and antibiotics such as gliotoxin and viridian along with various cell wall degrading enzymes such as exo and endoglucanase, cellobiase and chitinase by Gliocladium, all of which may potentially be involved in bio-control activity. Phillips (1986); Whipps and Budge (1990), evaluated the antagonistic capacity of G virens and found that the fungus reduced the viability of *S. sclerotiorum* by destroying the Sclerotia. Mukhopadhyay (1994) recorded that G. virens could strongly antagonize P. aphanidermatum, the incitant of tomato damping off. The hyphae of G. virens run Parallel, entwined around, penetrated into and killed the host hyphae. Mukherjee et al. (1995) reported that G. virens was highly antagonistic to R. solani and *S. rolfsii*. They found G. virens to colonize, penetrate and sporulate inside the Sclerotia of the pathogen and suggested parasitism of Sclerotia as the principle mechanism of biological control.

Rabeendran et al. (1998) reported that G. virens and G. roseum were effective in reducing petiole infection caused by *S. sclerotiorum* in cabbage by more than 70 per cent and complete prevention of Sclerotia production. Dubey (2000) demonstrated that the antagonists G. virens, T. viride and T. harzianum caused coiling of hyphae around the host hyphae i.e., Thanatephorus cucumeris causing web blight of groundnut and caused lysis of the pathogen hyphae. The hyphae of these antagonists were observed inside the colonized Sclerotia and finally they disintegrated. Amongst the tested fungal biological control agents, G. virens was the most effective agent and caused highest reduction (55.6%) in carpogenic germination of Sclerotia of *S. sclerotiorum* (Ghasolia and Shivpuri, 2005).

Antagonistic activity of Trichoderma spp. against *S. sclerotiorum*

Trichoderma, was first recognized as a biological control agent by Weindling in 1932 and 1937, who isolated fungi toxic substances from culture filtrates of Trichoderma lignorum (Tode) harz (= T. viride). Since this initial work, Trichoderma spp. have been used as bio-control agents against many soil-borne pathogens. The four general mechanisms believed to be associated with bio-control are a combination of competition, lysis, antibiosis and mycoparasitism. Trichoderma spp. are known to produce various antibiotics and other cell wall degrading enzymes that are lytic and detrimental to a number of fungi (Dennis and Webster, 1971). Shrinkage of hyphae, vacuolation of Sclerotia and parasitism due to T. harzianum by forming haustoria at the contact point by coiling around the hyphae and growing inside the hyphae have been reported by Elad et al. (1980) and Chet et al. (1981).

Lee and Wu (1984) showed that T. viride produced antibiotics that inhibited the mycelia growth of *S. sclerotiorum* completely and induced swelling and plasmolysis of affected cells. T. harzianum produced a pyrone compound i.e 6-n-phenyl-2H-pyran-2-one, that has an antibiotic properties. They showed that the compound inhibited the growth in vitro of a number of fungi and that it reduced the damping off in lettuce seedlings by R. solani. As bio-control

agents Trichoderma spp. are applied in soil (Trutman and Keane, 1990; Roiger and Jeffers, 1991) as seed treatment (Harman et al., 1981) or occasionally as foliar spray (Corke, 1978 and Tu, 1997). Lacicowa and Pieta (1986) reported that T. koningii was the most effective mycoparasite among different antagonistic saprophytes and recorded T. koningii to completely inhibit mycelia development and destroy Sclerotia after 4 weeks.

Krutova (1987) both in laboratory and field trials found T. hamatum and T. harzianum besides other antagonists on Sclerotia of *S. sclerotiorum* that showed hyperparasitic activity on the pathogen and were capable of destroying its Sclerotia in soil. Simon et al. (1988) also observed the production of antifungal antibiotic(s) from T. koningii. It has been observed that the sclerotinia spp. could be colonized by species of Trichoderma and resulted in lower germination and reduced their survivability in soil (Adams and Ayers, 1979; Huang, 1980; Zazzerini and Tosi, 1985; Knudsen et al., 1991). T. koningii parasitized the Sclerotia of *S. sclerotiorum* which was reported by Trutmann and Keanne (1990). They observed that at 20°C T. koningii required 2 weeks to infect 50 per cent of Sclerotia and there was an inverse relationship between infection and sclerotial viability. Under microscopic studies they observed that T. koningii either grew along or coiled around the hyphae of *S. sclerotiorum* and reported that, the host- parasite interaction involves antifungal antibiotics and cellulolytic enzymes. Working on *S. sclerotiorum* causing stalk rot of cauliflower, Dohroo et al. (1990) and Kansal et al. (1990) observed that sclerotial germination of *S. sclerotiorum* was reduced considerably in cultural filtrates of Trichoderma spp. Ghisalberti and Sivasithamparam (1991) studied the role of antibiotic production by various Trichoderma spp. and its involvement in bio-control activity. They found that Trichoderma spp. and are capable of producing an array of metabolites such as dermadin and trichoviridin which have antifungal activities. Sarma (1994) found that Trichoderma spp. inhibited the mycelial growth of *S. sclerotiorum* in dual cultures and reported that, T. harzianum and T. viride inhibited sclerotial germination in pot culture experiments reducing seedling mortality of chick pea stem rot caused by *S. sclerotiorum*.

Singh and Handique (1997) reported the destruction of Sclerotia of *S. sclerotiorum* by T. harzianum. They found that at 10 days, 50 per cent Sclerotia were destroyed whereas after 20 days, no Sclerotia were survived. Application of T. harzianum T39 conidia to the root zone of plants resulted significant reduction of foliar grey mold and powdery mildew. Menendez and Godeas (1998) observed that the cell walls of *S. sclerotiorum* were degraded by T. harzianum when it was grown in a medium containing the cell walls of the pathogens. It was reported that Trichoderma spp. could parasitize the Sclerotia of *S. sclerotiorum* and S. minor at a temperature of 25°C and 30°C (Rollen et al., 1999). Singh et al. (2000) observed that the stolen decay of field mint caused by *S. sclerotiorum* could be effectively reduced by employing biological control agent like T. harzianum and G. virens.

Integrated Management

Integrated disease management strategy is more effective, less expensive and more eco-friendly as compared to chemical control method. The integrated biological control of plant pathogens has been discussed by several workers (Baker and Cook, 1974; Elad et al., 1980; Klassen, 1981; Lewis and Papavizas, 1992). Reports on integrated control of R. solani using Trichoderma spp. and fungicides include that of Hadar et al. (1979).Who observed synergistic effect of T. harzianum and PCNB on R. solani damping off of eggplant seedlings in natural soil. Abd-El Moity et al. (1982) reported fungicide resistant or tolerant isolates of bio-agents for use in integrated controlled are readily obtained by selection on pesticide containing media. Chet et al. (1982) reported a synergistic effect resulting from the interaction between T. harzianum and reduced doses of PCNB when applied against *S. rolfsii* in peanut.

Kloepper (1983) reported that mixtures of two or more rhizobacteria resulted in significant reductions in daughter tuber infestations by Erwinia caratovora; however, control by mixed strains was not greater than that obtained by any single strain. Elad et al. (1980) and Katan (1985) showed that soil solarization in combination with T. harzianum enhanced disease control in potatoes against R. solani and *S. rolfsii*. Papavizas (1985) obtained highest yield of pea grown in P. ultimum infested field with combined seed treatment with Metalaxyl and T. harzianum. Combination of bio-control agent with broad spectrum fungicides is more common (Kraft and Papavizas, 1983; Ordentlich et al., 1990; Wokoche, 1990; Kaur and Mukhopadhyay, 1992). Several workers have reported that pathogen weakens by sub-lethal doses of fungicides and plant parts might be controlled more effectively by an antagonist (Mukhopadhyay et al., 1992; Hwang and Chakravarty, 1993; Bhatnagar, 1995).

Fungicides might have weakened the pathogen and made it vulnerable, thus allowing antagonist to become more virulent on a weak pathogen (Upadhyay and Mukhopadhyay, 1986). Shrestha and Mukhopadhyay (1992) reported that the integration of Gliocladium virens with systemic fungicide Carboxin (Vitavax) was found quite effective control efficiency of the fungicides by the integrated use of Trichoderma species has been reported by number of workers (Chet et al., 1982; Kraft and Papavizas, 1983; Ordentlich et al., 1990). Chet (1990) reported that the satisfactory control of bean blight caused by Sclerotium rolfsii could be achieved with T. harzianum combined with PCNB. Successful control of Rhizoctonia root rot of

pea by integration of bio-agents with chemicals has been also reported by Hwang and Chakravarty (1993). Since different Trichoderma spp. vary in the amount and types of antibiotics/ antifungal enzymes production, combination of more than one type may prove more useful (Ghisalbert et al., 1993). However, the possible role of edaphic factors in disease suppression and or on the activity of these bio-control agents also needs to be explored for their manipulation for practical disease control.

Integration of biological and chemical control constitutes a very promising way of controlling various soil-borne pathogens (Papavizas, 1973; Henis and Chet, 1975; Papavizas, 1981; Bora et al., 1999; Cole and Zevenyika, 1988; Sokhi, 1994 and Vyas, 1994). Jeyraj and Rambadran (1996) observed that T. harzianum and low doses of Carbendazim reduced dry root rot in mung bean. The result of these experiments also indicated that addition of sub-lethal doses of Carbendazim (0.02%) in combination with T. harzianum improved disease control. Enhanced plant height and biomass were also recorded in treatment involving bio-agent combination.Upadhyay and Mukhopadhyay (1986) and Chattopadhyay and Sen (1996) also made similar observation in sugar beet against Sclerotium and muskmelon against Fusarium oxysporum respectively. Solarization of nursery mixture, its fortification with Vesicular Arbuscular Mycorrhiza (VAM), T. harzianum and G. virens were found highly effective in ensuring robust disease free planting materials in black pepper and cardamom (Sarma and Anandaraj, 1996).

Sharma and Basandarai (1997) reported that biocides (T. viride, T. harzianum and G. roseum) and Carbendazim were highly effective in reducing the sclerotial viability of *S. sclerotiorum* in cauliflower individually and in combination. Dutta and Das (1999) reported lethal dose of Carbendazim was found to be compatibility of T. harzianum in management of stem rot of soybean. Bora et al. (1999) used T. harzianum against R. solani, the causal agent of sheath blight of rice with different carriers (food base) viz., methyl cellulose, gur along with sub-lethal dose (0.1 and 0.5%) of Thiram by seed treatment. Lower disease incidence was observed in T. harzianum + methyl cellulose + sub-lethal dose of Thiram (0.1 and 0.5%) which was followed by gur + sub-lethal dose of Thiram with antagonist as compared to control. Integration of antagonist (G. virens) and fungicide (Thiram) was found more effective in reducing the seedling mortality and increasing germination and yield in comparison to alone due to longer and more effective protection (Dubey, 2000).

Beena et al. (2000) reported that treatment with agrochemicals like Dithane M-45, Quinalphos, Malathion and Bio-Control Agent (BCA) minimized storage loss due to ginger rot caused by Fusarium and Rhizoctonia. Treatment with fungicides like Mancozeb and Metalaxyl alone and in combination with insecticides viz., Quinalphos and Malathion and BCA were all at par and showed increased percentage of recovery of healthy as well as plantable seeds. Increased percentage of germination was also noticed in these cases. Rajeswari et al. (2000) found that application of T. harzianum either alone or in combination with pathogen, solarization, Carbendazim, neem cake and with other bio-agents viz., T. viride and G. virens were found significantly superior over control and on par with each other in reducing the disease and in increasing plant height, biomass and grain yield. Bora and Das (2001) studied on IPM approach for the management of rice sheath blight (R. solani). They stated that when rice seeds were treated with suspension of T. viride, T. harzianum, A. terreus and B. subtilis at different concentration along with fungicide Thiram at 0.01, 0.05 per cent caused lower disease incidence as compared to seed treatment with Thiram 0.03 per cent alone. Muralia et al. (2001) evaluated the efficacy of three antagonistic fungi, namely, T. viride, T. harzianum and G. virens alone and in combination with seed dressing fungicides, that is, Carbendazim 50 per cent WP and Mancozeb. All the antagonists significantly controlled the stem rot disease caused by *S. sclerotiorum* in Brassica junce and improved the growth (plant height, dry weight and seed weight). However, these parameters were further improved when the antagonists were used in combination with the chemicals. Sankar and Sharma (2001) carried out an experiment in which maize seeds were treated with talc based powder formulation of fungal antagonist T. viride @ 4,8 and 12g kg^{-1} and Carbendazim (Bavistin 50 WP) at 4g kg^{-1}. The treated seeds were kept for 24 hrs and then sown in the field. They found better growth and dry matter production in all the treatments over the control. The T. viride (MR) 12g kg^{-1} treated plot gave maximum shoot length, dry matter and grain yield followed by T. viride (MR) 8 kg^{-1} treatment, but both were statistically at par. Two promising antagonists viz.,Pseudomonas fluorescens and T. viride in combination with Metalaxyl (apron 35 WS and Ridomil MZ-72 WP) were found to be most effective and compatible. No deleterious effect of these bio-control agents on the emergence of pigeon seedling was noticed when applied along with Apron 35 WS @ 3.0 g kg^{-1} seed. Moreover, this also reduced the disease severity significantly from 60.2 per cent to 42.8 per cent under field condition. Such interaction suggested that an integrated management may be possible to certain blight diseases (Singh et al., 2000).

Pant and Mukhopadhyay (2001) found that integration of G. virens and T. harzianum with Vitavax was effective against seed and seedling rot of soybean caused by R. solani under field conditions. They observed that integration of Vitavax (0.1%) with G. virens and T. harzianum improved seedling emergence, plant stand and yield significantly. Nath (2002)

evaluated integration of seed treatment with talc based formulation of T. harzianum along with Bavistin (0.05/0.1%) and found significant reduction in incidence of white rot of French bean and increased germinability and yield of the French bean in both pot and field condition. The bio-control agents applied in integration with fungicides, despite some inhibitory effect of the latter at early stages, could multiply and establish in the rhizosphere and surrounding soil, when the effect of the fungicides may have been lost (Vyas and Mathur, 2002). Soil application of bio-control agents has an edge over their seed treatment as these being natural soil inhabitants, establish and multiply more quickly in soil.

Tewari and Mukhopadhyay (2003) also found that integration of biological and chemical seed treatment gave better control against chickpea root rot and collar rot caused by R. solani. Surulirajan and Kandhari (2005) assessed disease severity and incidence of sheath blight of rice through different treatment combinations and found that out of sixteen treatments T. viride + Carbendazim 50 WP (0.1%) spray along with soil amendments (FYM 1% + saw dust) showed maximum reduction in sheath blight severity. Baiswar and Chandra (2007) reported that T. harzianum and T. viride were found compatible with each other for the management of corm rot of gladiolus. Bhai and Thomas (2010) studied compatibility of T. harzianum (Rifai) with fungicides, insecticides and fertilizers and reported Copper oxychloride was found compatible with T. harzianum. An integration of bio-agents with Captan significantly reduced white mold incidence of French bean caused by *S. sclerotiorum* under pot condition. Amongst the treatments, Maximum disease control was recorded with Captan @ 0.2 % integrated with both T. harzianum and G. virens (Bora, 2013).

Conclusion

sclerotinia sclerotiorum (Lib.) de Bary is a soil-borne pathogen capable of infecting more than 400 host plants worldwide. It is a major pathogen that plays a crucial role in reducing the yield in economically important crops. Management of sclerotinia with chemical fungicides though remains successful; exclusive reliance on chemical pesticides had resulted in significant loss to public health, environment and problems such as pesticides resistance, resurgence, residue and environmental pollution. Most of the conventional methods are not effective in management of *S. sclerotiorum*. The pathogen produces resting structures (Sclerotia) that could inhabit the soil upto 10 years without losing its viability. In addition, carpogenic germination of sclerotial structure releases millions of ascospores. The senescing petals of the crops serve as a nutrient source for the proliferation of ascospores to establish pathogenicity. Further, management of Sclerotia resting in the soil is alone not sufficient to reduce the disease, the infection court such as petals and the leaves has to be protected from the ingress of ascospores. In the midst of these obstacles, instead of adopting a single management technique, development of eco-friendly management strategies i.e., Integrated approach which involving co-ordinated use of multiple tactics for optimizing the control of pathogen is an ecologically and economically crucial method for effective and safe way of reduction or elimination of soil-borne inoculum of the pathogen.

Proposed Future Research Priorities

- Identification of native biocontrol agents (BCA) with high competitive saprophytic ability and rhizosphere competence, which possess wide spectrum of biological suppressive activity against more than one pathogen
- Investigate the epidemiology of ascospores to better understand *S. sclerotiorum*
- Improve disease assessment of *S. sclerotiorum* i.e., developing new rating Scales
- Development of a bio-control consortium, which would have wider adaptability to different ecological niches
- Monitoring the population stability of the BCA in relation to pathogen population and their ecological parameters that would ensure biological balance
- Developing BCAs having compatibility with agrochemicals to develop Integrated Disease Management (IDM) strategies

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