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Sclerotinia rot of rapeseed mustard: A comprehensive review

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Abstract: Indian mustard [*Brassica juncea* (L.) Czern & Coss.] is one of the major oilseed crops cultivated in India and around the world. It is extensively grown traditionally as a pure crop as well as intercrop (mixed crop) in marginal and sub-marginal soils in the eastern, northern and north western states of India. Cool and moist climate of winter months is the major factor for luxuriant growth and productivity of mustard in these states. Despite considerable increase in productivity and production, a wide gap exists between yield potential and yield realized at farmer's field, which is largely due to biotic and abiotic stresses. The destructive diseases of rapeseed-mustard include those caused by fungi, bacteria, viruses and phytoplasma. Among them, Sclerotinia stem rot is the most serious fungal disease that causes maximum damage in Indian mustard. This paper reviews the research and development of Sclerotinia rot in rapeseed-mustard during the past years in relation to pathogen taxonomy, biology, epidemiology, disease cycle and management. The paper also attempts to present future outlook and strategy for Sclerotinia rot of rapeseed mustard research.

Keywords: Management, Rapeseed-mustard, Sclerotinia rot, Survival

INTRODUCTION

Rapeseed-mustard is the third most important oilseed commodity in the world after soybean (*Glycine max*) and palm (*Elaeis guineensis Jacq*) in world agriculture and India is the third largestproducer with global contribution of 28.3 per cent acreage and 19.8 per cent production (Shekhawat *et al.,* 2012; Bandopadhyay *et al.,* 2013). In India, rapeseed-mustard crops are cultivated on an area of 6.45 million ha with a production of 7.28 million tones and with an average yield of 1128 kg/ha (Anonymous, 2015). Among the oilseed brassicas, mustard (*Brassica juncea*), yellow sarson (*B. campestris* var. yellow sarson), brown sarson (*B. campestris* var. brown sarson), toria (*B. campestris* var. toria), oilseed rape (*B. napus*) and Karan rai (*B. carinata*) are grown for edible oil, whereas black mustard (*B. nigra*) is used as a condiment and for pickle making. The leaves of young plants are used in the human diet as a green vegetable. The oilseed brassicas usually contain 38-57 per cent of erucic acid, 4.7-13 per cent of linolenic acid and 27 per cent of oleic acid and linoleic acids, which are of high nutritive value required for human health (Kumar *et al.,* 2014). Rapeseed- mustard [*Brassica juncea* (L.) Czern & Coss.] is one of the most important oilseed crops, are exposed to various pathogen, which infect and disturb the normal physiological functions during growth and development. Among the diseases that hampered the productivity of oilseed brassicas, Sclerotinia rot caused by *Sclerotinia sclerotiorum* (Lib.) de Bary is most predictable diseases worldwide. In the changing climate, diseases in mustard have been recognized as a major threat in causing more economic losses these years than before. Because, some of the diseases known as serious in past have become more or less serious depending upon the geographical locations in the country and some of the minor disease problems have now become major ones. In India, the Sclerotinia stem rot was considered as minor importance few decades ago but recently this disease is a serious handicap in successful cultivation of rapeseed-mustard (Rakesh, 2014).

DISTRIBUTION AND HOST RANGE

S. sclerotiorum causing Sclerotinia stem rot or white stem rot in rapeseed-mustard is worldwide in distribution and is pathogenic to more than 400 plant species (Purdy, 1979; Boland and Hall, 1994) and more than 500 plant species (Sharma *et al.,* 2015) at various developmental stages of plants. The pathogen poses a hazard to many dicotyledonous crops such as rapeseed -mustard, sunflower, soybean, edible dry bean, chickpea, peanut, dry pea, lentils and various vegetables, even also reported to infect monocotyledonous species such as onion, tulip and many others (Boland and Hall, 1994; Bolton *et al.,* 2006).

HISTORY OF PATHOGEN *SCLEROTINIA SCLEROTIORUM*

S. sclerotiorum was first described in 1837 by Libert as *Peziza sclerotiorum* and later named as *S. libertiana*

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Fuckel in 1870 (Purdy, 1979). This binomial was accepted until it was demonstrated as incoherent with the International Rules of Botanical Nomenclature, so the name *S. libertiana* was changed to *S. sclerotiorum* (Lib.) Massee (Wakefield, 1924). However, Wakefield (1924) incorrectly reported that the combination of *S. sclerotiorum* was first used by G.E. Massee in 1895, resulting in the citation *S. sclerotiorum* (Lib.) Massee. However, it was later found that de Bary had used this Latin name earlier, so the proper name was established as *S. sclerotiorum* (Lib.) de Bary (Purdy, 1979). The early taxonomy of the three species (*sclerotiorum*, *minor* and *trifoliorum*) was based on the size and general characteristics of the sclerotium, host range and dimensions of ascospores and asci (Willetts and Wong, 1980). However, several studies (Purdy, 1955; Price and Colhoun, 1975; Grogan, 1979) showed that this system was inadequate and a number of species, originally thought to be unique, were actually all members of the *S. sclerotiorum* species. *S. sclerotiorum* belonged to the Sclerotiniaceae Whetzel, a family of the class Ascomycotina. The pathogen belongs to kingdom-Fungi, phylum-Ascomycota, class-Discomycetes, order-Helotiales, family-Sclerotiniaceae and genus-*Sclerotinia.* Hyphae of *Sclerotinia* are hyaline, septate, branched and multinucleate; mycelium may appear white to tan and no asexual conidia are produced, however, numerous black coloured sclerotia are formed in the culture (Bolton *et al.,* 2006).

YIELD LOSSES

Sclerotinia rot occurs every year in all the rapeseed-mustard growing areas of the world. Yield loss estimates due to Sclerotinia disease have been made as high as 28 per cent in individual rapeseed fields in Alberta and 11.1 to 14.9 per cent in Saskatchewan, Canada (Morrall *et al*., 1976). This disease caused severe yield losses up to 50 per cent in winter oil seeds in Germany (Horning, 1983). At the time of harvesting and threshing, sclerotia of this fungus got mixed with seed and this represented an objectionable seed contaminant for export from one country to another and thus affected the marketability of the crop. The quality of seed even in partially infected plants has been adversely affected (Kruger *et al*., 1981). In Nepal, grain yield, plant height, number of siliquae per plant and 1000 grain weight were found reduced due to stem rot incidence and yield loss attributed to 75 per cent (Chaudhary, 1993). Infestation level of 40 per cent may cause yield losses up to 50 per cent in oilseed rape, as a result of a reduced thousand seed weight or an early shattering of pods (Pope *et al*., 1989). The pathogen has been reported to be endemic in North Dakota, with an annual average incidence of 13.6 per cent and had direct economic impact estimated as US 94 million for the period from 1991 to 2002 (Lamey, 2003). Del Rio *et al.* (2007) reported disease incidence from 1 to 59 per cent in North Dakota and observed that for every per cent of Sclerotinia rot incidence, yield reduced by an average of 13.1 kg/ha, *i.e*., 0.52 per cent of potential yield. Rape (*B. napus*) yield has also been observed decline in southern Australia from 0.39 to 1.54 t/ha due to Sclerotinia stem rot (Kirkegaard *et al*., 2006). In India, Shaw and Ajrekar (1915) were first to report *S. sclerotiorum* on several hosts including rapeseed-mustard causing Sclerotinia rot. The disease has assumed a serious proportion in mustard growing areas in India (Lodha *et al.,* 1992; Krishnia *et al.,* 2000) as the incidence of this disease was noticed up to 72 per cent in Rajasthan (Shivpuri *et al.,* 2000; Ghosolia *et al.,* 2004) and up to 80 per cent in Punjab and Haryana (Kang and Chahal, 2000). Sharma *et al.* (2001) also observed up to 49.2 per cent disease incidence from Haryana, however, in few areas the disease incidence has approached up to 80 per cent. Losses in yield up to 72 per cent from Uttar Pradesh and up to 50.9 per cent from Rajasthan in mustard due to this disease have been also reported (Chauhan *et al*., 1992; Singh, 1998). Plants infected at or before flower initiation resulted in 100 per cent yield loss, whereas, infection after flowering stage caused more than 50 per cent yield loss (Shukla, 2005).

DISEASE SYMPTOM

S. sclerotiorum causes more or less similar symptoms on leaves, stem and siliquae as fluffy white mycelia and sclerotia are produced after mycelial growth (Fig. 1.) when the nutrition is not sufficient or other conditions are favourable for sclerotial development (Christias and Lockwood, 1973). Sclerotinia stem rot in rapeseed mustard starts as elongated, water soaked lesions on stem especially at base or at internodes and later white mycelial growth covers these lesions and affected plants look whitish from distance. The disease becomes air borne and spread through infected flower petals which fall and become lodged between the main stem and side branches. Large oval to round shaped holes are also formed on leaves due to air borne infection. Under severe infection, defoliation, shredding of stem, wilting and drying of plants occurs. Infected plants will ripe earlier and stand out among green plants (Meena *et al.*, 2014).

ENZYME OF *SCLEROTINIA SCLROTIORUM*

The explosive pathogenicity of *S. sclerotiorum* under favourable conditions and the ability of its sclerotia to withstand adverse conditions allow it to be a flourishing pathogen. Peroxidase and phenylalanine ammonialyase (PAL) are two enzymes frequently associated with infection by phytopathogens (Hammerschmidt *et al.,* 1982; Shirasshi *et al.,* 1989; Southerton and Deverall, 1990). The stimulation of the activities of these enzymes has been correlated with resistance to infection in many of the available reports, but conclusive evidence of their role in the defense mechanism of plants is not yet available. The biochemical processes involved in the expression of resistance in *B. napus* are rarely known. However, the accumulation of a phy-

Fig. 1. a: *Pure culture of Sclerotinia sclerotiorum in Petri dish,* ***b*** *and* ***c:*** *cottony white mycelium on infected plant stem,* ***d:*** *formation of sclerotia on the stem,* ***e:*** *Sclerotia inside the infected pith of stem,* ***f:*** *production of apothecia from the sclerotia.*

toalexin in *Brassica* sp. in relation to a hypersensitive reaction toward *L. maculans* has been reported (Rouxel *et al.,* 1989). *S. sclerotiorum* is known to produce pectinolytic and cellulolytic enzymes (Lumdsen, 1969; Favaron *et al.,*1988; Marciano *et al.,* 1982). The level of these enzyme activities correlates with the development of disease symptoms (Favaron *et al.,* 1988; Lumdsen, 1976).

BIOLOGY AND DISEASE CYCLE OF *SCLEROTINIA SCLEROTIORUM*

S. sclerotiorum (Lib.) de Bary is necrotrophic fungal pathogen in the Ascomycota and Order *Helotiales* produce fluffy white mycelium on and in infected plant parts. This mycelium aggregates itself into sclerotia, which are the structures that allow *Sclerotinia* species to survive in soil in the absence of plant host. A sclerotium is a hyphal aggregate with an outer black rind, several cells thick containing melanin, a compound that is believed to play an important role in protection from adverse conditions and microbial degradation in many fungi (Bell and Wheeler, 1986; Henson *et al.,* 1999). Sclerotia buried in the plough layer of soil can survive and remain infective for up to 5 years and there are four stages in its life cycle: sclerotium, apothecium, ascospore, and mycelium (Hieu, 2007). Germination of fungal sclerotia has been reported to be of two types *i.e.* myceliogenic and carpogenic (Coley-Smith and Cooke, 1971). In myceliogenic germination, the sclerotium produces mycelium when soil remains wet and cool and the optimal conditions for germination are near-saturated wetness with soil temperatures of 12 to 24°C. These conditions are created by extended rainy periods, or by irrigation in combination with soil shading due to closure of the crop canopy. These hyphae infect roots and crowns and other plant parts that are touching the ground. Infection then spreads to aboveground plant parts (Coley-Smith and Cooke, 1971). Sclerotia of *S. sclerotiorum* most commonly produce a mushroom-like fruiting body termed as apothecium and carpogenic germination of apothecium usually requires the sclerotia to be in wet soil for one to two weeks prior to germination. At soil depths of up to 2 cm, apothecia can extend from the sclerotia to reach the soil surface. One or several apothecia can emerge from a single sclerotium. Apothecia are fleshy-colored discs measuring 4 to 8 mm in diameter. They produce ascospores as a result of a sexual process (Coley-Smith and Cooke, 1971). Ascospores release and petal fall should occur at the same time (Kruger, 1980). Morrall

and Dueck (1982, 1983) have reported severe infections in the fields with few or no apothecia. Clarkson *et al.,* (2003) reported that apothecia of *S. sclerotiorum* were produced at an optimum temperature of 15˚C and ascospores survived a wide range of conditions, but high temperature and humidity reduced the viability and no apothecial initials were produced at either 30˚C or 5˚C; continuous leaf wetness of 48-72 h were required for infection by ascospores. The infection by *S. sclerotiorum* in yellow sarson and *B. compestris* var. toria was aggravated by low temperature, heavy rain fall and closer spacing (Sexsena and Rai, 1987). The requirement of moisture for carpogenic germination and growth of the pathogen were the reasons why rainy periods or irrigation are coupled with outbreaks of disease on certain crops.

SURVIVAL/ VIABILITY OF SCLEROTIA OF *SCLEROTINIA SCLEROTIORUM*

S. sclerotiorum (Lib.) de Bary is a facultative parasitic Ascomycete fungus (Kirk *et al*., 2001), and can grow well even in an unfavourable environment and survive for up to 8 years in soil in the sclerotia form (Adams and Ayers, 1979). Holley and Nelson (1983) reported that when the inoculum density increased up to a certain level, after that there was not much effect on the disease incidence. The disease incidence increased with increase in the density of inoculums and decreased with increase in the age of inoculums. The average disease incidence with inoculums of different ages varied from 48.5-74.7 per cent with different loads of inoculums (Ghasolia and Shivpuri, 2009). Survival of fungal sclerotia has been reported to be of two types *i.e.* myceliogenic and carpogenic (Coley-Smith and Cooke, 1971). Crisan and Urlea (1979) reported that 3-12 apothecia per sclerotium could be produced distally under controlled conditions, while Kapoor *et al.* (1987) observed 7-9 apothecia per stripe with 3-21 stripes per activated sclerotia. Susan *et al.* (1990) recorded that the more apothecia were produced from sclerotia placed at 0-2 cm than from those buried deeper. Low pre-conditioning temperature and continuously high moisture are crucial for carpogenic germination (Huang and Kozub, 1991; Dillard *et al.,* 1995; Harvey *et al.,* 1994). Moreover, the percentage of apothecia producing sclerotia decreased significantly with increasing depth of burial in the soil (Ben-Yephet, 1993). Numerous apothecia developed after one month of placement from field collected sclerotia buried at 1 cm followed by 2 cm depth (Singh and Singh, 1988). No apothecia emerged from pseudosclerotia buried at depths of greater than or equal to 3 cm, and the critical depth of burial was determined at 2.6 cm (Ngugi, *et al.,* 2002). Ghasolia *et al.* (2005) reported that production of apothecia was higher when sclerotia were placed at soil surface (0 cm) and decreased significantly with the increasing depth of burial. Amongst different soil types, the least number of apothecia production was recorded in sandy soil whereas, sandy loam soil resulted in maximum number of apothecia production (Mehta *et al.,* 2009). Sclerotia, the resting structures which allow pathogen to survive for long periods of time under adverse conditions, are formed in the soil and in the stems of the infected tillers at the end of the season. As early as Dillion-Weston *et al.* (1946) estimated that sclerotia of *S. trifoliorum* causing disease in clover survived for 6-8 years in field and these sclerotia required prolong period of moist soil to germinate myceliogenically and/or carpogenically. These sclerotia play a major role in disease cycles as they produce inoculum and are the primary long-term survival structures (Adams and Ayers, 1979; Willetts and Wong, 1980). Few studies have also quantified the sclerotial survival of *Sclerotinia* species at different depths and duration in soil (Pande *et al*., 2008; Cosic *et al.*, 2014). The viability of sclerotia on the surface declined rapidly due to the alternate wetting and drying of soil (Maiti and Sen, 1988). Survival was significantly influenced by depth of burial and by sclerotial treatment before burial. About 11-31 per cent of the sclerotia buried deeper than 2.5 cm survived after 30 days, whereas 65-84 per cent of those on the soil surface remained viable (Smith *et al*., 1989). Significant reductions in the mean number of sclerotia and lettuce drop incidence occurred on the crop immediately after deep ploughing (Subbarao *et al.*, 1996). The effect of tillage on survival of sclerotia is poorly studied and no generalizations can be made to aid in management of the pathogen. There is evidence that leaving the sclerotia on the soil surface enhances degradation, whereas burying the sclerotia enhances survival. It is thought that the more dramatic changes in temperature and moisture on the soil surface are deleterious to sclerotia (Rakesh *et al.*, 2015). Gurjar *et al.* (2004) stated that viability of sclerotia of *Sclerotium rolfsii* in chilli plant was reduced when buried in soil beyond 4 cm depth and lost their viability completely beyond 14 cm depth after 19 months. Sclerotia on the soil surface had the highest viability (57.5 %) followed by those at 5 cm depth (12.5 %) and only 2.5 per cent of those placed at the 10 cm depth after twelve months (Duncan *et al.*, 2006). Gradual reduction in viability of sclerotia (germination percentage) of *S. sclerotiorum*, was observed with the increase in soil depth and duration of burial both under screen house and field conditions (Rakesh *et al.*, 2015).

MEASUREMENT OF THE DISEASE SEVERITY

An appropriate method of disease assessment is a prerequisite for the identification of resistance to Sclerotinia rot in rapeseed mustard. The appearance of dis-

ease is initially observed both on leaves and stem later on sclerotia formation on the stem and inside the infected piths of stem, which is responsible for yield losses (Meena *et al*., 2014). A key for the assessment of Sclerotinia rot, disease rating was recorded according to 0-4 scale of Lesovoi *et al*. (1987) with slight modification to assess disease severity [0= no visible lesion on stem; 1= ¼ stem girdled by lesion; 2= ½ stem girdled by lesion; 3= ¾ stem girdled; 4= more than ¾ stem girdled] and per cent disease severity was calculated (Wheeler, 1969) as [(Sum of all numerical ratings/ total number of main stems observed × maximum disease rating) × 100].

MANAGEMENT OF SCLEROTINIA ROT

Rapeseed-mustard are repeatedly exposed to a number of pathogens and, as a result, they have evolved intricate defense mechanisms to recognize and defend themselves against a wide array of these pathogens by structural defense (Meena *et al.,* 2010) and by including a set of defense responses that can defeat the invading pathogens (Vishwanath *et al.,* 1999).

SEARCH FOR RESISTANT GENOTYPES

Host resistance offers the only economic and sustainable method for effective management of this disease (Zhao *et al*., 2004). Due to severe losses caused by the Sclerotinia rot in rapeseed-mustard, the objective of oilseed breeders is the development of resistant lines against Sclerotinia rot. Several attempts have been made in past to find out the sources of resistance against Sclerotinia rot, but no complete resistance to *S. sclerotiorum* is lacking in all cultivated rapeseed-mustard crops, however, partial resistance was identified in some of the *B. napus* and to a lesser extent in *B. juncea* genotypes from China, Australia (Li *et al.,* 2008) and India (Singh *et al.,* 2008 and Singh *et al.,* 2010). *B. napus* and *B. juncea* cv. *rugosa* genotypes have been reported to possess resistance against Sclerotinia rot in the field as well as in green house conditions (Singh *et al*., 1994). Nine genotypes *viz.,* Cutton, ZYR 6, PSM 169, PDM 169, Wester, PYM 7, Parkland, Tobin and Candle also showed resistance to Sclerotinia rot (Shivpuri *et al*., 1997). Pathak *et al.* (2002) reported that four genotypes *viz.,* PCR-10, RW-8410, RW-9401 and RGN-8006 consistently proved promising against Sclerotinia stem rot of mustard. To date, complete resistance to the pathogen has not been identified, although partial resistance was reported in *B. napus* cv. Zhongyou 827 (Buchwaldt *et al*., 2003). A diverse range of 91 accessions of *Brassica* species of Australian, Chinese and Indian origin were tested against Sclerotinia rot at seedling stage under screen house conditions by Singh *et al.* (2008). The genotypes of *B. juncea* namely, JO 009, JN 031 and JN 033 of Australian origin were observed tolerant whereas, none of the Indian and Chinese lines was tolerant. However, in *B. napus* genotypes AG outback, Rainbow, RQ 011 and RQ 011-02M2 of Australian origin, Neelam and GSL 1 of Indian origin and YU 178 of Chinese origin were tolerant. Sharma *et al.* (2009) also reported that the genotypes of *B. juncea* namely EC 597328 (Montara), EC 597329 (Berry) and EC 597331 (Ringot I) of Chinese origin were tolerant, whereas none of the Indian lines was tolerant while, among *B. napus* genotypes EC 597258 (BLN 3343) of Australian origin was observed tolerant. Ninety eight genotypes (29 *B. napus,* 69 *B. juncea*) were also tested in the field conditions by Singh *et al.*, (2010) and found that the genotypes of *B. juncea* including RH 13, Ringot 1, *B. juncea* I, *B. juncea* II from China and JM 018 from Australia showed resistance to *S. sclerotiorum.* Goyal *et al.*, (2011) screened 70 genotypes under field conditions and found that Ringot 1 showed resistance, Brassica I and Brassica II showed moderately resistance, while Haoyou II, Jinshanhung, JM 6009, JM 6010, JM 6011, JM 6012, JM 6018 showed moderately susceptible reaction and Montara, Amora, RL, JM 6026, JM 6004 and JM 6014 showed susceptible reaction. Recently, Rakesh (2014), identified fourteen genotypes *viz.,* Varuna albino, RAUD 25, BIOYSR, PHR 2, Purple mutant, Montara, Ringot 1, *Brassica* I , *Brassica* II, EC 126743, EC 126745, EC 322090, EC 322091, Kiran showed moderately resistant reaction (10-20 % D.I) against stem rot disease.

USE OF FUNGICIDES

The explosive pathogenicity of *S. sclerotiorum* under favourable conditions and the ability of its sclerotia to withstand adverse conditions allow it to be a successful pathogen. Control of this disease by the use of different fungicides with varying degree of success has been reported in the literature (Mehta *et al*., 2005) but no economical and practical solution through the use of fungicides has been made so far. Moreover, chemical sprays in mustard is not feasible and economical as this disease appears late at pod formation stage to maturity in Haryana conditions (Rathi and Singh, 2009). However, significant control of disease through prophylactic sprays with systemic fungicides has been reported under field conditions (Rathi *et al*., 2012). Although efficacy of various fungicides against *Sclerotinia* species has been well demonstrated (Rowe 1982; Brenneman *et al.,* 1987), it has not been controlled consistently and economically due to prolonged viability and unpredictable nature of fungal propagules (Singh and Kapoor 1993). Shivpuri *et al.* (2001) observed that fungicides, carbendazim, thiophenate methyl and phenylpyrrole had completely inhibited the growth of the pathogen at all the concentrations tested *in vitro*. Mancozeb was found effective at higher concentration while, copper oxychloride was least effective as it did not cause substantial reduction in growth of the pathogen and Antracol was mildly effective ex-

hibiting mean growth of 40.6 mm as against 88.3 mm in check. Sharma *et al.,* 2006 observed that captan completely inhibited mycelia growth with EC_{50} value less than 1 µg a.i. ml^{-1}. Seed treatment with carbendazim and foliar spray of the same at 65 days after sowing (DAS) proved most effective in reducing disease incidence (91%), intensity (98%) and increasing seed yield (91%) over untreated check. Seed treatment with carbendazim alone (0.1%) without any type of spray provided 56 per cent reduction in disease incidence (Ghasolia and Shivpuri, 2008). Both myceliogenic and carpogenic infections were obsreved minimum in carbendazim treatment (Sharma *et al.,* 2011). Use of carbendazim and captan as foliar sprays has been reported to be very effective against Sclerotinia rot of pea (Sharma, 1987), rape (Shen, 1993). Benomyl, carbendazim and mancozeb @ 0.2% controlled *S. sclerotiorum* on mustard and reduced the disease by 91.3, 85.7 and 54.7 per cent, respectively (Singh *et al.,* 1994). Sasirekhamani *et al.,* (2013) reported that the hexaconazole exerted an excellent fungistatic effect at 100 µg/mL concentration on *S. sclerotiorum.* Therefore, this fungicide could be effectively used for the control of the notorious pathogen, *S. sclerotiorum* at a time when many pathogens are acquiring resistance to different classes of fungicides. The prophylactic foliar spray with carbendazim @ 0.1% twice at 45 and 60 DAS was most effective in controlling Sclerotinia stem rot disease in Indian-mustard (Rathi *et al*., 2012; Rakesh *et al.,* 2016).

BIOLOGICAL CONTROL

Biological control is a promising method of control of Sclerotinia rot diseases (Bardin and Huang, 2001). The first direct application of bio-control antagonists to control plant pathogens was made by Hartley (1921) inoculating soil with 13 antagonistic fungi in an attempt to control damping off of pine seedlings (Cook and Baker, 1983).

BIO-CONTROL MECHANISMS ANTAGONISTS

S. sclerotiorum being a destructive pathogen, it over winters as sclerotia in the soil or on the plant debris. Soil microbial community plays a vital role in reducing the inoculum build up of the pathogen. Among the microbes, both fungi and bacteria play a crucial role in degrading the sclerotial bodies. Activity of microbes is in its peak near the soil surface. Diurnal fluctuation of soil temperature, moisture and relative humidity lead to the development of cracks on the sclerotial rinds. It results in leakage of cell constituents and gets parasitized by the antagonistic microbes dwelling in the soil. The mycoparasitic fungi and bacteria associated with parasitized sclerotia include *Coniothyrium minitans*, *Trichoderma* spp., *Gliocladium* spp., *Sporidesmium sclerotivorum*, *Fusarium*, *Hormodendrum*, *Mucor*, *Penicillium*, *Aspergillus*, *Stachybotrys* and *Verticillium* (Adam *et al.,* 1979; Bedi, 1963; Makkonen and Pahjakallio, 1960). Among them, *C. minitans* and *Gliocladium virens* have shown practical potential for biological control of *S. sclerotiorum* (Budge *et al.,* 1995).

FUNGAL ANTAGONISTS

C. minitans occurs naturally in soil as a mycoparasite of *S. sclerotiorum*. It was involved in the decline of viable sclerotia of *S. sclerotiorum* during crop growth and thereby suppresses the ascospores release (Whipps and Gerlagh, 1992; Sandy-winsch *et al.,* 1993). *C. minitans* was first isolated from sclerotia of *S. sclerotiorum* in 1947. It was found to be associated with different soils and on several sclerotia forming fungi (Whipps and Gerlagh, 1992; Sandy-winsch *et al.,* 1993). It parasitizes sclerotia, destroys it and reduces airborne ascospore infections. Soil application of *C. minitans* to different host crops reduced the sclerotial viability by destroying the propagation units (McLaren *et al.,* 1996). *C. minitans* is also effective under a wide range of temperature and soil humidity (Hedke and Tiedemann, 1998). In general, the use of bio-control agents is restricted to controlled environments because they need stable environmental conditions for successful establishment in the infection court so as to prevent the infection of the pathogen (Whipps, 1994). However, it suggests the scope for using *C. minitans* for field-grown crops (McLaren *et al.,* 1996; McQuilken *et al.,* 1995). Soil application of *C. minitans* as mycoparasite was effective in reducing the incidence of Sclerotinia wilt in sunflower by parasitizing the sclerotia produced in the soil and in the plant system (Huang, 1980). However, *C. minitans* was successful in parasitizing the sclerotial bodies, it was unable to prevent the Eco-friendly methods in combating *S. sclerotiorum* (Lib.) de Bary, secondary spread of the actively growing mycelium (Huang, 1980). The carpogenic germination of sclerotial bodies of *S. sclerotiorum* was reduced by the mycoparasites *C. minitans* and *Talaromyces flavus* (Huang and Erickson, 2000). However, *T. flavus* suppressed the carpogenic germination of the sclerotia, it was inferior to *C. minitans.* Combined application of both *T. flavus* and *C. minitans* was not found to exert any synergistic or additive effect in the suppression of sunflower wilt (McLaren *et al.,* 1996). Consecutive application of fungal antagonist is a pre-requisite to suppress the establishment of pathogen in the infection court. Soil application of either the above two antagonists continuously for two years suppressed sunflower wilt up to three years. But the crop raised during the subsequent year without the application of antagonist resulted in being susceptible (McLaren *et al.,* 1996). Continuous monoculture of sunflower increased the natural population of *C. minitans* and *Trichoderma* spp., which in turn reduced the severity of sunflower wilt under field conditions (Huang and Kozub, 1991).

The build up of antagonistic fungal flora during monoculture would increase the degradation of the sclerotia and thereby reduce the inoculum potential of the pathogen. In general, irrespective of the host, disease severity of *Sclerotinia* increases only during bloom stage. Hence, protection of the petals of the susceptible crops by pre-colonization of the senescing petals with antagonist will favour the multiplication of antagonists and thereby could prevent the establishment of ascospores on the infection court. Spraying spore suspension of *C. minitans* performed better in suppressing the white mold of dry bean (Huang and Kokko, 1993). However, *C. minitans* performed better in controlling *S. sclerotiorum*, its performance was found to decline when the environmental conditions favour disease development (Boland, 1989) and its consistency was not stable compared to the application of benomyl under field conditions (Huang *et al.,* 2000). Treatment of sunflower seeds infected with *S. sclerotiorum* with conidia of *C. minitans* through film coating completely suppressed apothecial production of sclerotia and killed sclerotial bodies (McQuilken and Whipps, 2001). The research on *C. minitans* (Vrije *et al.,* 2001) has led to the development of a commercial biopesticide named "Contans". Application of "Contans" recorded 60 per cent disease suppression on oilseed rape in a two year trial, but their experimental design was based on macro plots surrounded by guard areas to prevent major influences of invading external ascopores (Hedke *et al.,* 1999). Davies, (1986) found that the presence of only a few apothecia in the field might still result in relatively high disease incidence levels. Instead the antibiotic producer *Epicoccum purpurescens* was not influenced by the change in environment. Foliar application of spore suspension of *E. purpurascen* effectively controlled white mold of bean (Huang *et al.,* 2000). The disease suppression was due to the effective saprophytic colonization of petals by the antagonistic fungi. Soil incorporation of sclerotial parasite *Sporidesmium sclerotivorum* was effective up to five years in controlling Sclerotinia stem rot of soybean, a major problem in USA (Martinson and del Rio, 2001).

BACTERIAL ANTAGONISTS

Our planet is enriched with biodiversity, especially of prokaryotes. Bacterial antagonists like plant growth promoting rhizobacteria are exploited for the management of both foliar and soil borne pathogens of various economically important crop plants. Several bacterial antagonists such as *Bacillus*, *Pseudomonas* and *Agrobacterium* species are commercialized, for their potential role in disease management. But, research on the use of bacterial antagonists for the management of Sclerotinia rot fungus still remains to be explored and poorly studied. Strains of *Bacillus* spp. were frequently isolated Eco-friendly methods in combating *Sclerotinia sclerotiorum* (Lib.) de Bary from the sclerotia of *S. sclerotiorum* from North Dakota in the USA. Itadversely reduced the germination of infected sclerotia. Examination of infected sclerotia revealed that the integrity and colour of medulla was adversely affected (Wu, 1988). Spraying of *B. cereus* strain alf-87A reduced the incidence of basal pod rot of pea caused by ascospore infection of *S. sclerotiorum* (Huang *et al.,* 1993). Fifty three per cent of sclerotial bodies of *S. sclerotiorum* recovered from the soils of North Dakota were infected by *Bacillus* species. It increased degradation and reduced germination of the sclerotia (Nelson *et al.,* 2001). Antagonistic *Pseudomonas* spp. (DF41) and *P. chlororaphis* (PA23) inhibited the germination of ascospores of *S. sclerotiorum* causal agent of stem rot of canola (Savchuk and Fernando, 2004). Delivering of DF41 and PA23 on to petals increased bacterial population after 24 h and later decreased between 96 and 120 h after application. Significant differences in disease severity were found with respect to timing of ascospore applications in the control treatments (ascospores only).One isolate completely suppressed disease when co-applied with ascospores, while only minor suppression occurred when applied 24 or 48 h after. Results from all studies indicated that PA23 and DF-41 are effective bio-control agents against *S. sclerotiorum* of canola (Savchuk and Fernando, 2004). A four-year study has shown that PA23and DF41 have a wide scope for the management of canola stem rot under field conditions (Savchuk, 2002; Zhang, 2004). *Pantoea agglomerans* isolated from leaves and flowers of canola produce oxalate oxidase and degrade oxalic acid produced by *S. sclerotiorum*, the pathogencity factor required for the successful establishment of the host-parasite relationship (Savchuk and Fernando, 2004).

FUTURE WORK STRATEGY

The following issues need to be addressed for Sclerotinia rot in rapeseed mustard:

Gene expression in Sclerotinia rot pathogens and host during infection of susceptible, tolerant and resistant varieties.

Search of resistant and moderately resistance sources against Sclerotinia rot in oilseed brassicas and wild relatives.

Search for alternative control methods of Sclerotinia rot of rapeseed-mustard.

Induced resistance and systemically acquired resistance (SAR).

Best use of IPM and IDM technology.

Coordination/cooperation/interaction with other researcher including plant breeders, statistician, soil scientist and institutions.

Conclusion

There is no doubt that Sclerotinia rot is the most destructive disease of rapeseed-mustard across the world.

Sclerotinia rot causes considerable reduction in the quality and quantity of harvested product of rapeseed-mustard and no proven source of resistance has been identified among cultivated oilseed brassica crop species. Due to lack of resistance in cultivated *Brassicas* against Sclerotinia rot, other methods could be used for management of this disease. One the most commonly used methods is the use of fungicides. In spite of tremendous use of fungicides against pathogens, theses fungicides cause serious health hazards to human beings and also they cause environmental pollution. Hence, at the present time more emphasis is made on other methods of disease management like growing moderately resistant varieties, use of plant and natural products, bio-control agents and alteration in agronomic practices because they are more economical, eco-friendly and safe.

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