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REVIEW

Biology and biocontrol of *Sclerotinia sclerotiorum* (Lib.) de Bary in oilseed Brassicas

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Abstract *Sclerotinia sclerotiorum* (Lib.) de Bary is a necrotrophic plant pathogen infecting over 500 host species including oilseed *Brassicas*. The fungus forms sclerotia which are the asexual resting structures that can survive in the soil for several years and infect host plants by producing ascospores or mycelium. Therefore, disease management is difficult due to the long term survivability of sclerotia. Biological control with antagonistic fungi, including *Coniothyrium minitans* and *Trichoderma* spp, has been reported, however, efficacy of these mycoparasites is not consistent in the field. In contrast, a number of bacterial species, such as *Pseudomonas* and *Bacillus* display potential antagonism against *S. sclerotiorum*. More recently, the sclerotia-inhabiting strain *Bacillus cereus* SC-1, demonstrated potential in reducing stem rot disease incidence of canola both in controlled and natural field conditions via antibiosis. Therefore, biocontrol agents based on bacteria could pave the way for sustainable management of *S. sclerotiorum* in oilseed cropping systems.

Keywords Biology · Biocontrol · Sclerotinia · Oilseed · Brassica

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Introduction

Sclerotinia sclerotiorum (Lib.) de Bary is a polyphagous, ubiquitous, necrotrophic fungal plant pathogen with a reported host range of over 500 plant species from 75 families (Saharan and Mehta 2008). The fungus damages plant tissue, causing cell death and appears as a soft rot or white mould on the crop (Purdy 1979). *S. sclerotiorum* was initially detected from infected sunflower in 1861 (Gulya et al. 1997). The oilseed producing crops belonging to the genus *Brassica* (canola, rapeseed-mustard) are major hosts of *S. sclerotiorum* (Bailey 1996; Sharma et al. 2015a). The yield and quality of these crops are reduced by the disease which can cost millions of dollars annually (Purdy 1979; Saharan and Mehta 2008). The first report of stem rot disease in rapeseed-mustard was published by Shaw and Ajrekar (1915). Canola production has been threatened by this yield-limiting and difficult to control disease in Australia (Barbetti et al. 2014). Sclerotinia stem rot (SSR) of canola has been reported to cause significant yield losses around the world including Australia (Hind-Lanoiselet and Lewington 2004). Several attempts have been made to manage sclerotinia diseases through agronomic practices such as, the use of organic soil amendments (Asirifi et al. 1994), soil sterilization (Lynch and Ebben 1986), zero tillage and crop rotation (Adams and Ayers 1979; Duncan 2003; Morrall and Dueck 1982; Gulya et al. 1997; Yexin et al. 2011), tillage and irrigation (Bell et al. 1998). The broad host range of the pathogen has restricted the efficacy of cultural disease management practices to minimize sclerotinia infection (Saharan and Mehta 2008).

Efforts have also been made to search for resistant genotypes to SSR, however no completely resistant commercial crop cultivars have yet been developed (Hayes et al. 2010; Alvarez et al. 2012). Breeding for SSR resistance is difficult because the trait is governed by multiple genes (Fuller et al.

1984). In addition, the occurrence of virulent pathotypes has hindered the progress towards the development of complete host resistance (Barbetti et al. 2014). In Australia, a limited number of fungicides have been registered however the mismatch of appropriate time between ascospore liberation and fungicide application often led to failure of disease management (Lindbeck et al. 2014). However, biological control agents have been reported as an alternative means in controlling the infection of the white mould pathogen (Yang et al. 2009; Fernando et al. 2007; Wu et al. 2014; Kamal et al. 2015). These naturally occurring organisms can significantly reduce disease incidence by inhibiting ascospore and sclerotial germination (Fernando et al. 2007). In this review, we discuss the economic importance, infection process, pathogenicity factors, symptoms, disease cycle, epidemiology and deployment of promising microbial biocontrol strategies for sustainable management of *S. sclerotiorum*.

Economic importance

SSR has threatened the global oil seed *Brassica* production by causing substantial yield losses (Sharma et al. 2015a). The yield losses depend on disease incidence and infection at a particular plant growth stage (Saharan and Mehta 2008). Infection at pre-flowering can result in up to 100 % yield loss in infected plants (Shukla 2005). Plants usually produce little or no seed if infection occurs at the early flowering stage whereas infection at a later growth stage may cause little yield reduction. Premature shattering of siliquae, development of small, sunken and chaffy seeds in rapeseed are the yield limiting factors of *S.sclerotiorum* infection (Sharma et al. 2015a; Morrall and Dueck 1983). Historically, the estimated yield losses were reported as 28 % in Alberta and 11.1–14.9 % in Saskatchewan, Canada (Morrall et al. 1976), 5–13 % in North Dakota and 11.2–13.2 % in Minnesota, USA (Lamey et al. 1998), up to 50 % in Germany (Pope et al. 1989), 0.3 to 34.7 % in Golestan, Iran (Aghajani et al. 2008), 60 % in Rajasthan, India (Ghasolia et al. 2004), 10–80 % in China (Gao et al. 2013) and 80 % in Nepal (Chaudhury 1993). North Dakota and Minnesota faced an income loss of $24.5 million in canola during 2000 (Lamey et al. 2001). In Australia, the annual losses are estimated to be $AUD 39.9 million and the cost of fungicide to control SSR was reported to be approximately $35 per hectare (Murray and Brennan 2012). Yield loss was reported as high as 24 % (Hind-Lanoiselet et al. 2003) and 0.39–1.54 t/ha (Kirkegaard et al. 2006) in southern New South Wales (NSW), Australia. In 2013–14, a higher inoculum pressure was observed in high-rainfall zones of NSW and SSR outbreak in canola was reported to occur across the wheat belt region of Western Australia (Khangura et al. 2014).

Plant infection

The asexual resting propagules of *S. sclerotiorum*, commonly known as sclerotia, are capable of remaining viable in the soil for 5 years (Bourdôt et al. 2001). Sclerotia can germinate either myceliogenically or carpogenically with favourable environmental conditions. Saturated soil and a temperature range of 10 to 20 °C can trigger the development of apothecia (Abawi et al. 1975a). *S. sclerotiorum* is homothallic and produces ascospores after self-fertilisation without forming any asexual spores (Bourdôt et al. 2001). Apothecia are the sexual fruiting bodies which form through sclerotial germination during favourable environmental conditions and liberate ascospores that can disseminate over several kilometres through air currents (Clarkson et al. 2004). The air-borne ascospores land on petals, germinate and produce mycelium by using the senescing petals as an initial source of nutrients (McLean 1958). The presence of water on plant parts enhances the mycelia growth and infection process (Abawi et al. 1975a; Hannusch and Boland 1996). The secretion of oxalic acid and a battery of acidic lytic enzymes kill cells ahead of the advancing mycelium and causes death of cells at the infection sites (Abawi and Grogan 1979; Adams and Ayers 1979). Upon nutrient shortages the fungal mycelia aggregate and turn into melanised sclerotia which are retained in the stem or drop to the soil and remain viable as resting structures for many years. Mycelium directly arising from sclerotia is not as infective as the primary inoculum of ascospores due to its low competitive saprophytic ability (Newton and Sequeira 1972). The mycelium can directly infect host plant tissue using either enzymatic degradation or mechanical means by producing appressoria (Le Tourneau 1979; Lumsden 1979).

Pathogenicity

Being a necrotrophic fungus, *S. sclerotiorum* kills cells ahead of the advancing mycelium and extracts nutrition from dead plant tissue. Oxalic acid plays a critical role in effective pathogenicity (Cessna et al. 2000). Several extracellular lytic enzymes such as cellulases, hemi-cellulases and pectinases (Riou et al. 1991), aspartyl protease (Poussereau et al. 2001), endo-polygalacturonases (Cotton et al. 2002) and acidic protease (Girard et al. 2004) show enhanced activity and degrade cell organelles under the acidic environment provided by oxalic acid. Oxalic acid is toxic to the host tissue and sequesters calcium in the middle lamellae which disrupts the integrity of plant tissue (Bateman and Beer 1965; Godoy et al. 1990a). The reduction of extracellular pH helps to activate the secretion of cell wall degrading enzymes (Marciano et al. 1983). Suppression of an oxidative burst directly limits the host defence compounds (Cessna et al. 2000). The fungus ramifies inter or intra-cellularly colonizing tissues and kills

cells ahead of the invading hyphae through enzymatic dissolution. Pectinolytic enzymes macerate plant tissue which cause necrosis followed by subsequent plant death (Morrall et al. 1972). The release of lytic enzymes and the oxalic acid from the growing mycelium work synergistically to establish the infection (Fernando et al. 2004).

Characteristic symptoms

SSR produces typical soft rot symptoms which first appear on the leaf axils. Spores cannot infect the leaves and stems directly and must first grow on dead petals or other organic material adhering to leaves and stems (Saharan and Mehta 2008). The petals provide the necessary food source for the spores to germinate, grow and eventually penetrate the plant. Infection usually occurs at the stem branching points where the airborne spores land and droplets of water can be frequently found (Fernando et al. 2007). Rainfall or heavy dew helps to create moist conditions, which may keep leaves and stems wet for 2 to 3 days. Spores can remain alive and able to penetrate the tissue up to 21 days after liberation (Rimmer and Buchwaldt 1995). Two to three weeks after infection, soft watery lesions or areas of very light brown discolouration become obvious on the leaves, main stems and branches (Fig. 1a). Lesions expand, turn to greyish white, and may have faint concentric markings (Fig. 1b and c). Plants with girdled stems wilt prematurely ripen and become conspicuously straw-coloured in a crop that is otherwise still green (Fig. 1d and e). Infected plants may produce comparatively fewer pods per plant, fewer seeds per pod or tiny shrivelled seeds that blow out the back of the combine. The extent of damage depends on time of infection during the flowering stage as well as infection time on the main stems or branches. Severely infected crops result in lodging, shattering at swathing and are difficult to swathe. The stems of infected plants eventually become bleached and tend to shred and break. When the bleached stems of diseased plants are split open, a white mouldy growth and hard, black resting bodies (sclerotia) become visible (Fig. 1f) (Rimmer and Buchwaldt 1995; Dueck 1977).

Life cycle

S. sclerotiorum spends most of its life cycle as sclerotia in the soil. The sclerotia from infected plants can become incorporated into the soil following harvest which provides a source of inoculum for future years. Sclerotia are hard walled, melanised resting structures that are resilient to adverse conditions and remain viable in the top five centimetres of the soil for approximately 4 years and up to 10 years if buried deeper (Khangura and MacLeod 2012). Sclerotia can infect the base of the stem through direct mycelial germination in the presence of an exogenous source of energy. However, the direct myceliogenic germination of sclerotia is limited as a means of infection in oilseed *Brassicas* (Sharma et al. 2015a). Carpogenic germination of sclerotia results in the formation of apothecia which occurs after "conditioning" for at least 2 weeks at 10–15 °C in moist soil (Smith and Boland 1989). Apothecia are small mushroom-like structures that can release over two million tiny air-borne ascospore during a functioning period of 5 to 10 days (Sharma et al. 2015a). These ascospores are mainly deposited within 100 to 150 m from their origin but can travel several kilometres through air currents (Bardin and Huang 2001). The ascospores can survive on the plant surface or in the soil for 2 to 3 weeks in the absence of petals (Sharma et al. 2015a; Rimmer and Buchwaldt 1995). The petals act as a food source for the germinating spores of *S. sclerotiorum* and to establish the infection (Rimmer and Buchwaldt 1995). Infected and senescing petals then lodge on leaves, leaf axils or stem branches and commence infection as water soaked tan coloured lesions or areas of very light brown discolouration on the leaves, main stems and branches 2–3 weeks after infection. Lesions turn to greyish white covering most plant parts and eventually become bleached and tend to shred and break. At the end of growing season the fungal mycelia aggregates and develop sclerotia. These sclerotia then return to the soil on crop residues or after harvest, overwinter and the disease cycle is complete under favourable environmental conditions (Fig. 2).

Epidemiology

Several studies conducted in Australia, Canada, China, USA and Norway have demonstrated the effect the environment plays in increasing the disease severity caused by *S. sclerotiorum* (Koike 2000; Kohli and Kohn 1998; Zhao and Meng 2003). Spore dispersal usually occurs in windy, warm and dry conditions. The apothecia shrivel in dry conditions and excessive rain washes out the spore bearing fruiting body into the soil (Krüger 1975). The frequency of apothecial production was found to be similar in both saturated and unsaturated soil but moisture is essential for initial infection and disease progression (Teo and Morrall 1985; Morrall and Dueck 1982). The germination of apothecia also varies with temperature, for example the consecutive temperature of 4, 12, 18 and 24 °C is conducive to maximize germination rate (Purdy 1956). A comprehensive study demonstrated that the mean percent petal infestation was comparatively higher in the morning compared to the afternoon and was greatly favoured during lodging, but secondary infection through plant contact was limited for disease development and dissemination (Turkington et al. 1991; Venette 1998). Honey bees are also responsible for the spread of infected pollen grains causing

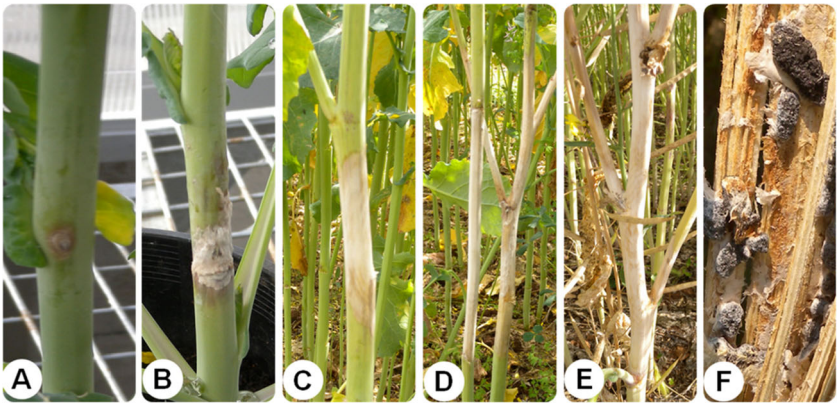

Fig. 1 Typical symptoms of sclerotinia stem rot in canola. **a** Initial lesion development on stem **b-d** Gradual lesion expansion **e** Lesion covered the whole plant and caused plant death **f** Sclerotia developed inside dead stem

pod rot of rapeseed (Stelfox et al. 1978). The spores can survive generally for 5 to 21 days on petals while survivability was found to be higher on lower and shaded leaves (Venette 1998; Caesar and Pearson 1982).

Management of *Sclerotinia*

Host resistance

S. sclerotiorum has a diverse host range and a completely resistant genotype of canola against the pathogen have not yet been reported (Fernando et al. 2007). The first report of genetic resistance against *S.sclerotiorum* was cited a century ago, observed in *Phaseolus coccineus* (Steadman 1979; Bary et al. 1887). A number of broad leaf crops are now reported to have resistance to *S. sclerotiorum*, including *Phaseolus vulgaris* (Lyons et al. 1987) and *Phaseolus coccineus* (Abawi et al. 1975b); *Solanum melongena* (Kapoor et al. 1989); *Pisum sativum* (Blanchette and Auld 1978); *Arachis hypogaea* (Coffelt and Porter 1982); *Carthamus tinctorius* (Mündel et al. 1985); *Glycine max* (Nelson et al. 1991); *Helianthus annuus* (Sedun and Brown 1989) and *Ipomoea batatas* (Wright et al. 2003). The Australian, Canadian and Indian registered canola varieties possess minimal or no resistance to *S. sclerotiorum* to date and complete resistance to the

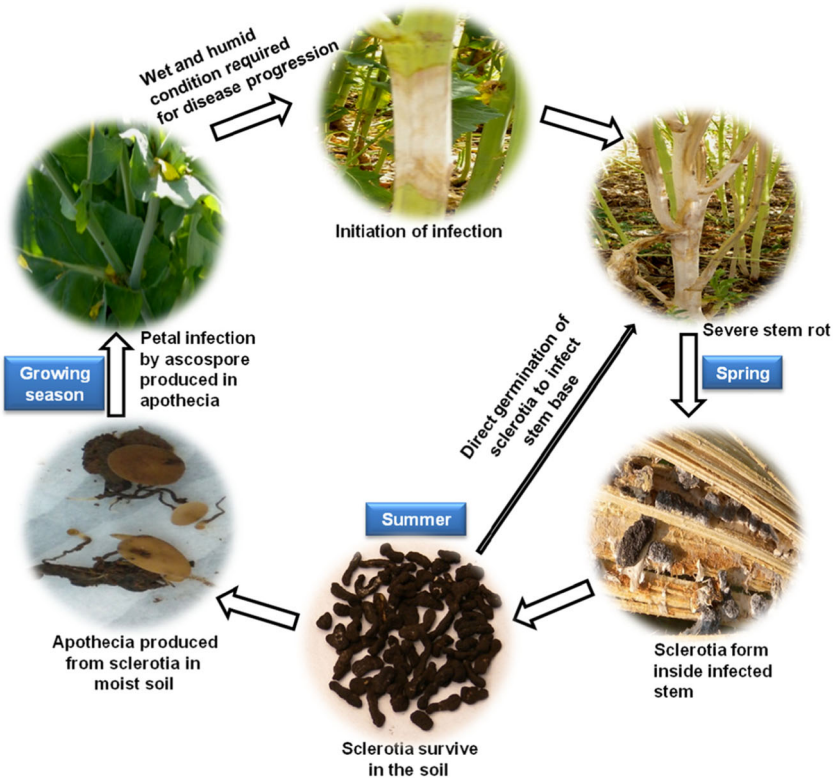

Fig. 2 Disease cycle of *Sclerotinia sclerotiorum* on canola

pathogen has not been identified (Kharbanda and Tewari 1996; Sharma et al. 2009; Li et al. 2009). In China, two canola cultivars namely Zhongyou 821 and Zhongshuang no.9 were reported to be partially resistant to *S. sclerotiorum* in addition to containing low glucosinolates and erucic acid with high yield potential (Gan et al. 1999; Buchwaldt et al. 2003; Wang et al. 2004). Also a less susceptible canola cultivar was developed against stem rot disease in France, however yields were observed to be lower under disease free conditions (Winter et al. 1993; Krueger and Stoltenberg 1983).

Cultural management

A number of cultural strategies including disease avoidance, pathogen exclusion and eradication can be used to minimise stem rot disease in canola (Kharbanda and Tewari 1996). Crop rotation is also an efficient strategy to reduce the sclerotia; but 3–4 years rotation cannot significantly eliminate the sclerotia from field (Williams and Stelfox 1980; Morrall and Dueck 1982; Bailey 1996). Furthermore, a long rotation of 5–6 years is not adequate to completely eliminate sclerotia that can survive below 20 cm of soil depth (Nelson 1998). Tillage is regarded as a potential method of minimising disease through burying of sclerotia (Gulya et al. 1997). Generally, the sclerotia are not functional if residing below 2–3 cm soil profile but exceptionally low carpogenic germination was observed at 5 cm soil depth (Kurle et al. 2001; Abawi and Grogan 1979; Duncan 2003). The survival of sclerotia is prolonged when buried near the soil surface (Gracia-Garza et al. 2002; Merriman et al. 1979). Burying of sclerotia through deep ploughing reduced germination and restricted the production of apothecia (Williams and Stelfox 1980). On the other hand, stem rot incidence was found to be greater when fields are cultivated with a mouldboard plough compared with zero tillage (Mueller et al. 2002b; Kurle et al. 2001). However, minimum or zero tillage may create a more competitive and antagonistic environment as well as restrict the ability of the pathogen to survive (Bailey and Lazarovits 2003).

Chemical control

The application of foliar fungicides is a widely used practice to manage SSR (Hind-Lanoiselet and Lewington 2004; Hind-Lanoiselet et al. 2008). The efficacy of foliar fungicides depends on several factors including time of application, crop phenology, weather conditions, disease cycle, spraying coverage, protection duration (Mueller et al. 2002c; Hunter et al. 1978; Mueller et al. 2002a). The general reason behind poor management of disease is poor timing of the fungicide application (Mueller et al. 2002a; Hunter et al. 1978; Steadman 1983) where protectant fungicides are recommended to be applied at the pre-infection stage (Rimmer and Buchwaldt 1995; Steadman 1979). The early blooming stage before petal fall is the best time to spray a foliar fungicide for a significant reduction in disease incidence (Dueckz and Sedun 1983; Morrall and Dueck 1982; Rimmer and Buchwaldt 1995; Dueck et al. 1983). The fungicides widely used against sclerotinia in Canada are Benlate (benomyl), Ronilan (vinclozolin), Rovral (iprodione), Quadris (azoxystrobin), Sumisclex (procymidone), Fluazinam (shirlan) and cyprodinil plus fludioxonil (Switch). Fungicides registered in Australia include Rovral Liquid, Chief 250, Iprodione Liquid 250, Corvette Liquid (iprodione), Fortress 500 (procymidone), Sumisclex 500, Sumisclex Broadacre (procymidone) and Prosaro® (prothioconazole and tebuconazole) (Annonymous 2001; Hind-Lanoiselet et al. 2008). The registration of Benlate was ceased in Canada due to public health concerns and crop damage caused by the fungicide as claimed by canola growers (Gilmour 2001).

Biological control

The use of microorganisms to suppress plant diseases was observed nearly a century ago and since then plant pathologists have attempted to apply naturally occurring biocontrol agents for managing important plant diseases. Over 40 microbial species have been explored and studied for managing *S. sclerotiorum* (Li et al. 2006). Since its first report in 1837, a total of 185 studies have been reported on mycoparasitism and biocontrol of the pathogen (Sharma et al. 2015b). The interest in biocontrol of *Sclerotinia* diseases has increased over the last few decades as chemical pesticides failed to properly control the pathogen and concerns of their impact on the environment (Saharan and Mehta 2008; Agrios 2005). Key strategies for potential biocontrol of *Sclerotinia* diseases include a reduction in the density of primary and secondary inoculum by killing sclerotia or restricting germination, infection in the rhizosphere and phyllosphere, as well as reduction in virulence (Saharan and Mehta 2008). Several procedures including mycelial and sclerotial baiting as well as direct isolation from the natural habitat have been used to explore biocontrol organisms against *Sclerotinia* spp (Sandys-Winsch et al. 1994). A wide range of micro-organisms have been screened, recovered from the rhizosphere, phylosphere, sclerotia and other habitats to detect antagonism that are suitable potential biocontrol agents. Screening of antagonistic organisms in soil or on plant tissue instead of artificial nutrient media have been shown to better predict the potential of the agent as field assays are expensive and impractical for large numbers of isolates (Sandys-Winsch et al. 1994; Andrews 1992; Whipps 1987). More than 100 species of fungal and bacteria biocontrol agents have been identified against *Sclerotinia*. These biocontrol agents parasitise, reduce, weaken or kill sclerotia as well as protect

plants from ascospore infection (Mukerji et al. 1999; Saharan and Mehta 2008).

Fungal antagonists

Several sclerotial mycoparasitic fungi have been studied to control *S. sclerotiorim* for example, *Coniothyrium minitans*, *Trichoderma* spp., *Gliocladium* spp., *Sporidesmium sclerotivorum*, *Talaromyces flavus*, *Epicoccum purpurescens*, *Streptomyces* sp., *Fusarium*, *Hormodendrum*, *Mucor*, *Penicillium*, *Aspergillus*, *Stachybotrys* and *Verticillium* (Adams 1979; Saharan and Mehta 2008). The sclerotial mycoparasite *C. minitans* was discovered by Campbell (1947) and is considered as the most studied and widely available fungal biocontrol agent against *S. sclerotiorum* (Whipps et al. 2008). Application of *C. minitans* to the soil can infect and destroy sclerotia of *S. sclerotiorum*, resulting in reduced carpogenic germination and viability of sclerotia (McLaren et al. 1996). Sclerotial destruction by *C. minitans* was observed when 1×10^9 viable conidia was applied against *S. sclerotiorum* in different hosts including oilseed rape (Luth 2001). Soil incorporation of *C. minitans* 3 months before planting to 5 cm depth allows maximum colonisation and degradation of sclerotia (Peltier et al. 2012). The commercial formulation of *C. minitans* has been registered in many countries including Germany, Belgium, France and Russia under various trade names (De Vrije et al. 2001). The use of *C. minitans* to control sclerotinia diseases has been extensively reviewed by Whipps et al. (2008).

T. harzianum has been reported to reduce linear growth of mycelium and apothecial production of *S. sclerotiorum* as well as reducing the lesion length and disease incidence when applied simultaneously, or 7 days prior to pathogen inoculation under glass house conditions (Mehta et al. 2012). Reduction of mycelia growth was also observed through culture filtrates of *T. harzianum* and *T. viride* (Srinivasan et al. 2001). The use of *T. harzianum* as a soil inoculant, seed treatment and foliar spray, singly or in combination, showed significant efficacy against *S. sclerotiorum* in mustard (Meena et al. 2014). Application of *T. harzianum* isolate GR in soil and farm yard manure infested with *T. harzianum* isolate SI-02 minimised disease incidence by 69 and 60.8 %, respectively (Meena et al. 2009). Seed treatment with *T. harzianum* and foliar sprays with garlic bulb extract, not only significantly reduced disease incidence, but also provided higher economic return (Meena et al. 2011). *T. harzianum* and *T. viride* significantly reduced disease incidence when mustard seeds were treated with chemicals prior to soil treatment of microbes (Pathak et al. 2001). Rhizosphere inhabiting strains of *T. harzianum* and *Aspergillus* sp. also showed inhibitory effect against the pathogen (Rodriguez and Godeas 2001). The mycoparasite S*poridesmium sclerotivorum*, detected in soils of several states of the USA, has been considered as a promising invader of sclerotia. Soil incorporation of the sclerotial parasite *S. sclerotivorum* and *Teratosperma oligocladum* caused 95 % reduction in inoculum density (Uecker et al. 1978; Adams and Ayers 1981). The unique characteristics of *S. sclerotivorum* is its ability to grow from one sclerotium to another through soil, producing many new conidia throughout the soil mass which are able to infect surrounding sclerotia (Ayers and Adams 1979). Application of 100 spores of *S. sclerotivorum* per gram of soil caused a significant decline in the survival of sclerotia (Adams and Ayers 1981).

Gliocladium virens has also been evaluated as a mycoparasite of both mycelia and sclerotia of *S. sclerotiorum* (Phillips 1986; Tu 1980; Whipps and Budge 1990). Sclerotial damage was found to occur over a wide range of soil moistures and pH (5–8), but bio-activity was reduced at temperatures below 15 °C (Phillips 1986). The type and quality of substrates used to raise the *G. virens* inoculum affected its ability to infect and reduced the viability of sclerotia (Whipps and Budge 1990). The use of sand and spores as substrate and inoculum, respectively, has provided the easiest method for screening *G. virens* as a mycoparasite (Whipps and Budge 1990). Other researchers also indicated that the ability of various strains of *G. virens* to parasitise *S. sclerotiorum* varied, and that strain selection will play an important role in the mycoparasite-pathogen interaction (Phillips 1986; Tu 1980). *G. virens* has great potential as a mycoparasite due to its ability to grow and sporulate quickly, spread rapidly and produce metabolites such as glioviren, an antibiotic with significant antagonistic properties (Howell and Stipanovic 1995). Other *Gliocladium* spp. including *G. roseum and G. catenulatum* can parasitise the hyphae and sclerotia of *S. sclerotiorum*as well as produce toxins and cell wall degrading enzymes such as β-(1-3)-glucanases and chitinase (Huang 1978; Pachenari and Dix 1980).

Bacterial antagonists

Plant growth promoting rhizobacteria have been exploited for sustainable management of both foliar and soil borne plant pathogens. Some antagonistic bacteria belonging to *Bacillus, Pseudomonas, Burkholderia* and *Agrobacterium* species are commercially available for their potential role in disease management (Fernando et al. 2004). A number of bacterial isolates have been investigated against *S. sclerotiorum* for their potential antagonistic properties. Research on the application of antagonistic bacteria for the control of *S. sclerotiorum* still demands to be fully explored (Boyetchko 1999). The gram positive *Bacillus* spp are often considered as potential biocontrol agents against foliar and soil borne plant diseases (McSpadden Gardener and Driks 2004; Jacobsen et al. 2004). *Bacillus* species have been less studied than *Pseudomonas* but their ubiquity in soil, greater thermal tolerance, rapid multiplication in liquid culture and easy

formulation of resistant spores has made them potential biocontrol candidates (Shoda 2000).

Bacillus cereus strain SC-1 isolated from the sclerotia of *S. sclerotiorum* shows strong antifungal activity against canola stem rot and lettuce drop disease caused by *S. sclerotiorum* (Kamal et al.: Bacterial biocontrol of sclerotinia diseases in Australia (unpublished data); Kamal et al. 2015). Dual cultures have demonstrated that *B. cereus* SC-1 significantly suppressed hyphal growth, inhibited the germination of sclerotia and was able to protect cotyledons of canola from infection in the glasshouse. Significant reduction in the incidence of canola stem rot was observed both in glasshouse and field trials (Kamal et al. 2015). In addition, *B. cereus* SC-1 showed 100 % disease protection against lettuce drop caused by *S. sclerotiorum* in glasshouse studies. The biocontrol mechanism of *B. cereus* SC-1 was observed to be through antibiosis. PCR amplification of genomic DNA using gene-specific primers revealed that *B. cereus* SC-1 contains four antibiotic biosynthetic operons responsible for the production of bacillomycin D, iturin A, surfactin and fengycin. Mycolytic enzymes of chitinase and β-1,3-glucanase corresponding genes were also documented. Sclerotia submerged in cell free culture filtrates of *B. cereus* SC-1 for 10 days showed degradation of melanin, the formation of pores on the surface of the sclerotia and failure to germinate. Significant reduction in lesion development caused by *S. sclerotiorum* was observed when culture filtrates were applied to 10 day old canola cotyledons. Histological studies using scanning electron microscopy of the zone of interaction of *B. cereus* SC-1 and *S. sclerotiorum* demonstrated restricted hyphal growth and vacuolated hyphae with loss of cytoplasm (Fig. 3a). In addition, cells of the sclerotial rind layer were ruptured and heavy colonisation of bacterial cells was observed (Fig. 3b). Transmission electron microscopy studies revealed that the cell content of fungal mycelium and the organelles in sclerotial cells were completely disintegrated, suggesting the direct antifungal action of *B.cereus* SC-1 metabolites on cellular components through lipopeptide antibiotics and mycolytic enzymes (Kamal et al.: Elucidating the mechanism of biocontrol of *Bacillus cereus* SC-1 against *Sclerotinia sclerotiorum* (unpublished data)).

An endophytic bacterium, *B. subtilis* strain EDR4, was reported to inhibit hyphal growth and sclerotial germination of *S. sclerotiorum* in rapeseed. The maximum inhibition was observed at the same time of inoculation either by cell suspension or cell free culture filtrates. Two applications of EDR4 at initiation of flowering and full bloom stage in the field resulted in the best control efficiency. Scanning electron microscopy showed that strain EDR4 caused leakage, vacuolization and disintegration of hyphal cytoplasm as well as delayed the formation of infection cushion (Chen et al. 2014). Another endophytic strain of *B. subtilis* Em7 has performed a broad antifungal spectrum on mycelium growth and sclerotial

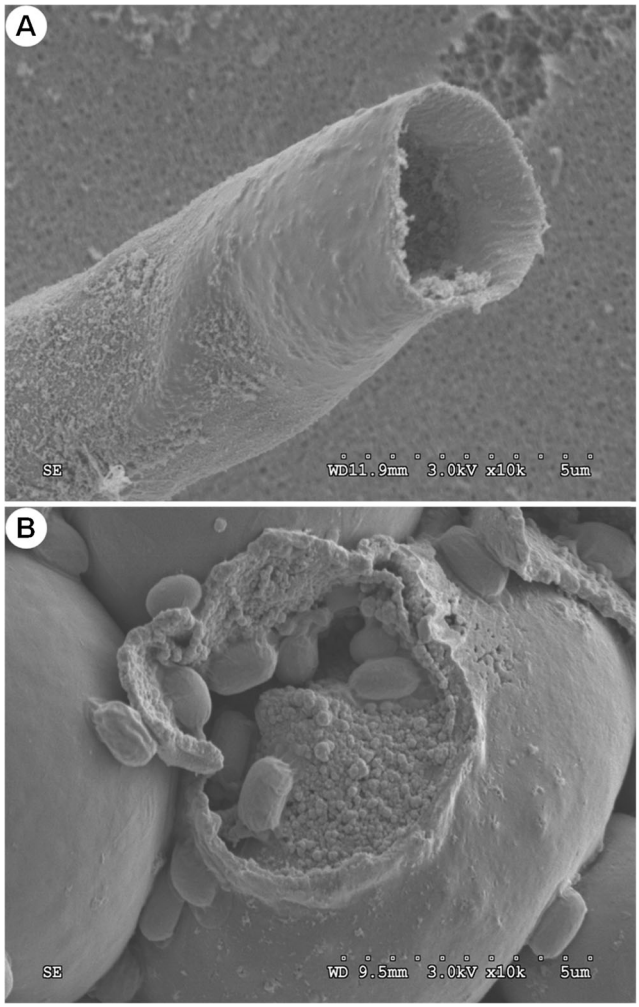

Fig. 3 SEM observation demonstrated (**a**) vacuolated hyphae and (**d**) perforated sclerotial rind cell of *Sclerotinia sclerotiorum* challenged by *Bacillus cereus* SC-1. Figure produced from Kamal et al.: Elucidating the mechanism of biocontrol of *Bacillus cereus* SC-1 against *Sclerotinia sclerotiorum* (unpublished data)

germination *invitro* and significantly reduced stem rot disease incidence in the field by 50–70 %. The strain Em7 caused leakage and swelling of hyphal cytoplasm and subsequent disintegration and collapse of the cytoplasm (Gao et al. 2013).

The application of *B. subtilis* BY-2 was demonstrated to suppress *S. sclerotiorum* on oilseed rape in the field in China. The strain BY-2 as a coated seed treatment formulation or sprays at flowering or combined application of both treatments provided significant reduction of disease incidence (Hu et al. 2013a). In addition, *B. subtilis* Tu-100, a genetically distinct strain also demonstrated its efficacy against SSR of oilseed rape in Wuhan, China (Hu et al. 2013a). Evaluation of cell suspension, broth culture and cell-free filtrate derived from *B. subtilis* strain SB24 showed significant suppression of SSR of soybean under control glass house conditions. The strain SB24 originating from soybean root showed maximum disease reduction at 2 days prior to inoculation of *S. sclerotiorum* (Zhang and Xue

2010). The plant-growth promoting bacterium *Bacillus megaterium* A6 isolated from the rhizosphere of oilseed rape was shown to suppress *S. sclerotiorum* in the field. The isolate A6 was applied as pellet and wrap seed treatment formulations and produced comparable reduction in disease as the chemical control (Hu et al. 2013b).

Furthermore, another attempt to control white mould with *B. subtilis* showed inconsistent results between fields. Spraying of *Bacillus* in soil significantly reduced the formation of apothecia and thereby reduced yield losses in oilseed rape (Lüth et al. 1993). Members of *Bacillus* species showed reduced disease severity and inhibited ascospore germination of *S. sclerotiorum* due to pre-colonization of petals when treated 24 h before ascospore inoculation of canola (Fernando et al. 2004). *Bacillus amyloliquefaciens* (strains BS6 and E16) performed better than other strains under the greenhouse environment in spray trials against *S. sclerotiorum* in canola (Zhang 2004). The results demonstrated that both strains reduced stem rot by 60 % when applied at 10^{-8} cfu mL^{-1} at 30 % bloom stage. In addition, HPLC analysis revealed that defence associated secondary metabolites in leaves of canola were found to increase after inoculation, which might supress the germination of ascospores (Zhang et al. 2004).

In North Dakota the sclerotia associated with *Bacillus* spp. showed reduced germination and the sclerotial medulla was infected by the bacterium (Wu 1988). Further studies revealed that more than half of the total number of sclerotia recovered from the soil were infected by *Bacillus* spp. contributing to their degradation and inhibition of germination (Nelson et al. 2001). In western Canada, 92 canola associated bacterial strains belonging to *Pseudomonas*, *Xanthomonas*, *Burkholderia*, and *Bacillus* were selected for biocontrol activity in vitro against *S. sclerotiorum* and other canola pathogens. The bacteria were able to protect the root and crowns of susceptible plants from infection more efficiently than fungal antagonists by inhibiting myceliogenic sclerotial germination and limiting ascospore production (Saharan and Mehta 2008). The bacterial antagonist, *Bacillus polymixa* has also been demonstrated to reduce the growth of *S. sclerotiorum* under controlled environment conditions (Godoy et al. 1990b).

Biological control against SSR of canola using bacterial isolates of *Pseudomonas chlororaphis* PA-23 and *Pseudomonas* sp. DF41 was also demonstrated in vitro through the inhibition of mycelial growth and sclerotial germination in canola (Savchuk 2002). Inconsistent results were observed in the green house and field where both of the strains suppressed the disease through reductions in the germination of ascospores (Savchuk 2002; Savchuk and Dilantha Fernando 2004). Several plant pathogens including *S. sclerotiorum* have been controlled successfully through antibiotics extracted from *P. chlororaphis* PA23 which demonstrated inhibition of sclerotia and spore germination, hyphal lysis, vacuolation, and protoplast leakage (Zhang and Fernando 2004a). Synthesis of two antibiotics namely phenazine and pyrrolnitrin from the PA23 strain involved in the inhibitory action was confirmed through molecular studies (Zhang 2004; Zhang and Fernando 2004b). The results recommended that *P. chlororaphis* strain PA23 might potentially be used against *S. sclerotiorum* and several other soil-borne pathogens. Bacterial strains *Pseudomonas aurantiaca* DF200 and *P. chlororaphis* (Biotype-D) DF209 isolated from canola stubble generated a number of organic volatile compounds in vitro including benzothiazole, cyclohexanol, n-decanal, dimethyl trisulphate, 2-ethyl 1-hexanol and nonanal (Fernando et al. 2005). These compounds inhibited the mycelial growth as well as reduced sclerotia and ascospore germination both in vitro and in soil. Both strains released volatiles into the soil which interrupted sclerotial carpogenic germination and prevented ascospore liberation (Fernando et al. 2005). *Pantoea agglomerans* formerly known as *Enterobacter agglomerans,* is a gram negative bacterium isolated from canola petals and has demonstrated a capacity to produce the enzyme oxalate oxidase which can successfully inhibit the pathogenic establishment of *S. sclerotiorum* through oxalic acid degradation (Savchuk and Dilantha Fernando 2004).

A number of *Burkholderia* species have been considered as beneficial organisms in the natural environment (Heungens and Parke 2000; Li et al. 2002; Meyer et al. 2001; Parke and Gurian-Sherman 2001; McLoughlin et al. 1992; Hebbar et al. 1994; Bevivino 2000; Mao et al. 1998; Jayaswal et al. 1993; Kang et al. 1998; Meyers et al. 1987; Pedersen et al. 1999; Bevivino et al. 2005; Chiarini et al. 2006). The antimicrobial activity and plant growth promoting capability of *B. cepacia* isolates depends on a number of beneficial properties such as indoleacetic acid production, atmospheric nitrogen fixation and the generation of various antimicrobial compounds, such as cepacin, cepaciamide, cepacidines, altericidins, pyrrolnitrin, quinolones, phenazine, siderophores and alipopeptide (Parke and Gurian-Sherman 2001). In the early 1990s four *B. cepacia* strains obtained registration from the environmental protection agency (USA) for use as biopesticides which were later classified as *B. ambifaria* and one as *B. cepacia* (Parke and Gurian-Sherman 2001; McLoughlin et al. 1992). The bacterial antagonist *Erwinia herbicola* has also been demonstrated to reduce the growth of *S. sclerotiorum* under controlled environment conditions (Godoy et al. 1990b).

Mycoviruses

As the name explains, mycoviruses are viruses that inhabit and affect fungi. They are either pathogenic or symbiotic and differ from other viruses due to lack an extracellular stage in their life cycle and harbour entire life in the fungal cytoplasm (Cañizares et al. 2014). The potential of hypovirulence-associated mycoviruses including *S. sclerotiorum* hypovirulence-associated DNA virus 1 (SsHADV-1), *Sclerotinia debilitation*-associated RNA virus (SsDRV),

Sclerotinia sclerotiorum RNA virus L (SsRV-L), *Sclerotinia sclerotiorum* hypovirus 1 (SsHV-1), *Sclerotinia sclerotiorum* mitoviruses 1 and 2 (SsMV-1, SsMV-2), and *Sclerotinia sclerotiorum* partitivirus S (SsPV-S) have attracted much attention for biological control of *Sclerotinia* induced plant diseases (Jiang et al. 2013). The mixed infections of these mycoviruses are commonly observed with higher infection incidence on *Sclerotinia*. Recent investigation revealed that some hypovirulence-associated mycoviruses are capable of virocontrol of stem rot of oilseed rape under natural field condition (Xie and Jiang 2014). *S. sclerotiorum* hypovirulence-associated DNA virus 1 (SsHADV-1) was discovered as the first DNA mycovirus to infect the fungus and confer hypovirulence (Yu et al. 2010). Strong infectivity of purified SsHADV-1 particle on healthy hyphae of *S. sclerotiorum* was observed (Yu et al. 2013). Application of SsHADV-1infected hyphal fragment suspension of *S. sclerotiorum* on blooming rapeseed plants significantly reduced stem rot disease incidence and severity as well as increased seed yield. Moreover, SsHADV-1 was recovered from sclerotia that were collected from previously infected hyphal fragment applied field indicated that SsHADV-1might transmitted into other vegetative incompatible individuals. The direct applicatioin of SsHADV-1 ahead of virulent strain of *S. sclerotiorum* on leaves of *Arabidopsis thaliana* protected plants against the pathogen (Yu et al. 2013). *S. sclerotiorum* debilitation-associated RNA virus (SsDRV) and *S. sclerotiorum* RNA virus L (SsRV-L) was shown to coinfect isolate Ep-1PN of *S. sclerotiorum* (Xie et al. 2006; Liu et al. 2009). Further intensive investigation of *Sclerotinia* mycovirus system could help for better understanding of mycovirology and their potential application as biocontrol agent. The production of mycovirues could affect the economic viability of this approach to biological control.

Conclusion

The advantages of biological control over other types of disease management includes sustainable control of the target pathogen, limited side effects, selective to target pathogen, self-perpetuating organisms, non-recurring cost and risk level of agent identified prior to introduction (Pal and Gardener 2006). Biocontrol agents are more environmentally friendly because they tend to be endemic in most regions. No significant negative effects on the environment have been reported when antagonistic microorganism have increased in the soil (Cook and Baker 1983). The major limitation of biological control is that it demands more technical expertise, intensive management and planning, sufficient time even years to establish and most importantly the environmental conditions often exclude some agents (Cook and Baker 1983). Biocontrol agents are likely to perform inconsistently under different environment and this may explain why the performance of *C. minitans* against *S. sclerotiorum* was not sustainable in natural field conditions (Fernando et al. 2004). However, *Bacillus* spp which are less sensitive to environmental conditions are well adapted in rhizosphere and are able to successfully manage soil borne pathogens including *S. sclerotiorum*. Future research could be directed towards purification of antimicrobial compounds released from biocontrol agents and their exploitation as fungicides.

The cosmopolitan plant pathogen *S. sclerotiorum* is challenging the available control strategies. The broad host range and prolonged survival of resting structures has led to difficulties in consistent, economical management of the disease. It is necessary to design an innovative management strategy that can destroy sclerotia in soil and protect canola petals, leaves and stems from ascospores infection. A number of mycoparasites have demonstrated significant antagonism against *S. sclerotiorum* and reduced disease incidence. *C. minitans* is the pioneer mycoparasite that has been commercially available as Contans WG® for the management of the white mould fungus, however, the commercial product has performed inconsistently under field conditions.

The use of bacterial antagonists to combat the white mould fungus is limited compared to mycoparasites. Very recently our lab has explored the sclerotia inhabiting bacterium *B. cereus* SC-1 which suppressed Sclerotinia infection in canola both under greenhouse and field conditions (Kamal et al. 2015). The bacterium was demonstrated to produce multiple antibiotics and mycolytic enzymes and also provided broad spectrum activity against Sclerotinia lettuce drop (Kamal et al.: Elucidating the mechanism of biocontrol of *Bacillus cereus* SC-1 against *Sclerotinia sclerotiorum* (unpublished data)). Histological studies demonstrated that *S. sclerotiorum* treated with *B. cereus* SC-1 resulted in restricted hyphal growth and vacuolated hyphae void of cytoplasm. Heavy proliferation of bacterial cells on sclerotia and a complete destruction of the sclerotial rind layer were also observed. The disintegration of mycelial cell content and sclerotial cell organelles was the direct outcome of the antifungal activity of *B. cereus* SC-1 through lipopeptide antibiotics and mycolytic enzymes (Kamal et al.: Elucidating the mechanism of biocontrol of *Bacillus cereus* SC-1 against *Sclerotinia sclerotiorum* (unpublished data)). Owing to the benefits of antagonists, development of commercial formulations with promising agents could pave the way for sustainable management of *S. sclerotiorum* in various cropping systems including oilseed *Brassicas*.

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