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Sclerotinia sclerotiorum (Lib.) de Bary causing Sclerotinia rot in oilseed *Brassicas*: A review

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Abstract

Sclerotinia sclerotiorum (Lib.) de Bary, the causal organism of stem rot of *Brassica* and over 500 host plants is distributed worldwide. Sclerotinia rot is menace to cultivation of oilseed *Brassica* crops in the world. Infection occurs on leaves, stems and pods at different developmental stages, causing seed yield losses of up to 80%, as well as significant reductions in oil content and quality. The initial mycelial infection at the base of the stem is an appearance of elongated water-soaked lesions that expand rapidly. Ascosporic (carpogenic) infection is quite general and occurs on the leaves or leaf axil. Effective pathogenesis by *S. sclerotiorum* requires the secretion of pathogenicity factors including oxalic acid and extracellular lytic enzymes. Germination of overwintered sclerotia, and release, survival and germination of ascospores are important factors for the development of disease and in the life cycle of this pathogen. Isolates of *S.sclerotiorum* show a high level of morphological variability and molecular diversity. Management of *S. sclerotiorum* is a major challenge, and the best being the integration of various IPM measures. Partial resistance has been identified in some *Brassica napus* and, *B. juncea*, genotypes, though, wild *Brassicas* show better resistant reactions. This review summerizes current information on biology, physiology, epidemiology and molecular aspects of pathogenicity. In addition, current tools for research and stratagies to combat *S. sclerotiorum* have also been discussed.

***Key words*:** *Brassica* spp, epidemiology, host resistance, management, pathogenicity, physiology, *Sclerotinia sclerotiorum,* symptoms, variability.

Introduction

The brassicaceae family, to which the genus *Brassica* belongs, contains many important species yielding high quality edible and industrial oils, common vegetables and weeds. The Brassica crops are grown in tropical as well as in temperate zones, and prefer cool moist weather during growing period, and dry weather during harvesting. The oil yielding *Brassica* crops grown in India include rai or raya or mustard [*Brassica juncea* (L.) Czern. & Coss.], and rapeseed (*B. rapa* sp. *oleifera*] with three varieties: *B. rapa* var. Yellow Sarson; *B. rapa* var. Brown Sarson and *B. rapa* var. Toria. The other *Brassica* species grown to a limited extent in different parts of the country include *B. napus, B. chinensis, B. pekinensis* and *B. tournefortii.* The Indian mustard (*B. juncea*) is the main source of cooking oil in Asia. India is one of the leading oilseeds producing country in the world accounting for 11.12 per cent of the world's rapeseed-mustard production, and ranks third in the world next to China and Canada. In India, oilseed *Brassicas* are grown over an area of about 6.3 million hectares with an

annual production of 7.4 million tonnes and an average yield of 1176kg/ha *(www.drmr.res.in;* Kumar, 2014). The severe attack of many diseases not only deteriorates the quality of the seed, but also reduces the oil content considerably. Various endeavours including expansion of cropping area by diversification in agriculture, improved methods of cultivation, proper fertilization and use of improved varieties are currently being used to increase the production and productivity of various oilseed crucifers. Unfortunately, diseases and insect pests are the important limiting factors. More than thirty diseases are known to occur on *Brassica* crops in India (Saharan *et al*., 2005). Amongst the major fungal pathogens *Sclerotinia sclerotiorum* (Lib) de Bary, the causal organism of Sclerotinia rot (SR) is the most ubiquitous, omnivorous, soil-borne and destructive plant pathogen distributed worldwide. The pathogen is known to infect over 500 plant species of diverse phylogenetic backgrounds including 278 genera in 75 families of dicotyledonous, and a number of significant monocotyledonous plants (Purdy, 1979; Willetts and Wong 1980; Steadman, 1983; Boland and Hall, 1994; Saharan and Mehta, 2008; Sharma, 2014). Sclerotinia rot is more common and severe in temperate and subtropical regions of cool and wet seasons (Purdy, 1979; Willets andWong, 1980; Saharan and Mehta, 2008).

Economic importance

Sclerotinia stem rot, although, occurs most frequently in cool and moist regions (Purdy 1979; Saharan and Mehta, 2008), it has also been reported in some semi-arid regions where conditions seem unfavourable for disease development. Infection by *S. sclerotiorum,* a necrotrophic pathogen with a wide host range results in damage of the plant tissue, followed by cell death and development of soft rot or white mould (Purdy, 1979). Yield losses vary with the percentage of plants infected, and the growth stage of the crop at the time of infection. Plants infected at the early flowering stage produce little or no seeds, and those infected at the late flowering stage will set seed and may suffer little yield reduction. Sclerotinia stem rot was first reported by Shaw and Ajrekar (1915) on several host plants including rapeseed-mustard. Since then, frequent occurrences of the disease in mild to severe form have been reported from Brazil (Neto, 1955), Canada (Dueck and Morrall, 1971; Platford and Branier, 1975; Morrall *et al*., 1976; Dueck, 1977), China (Yang, 1959), Denmark (Buchwald, 1947), Finland (Jamalainen, 1954), France (Hims, 1979a), Germany (Krüger, 1976), India (Butler and Bisby, 1960; Roy and Saikia, 1976; Saharan *et al*., 1985; Saharan, 1992), Sweden (Loof and Appleqvist, 1972), and United Kingdom (Rawlinson and Muthyalu, 1979; Hims, 1979b). Disease outbreaks even in the drier areas occurr in irrigated fields since irrigation provides favourable conditions for disease development even though the macroclimatic conditions were unfavourable. Yield losses due to SR in susceptible crops vary and may be as high as 100 per cent (Purdy, 1979). The shattering of prematurely-ripened seed pods before harvest, and loss of quality in the form of smaller, shrunken and chaffy seeds especially in rapeseed has been observed. Reported yield loss estimates due to SR in rapeseed varied from very heavy in Germany (Horning, 1983), 11.4-14.9 per cent in Saskatchewan, Canada (Morrall *et al.,* 1976), 5-13 per cent in North Dakota, and 11.2-13.2 per cent in Minnesota, USA during 1991-1997 (Lamey *et al*., 1998). In central and eastern parts of Finland, losses by SR were so great that the cultivation of rapeseed is considered beneficial only in the southern and western areas (Jamalainen, 1954). In addition to causing 75 per cent yield loss, SR in Nepal also significantly reduced plant height, number of siliquae/plant and 1000 seed weight (Chaudhary, 1993). In NSW, Australia, yield losses due to SR in *B. napus* varied from 0.39 to 1.54 t/ha (Kirkegaard *et al*., 2006).

In India, during the eightees and ninetees, the SR disease in rapseed-mustard was of very minor importance, because the mycelial infection at the ground level occurred very infrequently only on the isolated plants. Significant increase in the sclerotial population in the soil due to monocropping and cultivation of rapeseed-mustard under irrigated conditions, has made SR very serious disease of oilseed *Brassica* crops in states including Rajasthan, Haryana, Punjab, Assam, West Bengal, Madhya Pradesh, Uttar Pradesh, and Bihar (Aggarwal *et al*., 1997; Saharan and Mehta, 2002). In fact, the disease incidence upto 80% has been reported in

Punjab and Haryana (Kang and Chahal, 2000; Sharma *et al.,* 2001), and 72% in Uttar Pradesh (Chauhan *et al.,* 1992). Kumar and Thakur (2000) from Himachal Pradesh have reported that stem rot appears regularly in mild to severe form in major mustard growing areas and cause considerable loss in yield. In Rajasthan, 60% seed yield loss has been reported in severely infected plants (Krishnia *et al*., 2000; Ghasolia *et al.,* 2004). It is also one of the most devastating diseases in China, causing yield losses between 10 to 80% with low oil quality (Oil Crop Research Institute, Chinese Academy of Sciences, 1975). Sclerotinia rot is also a serious threat to oilseed rape production with substantial yield losses worldwide including Australia, Europe, India and North America (McCartney and Lacey, 1999; Hind *et al.,* 2003; Sprague and Stewart-Wade, 2002; Koch *et al.,* 2007; Malvarez *et al.,* 2007; Singh *et al.,* 2008; Saharan and Mehta, 2008). The quality of the seed has also been adversely affected in partially infected plants. According to Shukla (2005), plants infected at or before flower initiation, can result in 100 per cent yield loss, where as the plants infected after flowering suffer only 50 per cent yield loss.

History, Host Range and Nomenclature

History: The SR or white stem rot is caused by *Sclerotinia sclerotiorum* (Libert) de Bary [Syn. *S. libertiana* Fuckel; *Whetzelinia sclerotiorum* (Lib.) Korf and Dumont]. The pathogen was first described from Belgium by Madame M. A. Libert (1837) as *Peziza sclerotiorum* Libert (Libert, 1837). This binomial for the fungus stood until Fuckel (Fuckel, 1870) erected and chose to honour Madame Libert by renaming *Peziza sclerotiorum* with a newly coined binomial, *Sclerotinia libertiana* Fuckel. According to Wakefield (1924), Fuckel apparently disliked the combination *S. sclerotiorum* and elected to establish the new one. Authors in the United States, and elsewhere, accepted and used *S. libertiana* Fuckel until Wakefield (1924) showed it to be inconsistent with the International Rules of Botanical Nomenclature, and cited G. E. Massee as the proper authority for *Sclerotinia sclerotiorum* (Lib.) Massee, because he had used that binomial in 1895. However, since de Bary used it in his contributions (de Bary *et al.,* 1884; de Bary, 1886), the name and the authority for the fungus has generally been accepted to be *Sclerotinia sclerotiorum* (Lib.) de Bary.

Host range: *Sclerotinia sclerotiorum* appears to be among the most nonspecific, omnivorous and successful plant pathogen. The broad host range itself makes control of disease in agricultural crops very difficult, because it restricts the number of non-host crops that can be included in crop rotations. Records of susceptible hosts of this pathogen are scattered throughout the published scientific literature. Partyka and Mai (1962) indicated that 172 species from 118 genera in 37 plant families are known to be susceptible hosts. Farr *et al*., (1989) listed 148 genera of plants that are susceptible to *S. sclerotiorum.* Schwartz (1977) reported a host range of 374 plant species from 237 genera in 65 families. Purdy (1979) referred to a compilation by P.B. Adams that included 361 species from 225 genera in 64 families. The most recent host index for *S. sclerotiorum* prepared by Boland and Hall (1994) contains 42 subspecies or varieties, 408 species, 278 genera, and 75 families of plants.

Nomenclature: *S. scleotiorum* belongs to kingdom 'fungi', phylum 'ascomycota', class 'discomycetes', order 'Heliotiales', and family 'sclerotiniaceae'. Species produce inoperculate asci from brownish sitipitate apothecia that arise from sclerotial stromata within or associated with a host plant (Whetzel, 1945). Hyphae are hyaline, septate, multinucleate, thin walled (9-18 µm) in width and branching is never at right angles. Mycelia may appear white to tan in culture. Individual sclerotia are embedded in white mycelial net and are round, semi spherical to irregular in shape, measuring 2-10 x 3-15 mm in size. Sexually produced apothecia are cup shaped with concave disc, light yellowish brown, and vary in size from 2-11 mm (average 4-5mm) in diameter. Apothecia are formed on a slender stalk of 20-80 mm in length called stipe (Kosasih and Willets, 1975). Asci are arranged on periphery of ascocarp, measuring 119-162.4x6.4-10.9µ in size, and are inoperculate, cylindrical, narrow, rounded at the apex with eight ascospores per ascus. Ascospores are uniform, hyaline, ellipsoid with smooth walls, measuring 10.2-14.0µ x 6.4-7.7µ in

size, and each containing 8 chromosomes.

Taxonomic decisions are based upon observation and evaluation of characters falling into four principal categories: macroscopic, cultural, biological and microscopic. The publication of Nannfeldt in 1932 entitled "Studien fiber die Morphologie und Systematik der nicht lichenisierten inoperculaten Discomyceten" revolutionized the description and classification of discomycetes by introducing micro anatomical studies of sterile tissues as a source of additional taxonomic characters. Using pre-Nannfeldt characters, as employed by many workers who described species of *Sclerotinia,* a description of a species was limited to the following range of characters:

1. Macroscopic characters, such as colour, size and shape of the apothecium, stipe and sclerotium. Cultural characters, often the size and distribution of sclerotia on agar plates.

2. Biological characters, such as host, season and part of substrate invaded.

3. Microscopic characters, usually limited to the size, shape and colour of the ascospores, asci and paraphyses.

Although these characters are useful, and indeed several have been heavily weighted in making the taxonomic decisions, the micro anatomical characters introduced by Nannfeldt in his classification offer further information on zones of the apothecium, stipe and sclerotium in addition to the hymenium, which has long been the center of attention. The sterile zones of the apothecium and sclerotium show diverse and distinctive tissue types including the sub-hymenium, the medullary excipulum, and the ectal excipulum subdivided into three component zones; the margin, the flank, the stipe, including any hairs, as in the Sclerotiniaceae, tomentum hyphae (Korf, 1973).

The tissue types of the apothecial and sclerotial zones are characterized within the genus *Sclerotinia.* The sub-hymenium, a compact zone of interwoven prosenchyma, is usually brown-walled and bound in gel. The medullary excipulum is composed of loosely interwoven textura intricate oriented more or less parallel to the surface of the apothecium. The most characteristic zone, the ectal excipulum is composed of textura prismatica which turns out at the apothecial margin perpendicular to the apothecial surface, and further down the flank, develops into textura globulosa as cells become inflated, round off, and somewhat disarticulated. Globose cells, and often tomentum hyphae produce from globose cells, comprise the ectal excipulum of the stipe and are often brown-walled. The sclerotial medulla in *Sclerotinia* does not include suscept tissues, but is composed of hyaline textura oblita with heavily gelatinized hyphal walls (composed of â-1, 3-g1ucans and proteins) as reported by Saito (1977). The sclerotial rind is composed of the apices of the medullary cells, which grow perpendicularly to the sclerotial surface and develop into textura prismatica. Pigmentation of the rind cells may occur in the walls of a two to six deep layers of the outermost cells. All species of *Sclerotinia* show a positive reaction of the ascus pore channel wall in Melzer's Reagent (0.5 g iodine, 1.5 g potassium iodide, 20 g chloral hydrate and 20 ml distilled water). Dimorphism in spore size has been observed by Kohn (1979) in one species as it has for some species of *Monilinia* (Woronin, 1888) and in *Sclerotinia allii* (Sawada, 1919), which is a species of *Ciborinia.* Kohn (1992) suggested some new characters for fungal systematic, which can also be used for *Sclerotinia* taxonomy to resolve the disputed points:

However, Ekins *et al*. (2005) suggested comparison of characters like host species, sclerotial diameter, ascosporic morphism and breeding type, and RFLP probes for separating *S. minor* from *S. sclerotiorum* and *S. trifoliorum*.

Phylogeny of *Sclerotinia* and related genera: Phylogenies have been constructed based on nuclear ribosomal internal transcribed spacer (ITS) DNA sequences from an in-group consisting of 50 isolates representing 24 species of the discomycete family Sclerotiniaceae and an out-group consisting of five related taxa of the same family. The in-group taxa are: 3 *Botrytis* spp., 2 *Botryotinia* spp., 1 *Ciborinia* sp., 1 *Dumontinia* sp., 1 *Grovesinia* sp., 6 *Myriosclerotinia* spp., 9 *Sclerotinia* spp. and

New characters		Expected resolution level
Morphological		
	Histochemistry	Species, Genus, family
	Ultra structure	Any level
	Anamorph connections	Genus, family
	Anamorph morphology	Species, Genus
Genetic		
	Ability to mate and form viable F1	Species
	Vegetative incompatibility	Intra-specific
	Mycelial inter-sterility	Species
Biological		
	Host or substrate	Inter-specific, species, Genus
	Biogeography	Any level
Molecular - Proteins – Immunology		Any level
	- Sequencing	Any level
	- Isozyme electrophoresis	Population, intra-specific, species
DNA - Restriction analysis – RFLPs		Intra-specific, species (any level)
	- Restriction mapping	Any level
PCR - Length polymorphism		Any level
	- Restriction analysis	Any level
	- Direct sequencing	Any level
	- RAPD	Intra-specific (genetic)

1 *Sclerotium* sp. The out-group taxa are: 1 *Ciboria* sp., 1 *Encoelia* sp. and 3 *Monilinia* spp. The type species is included for all taxa except for *Ciborinia* and *Encoelia*. Several of the included taxa are important plant pathogens. The resulting phylogenies are discussed with regard to morphology, life history and taxonomy. A suspected relationship between *Sclerotinia borealis* and *S. tetraspora*, and *Myriosclerotinia* is rejected, while a suspected relationship between *Ciborinia ciborium* and *Myriosclerotinia* is strongly supported. *Sclerotinia ulmariae*, previously synonymized with *Dumontinia tuberosa*, is reinstated as an independent species of *Dumontinia*. Two new combinations, *Dumontinia ulmariae* and *Myriosclerotinia ciborium* are proposed. The imperfectly known taxon *Sclerotium cepivorum* seems most closely related to *Dumontinia*. It is concluded that *Dumontinia* and *Myriosclerotinia* are monophyletic, and that *Botryotinia* along with *Botrytis* anamorphs probably also constitute a monophyletic lineage. The genus *Sclerotinia* is probably polyphyletic and characterized by simple isomorphies rather than synapomorphies. Two new taxa, *Sclerotinia* sp.1 and *Sclerotinia* sp. 2, are most closely related to *S. minor*, *S. sclerotiorum* and *S. trifoliorum* and to *S. borealis*, respectively (Holst-Jensen *et al*., 1998).

Pathogenicity factors

Sclerotinia sclerotiorum is responsible to secrete multiple pathogenicity factors. Degradation of plant cell wall, its components and tissue maceration occur by the concerted action of several extracellular lytic enzymes. Effective pathogenesis by *S. sclerotiorum* requires the secretion of oxalic acid (Cessna *et al.,* 2000), extracellular lytic enzymes including cellulases, hemicellulases 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). These enzymes are highly active under the acidic conditions provided by oxalic acid and degrade the plant cell wall and tissues beneath it. 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; Godoy *et al*., 1990). Reduction in extracellular pH, activates the production of cell wall degrading enzymes (Marciano *et al*., 1983). Oxalic acid (OA) directly limits host defense compounds by suppressing the oxidative burst. In conjunction, plant cell wall-degrading enzymes, including cellulolytic and pectinolytic, cause maceration of plant tissues, and necrosis followed by plant death (Collmer and Keen, 1986). Thus, the release of an array of lytic enzymes and the oxalic acid from the growing mycelium are the important pathogenicity factors that are required for the establishment of the host-parasite relationship. However, *S. sclerotiorum* is poorly characterized at the molecular level and only a few genes encoding hydrolytic enzymes (Reymond *et al*., 1994; Fraissinet-Tachet *et al*., 1995; Fraissinet-Tachet and Fevre, 1996; Poussereau *et al*., 2001; Cotton *et al*., 2002; Li *et al*., 2004) have been reported. Expressed sequence tag (EST) analysis has proved to be an efficient approach to identify genes expressed under a wide variety of conditions in other systems (Adams *et al*., 1991). Indeed, cDNAs encoding four endo-(SSPG1063, SSPG544, SSPG427 and ZY210R), and two exo-polygalacturonases (SSPG851 and SSPG1033) were found; SSPG1063, an endo-polygalacturonase, denoted as SSPG1d, was nearly identical to SSPG1a–c (Reymond *et al*., 1994; Cotton *et al*., 2002) and BcPG1, which are responsible for full pathogenicity of *Botrytis cinerea* (ten Have *et al*., 1998). SSPG1 has also been implicated in the initiation and establishment of the infection as well as lesion progression by *S. sclerotiorum* in *B. napus* (Li *et al*., 2004).

Sclerotia

The primary survival (overwintering) structure of *S. sclerotiorum* is the sclerotium. Sclerotium is a hard resting structure consisting of a light colored interior called medulla, and an exterior black protective covering the rind. The rind contains melanin pigments which are highly resistant to degradation, while the medulla consists of fungal cells rich in β-glucans and proteins. In *S. sclerotiorum*, sclerotial development can be divided into three distinguishable stages (Townsend and Willetts, 1954): (i) initiation, the appearance of small distinct initial forms of interwoven hyphae; which develop terminally by repeated branching of long, aerial, primary hyphae, ii) development, increase in size, and iii) maturation, characterized by surface delimitation, internal consolidation, and melanization, and often associated with droplet secretion. These phases are accompanied by both morphological and biochemical differentiations. The initiation and maturation stages of sclerotial development are affected by numerous factors, including photoperiod, temperature, oxygen concentration, mechanical factors, and nutrients (Chet and Henis, 1975). The production of OA has been correlated with sclerotial development which is known to be an important factor in pathogenicity of *S. sclerotiorum* (Donaldson *et al*., 2001; Zhou and Boland, 1999).

Sclerotial development is a complex, multistage process that is thought to be regulated by signal-transduction pathways such as MAPK and PKA (Chen and Dickman, 2005; Chen *et al*., 2004; Harel *et al*., 2005; Rollins and Dickman, 1998). Recently, evidence has been produced for the existence of calcineurin-MAPK and calcineurin-PKA-associated pathways. For example, in *S. cerevisiae*, PKA has been shown to phosphorylate and, consequently, negatively regulate the activity of the calcineurin-regulated Zn-finger transcription factor Crz1p by inhibiting its nuclear import (Kafadar and Cyert, 2004). In human cells, transcriptional activity of NFATc2 (aCrz1p homolog) is unregulated by phosphorylation of the MAPK JNK (Ortega-Perez *et al*., 2005). If similar pathways exist in *S. sclerotiorum*, current analysis demonstrating calcineurin playing a significant role in the regulation of morphogenesis and pathogenesis in this pathogen may require further dissection of these pathways. Understanding of the physiological and molecular mechanisms involved in sclerotial development and pathogenicity of *S. sclerotiorum*

may well reflect the development and pathogenesis of other sclerotium-producing fungi and may provide new avenues for intervention in these processes, leading to improved control of diseases caused by other sclerotium-producing fungi.

The basic disease cycle of *Sclerotinia* begins with the overwintering of sclerotia in the soil. Sclerotia are conditioned to germinate by the overwintering process. At certain times during the growing season, depending on the inherent nature of the fungus and the various environmental factors, the overwintered sclerotia can germinate in one of two methods. Probably the most common is carpogenic germination which results in the production of a small mushroom called an apothecium. Carpogenic germination usually requires the sclerotia to be in wet soil for one to two weeks prior to germination. The apothecia produce ascospores which are ejected into the environment. Most ascospores fall on susceptible plants in the immediate vicinity of the apothecia, but some can travel long distances by wind. The requirement of moisture and relatively cool temperatures under the plant canopy for carpogenic germination and growth of the pathogen are reasons why rainy periods or irrigation are associated with outbreaks of disease on most crops. The other method of germination is myceliogenic, where the sclerotium produces mycelium. Infection of host plants by mycelium often occurs at or beneath the soil-line. Sclerotia germinate in the presence of exogenous nutrients and produce hyphae which invade nonliving organic matter, forming mycelium which then infects living host tissues (Saharan and Mehta, 2008).

There are many factors affecting survival of the sclerotia including soil type, previous crops, and environmental conditions, but how and to what degree they affect survival is not well understood. It has also been observed that type of the soil as well as frequency and amount of irrigation play an important role both in germination of sclerotia and in development of apothecium. It has been reported that least number of apothecia was recorded in the sandy soil whereas sandy loam soil resulted in production of maximum number of apothecia (Mehta *et al*., 2009). Further, it was observed that flooding of the field (once in week) prior to sowing resulted in least disease incidence and minimum lesion length. The optimum irrigation applied once in 3 or 7 days intervals also had low disease intensity as compared to control (Mehta *et al*., 2009).

High temperature and high soil moisture combined are probably the two most deleterious environmental factors. Microbial degradation, however, is the principal reason for a decline in the populations of sclerotia. There are many fungi, bacteria and other soil organisms that parasitize or utilize sclerotia as carbon sources. One reason that crop rotation is recommended for *Sclerotinia* is to allow the natural microbial population to degrade sclerotia. 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. *Sclerotinia sclerotiorum* is genetically variable (Carpenter *et al.,* 1999), and sclerotia of different geographic origin are known to have different carpogenic germination temperature optima (Huang and Kozub, 1991). Temperature and soil moisture are key factors affecting carpogenic germination of *S. sclerotiorum* (Phillips, 1987; Clarkson *et al*., 2001). Carpogenic germination of *S. sclerotiorum* sclerotia has been studied widely (Schwartz and Steadman, 1978; Phillips, 1986, 1987; Huang and Kozub, 1991, 1994; Dillard *et al*., 1995, Sun and Yang, 2000; Thaning and Nilsson, 2000; Ekins *et al*., 2002; Hao *et al* 2003; Clarkson *et al*., 2004).

Apothecia formation

The structure and development of of *S. sclerotiorum* apothecium, has been well documented by Saito, (1973), Jones, (1974), Kosasih and Willetts (1975) and Jayachandran *et al*. (1987). Production of apothecia requires mature and preconditioning of sclerotia for at least two weeks at 10-15 ºC in moist soil with nonliving food base in rhizosphere within top 2 cm of the soil surface (Abawi and Grogan, 1979). Minimum or shallow cultivation places many sclerotia 0.25 to 1.25 inch depth which is optimum for emergence of sexual fruiting bodies called apothecia. Apothecia are generally produced after

a certain dormancy period during which the sclerotia are chilled or frozen. Cold temperature seems to be a predominate factor in "conditioning" sclerotia to produce apothecia when soil conditions are suitable with >50% field capacity moisture, and 15-17 ºC temperature for 10-14 days. Sharma and Meena (2011) observed apothecia in *B. juncea* field during favourable weather conditions including 17.5 ºC maximum, 4.4 ºC minimum, RH 98.3%, low sunshine hours of 4.0, and 16% soil moisture (Fig. 1j). Carpogenic germination begins with the active fungal growth in the regions of the sclerotial cortex or medulla. Growing fungal cells form dense primordia which break through the rind of the sclerotium, and continue growth as tube-shaped stalks called stipes (Fig.1k). After the stipes emerge from the soil, they continue to grow upward to a height of about 1 cm and if they are exposed to ultraviolet light (<390 nm), they differentiate into apothecia. In the formation of an apothecium, the tip of the stipe expands to form a top surface made up of the hymenium and hypothecium, layers which are supported by the tapering tissues of the former stipe, now called the excipulum, giving the overall structure the appearance of a tan- to buff-colored trumpet bell or a golf tee. In the hymenium are born numerous asci and sterile supportive hairlike structures, the paraphyses. In the asci, sexual recombination occurs, and the products are eight ascospores, neatly lined-up near the tip of each ascus. A vacuole, which is responsible for increasing the hydrostatic pressure within the ascus, forms below the string of ascospores. As the hydrostatic pressure builds, the ascus expands, but its lateral expansion is restricted by the paraphyses and neighboring asci. Therefore, most expansion is near the tip, and expansion continues until each ascus protrudes beyond the paraphyses. At some point, the pressure exceeds, the supportable wall stretches and the ascus explodes.

Ascospore

Ascospores release and their survival are important factors for the development of disease and in the life cycle of this pathogen. Induced forcible ejection or 'puffing' of ascospores was first photographed by Dickson and Fisher (1923) followed by Harthill and Underhill (1976). This puffing phenomenon has also been observed in several other ascospores-producing fungi using a 'spore clock' apparatus developed by Ingold (1971), who was able to induce the puffing phenomenon by sudden changes in environmental conditions including light and dark period, or a decrease in relative humidity and temperature. Each apothecium can produce from 2 to 30 million ascospores over a period of several days. Ascospores are covered with sticky mucilage which could either be residue of the liquid from asci, or part of the cell wall. The mucilage not only cements the spore to any object it contacts, it also glues spores together in clumps. In laboratory conditions, dried frozen *Sclerotinia* ascospores have remained viable for years, but freshly produced spores generally survive 5 to 21 days depending on relative humidity (Wu, 1988). Puffing of spore clouds by apothecia of *S. sclerotiorum* is easily induced by removing the lid of a sealed container containing apothecia in a saturated environment. Ascospores of *Sclerotinia* are physically shot out from the surface of the hymenium in an upward direction. The display is very spectacular because tens of thousands of asci fire their ascospores almost simultaneously producing a large puff of spores. In bright light, the hundreds of thousands of spores can be seen with the naked eye as a smoke-like cloud. The kick from the firing is often strong enough to shake the entire apothecium, and in quiet air, the spores are propelled several centimeters above the hymenium. In the field, spore discharge often occurs for several hours beginning about noon, and the timing is an important adaptation for increased spore dispersal. Stipes are positively phototropic because they align the hymenium with the strongest light source. The tips of the asci are also positively phototropic which helps fine-tune the trajectory of spores for maximum dispersal potential. The sun warms the soil creating thermal air turbulence that helps carry spores out of the canopy. The change in temperature also brings about a change in relative humidity which can trigger puffing. Puffing phenomenon often reported to occur due to a sudden decrease in humidity or pressure. Although, it has been observed on occasion (Newton and Seqeuira, 1972), it is questionable whether such sudden changes happen regularly in the field.

Although forcible ejection by puffing seems to be the main method of spore discharge in many ascomyctes in *S. sclerotiorum*, ascospores release is vey poorly understood. This is perhaps because it is generally observed in the laboratory when contained apothecia are suddenly exposed to the air. The information on the effect of environmental factors affecting survival of ascospores is also very scant, although Caesar and Pearson (1983) reported reduction of ascospores survival at high temperature and RH. Ascospore release and survival are the key stages in the lifecycle of *S. sclerotiorum,* and hence information on environmental factors affecting these stages could not only increase our understanding of the pathogen, but will also be important in future disease control strategies. High RH has also been reported to reduce spore survival by other researchers (Partyka and Mai, 1962; Abawi and Grogan, 1975). The factors affecting the availability of viable ascospore inoculum in the field are complex. Ascospore release in the field takes place during the night or day over a range of conditions in a continuous manner during the life of the apothecium. The effect of soil moisture which, when high, may also help maintain a functioning apothecium (Raynal, 1990). The production of stipes requires continuous moisture at the optimum temperature, which is dependent on the origin and conditions during the production and development of sclerotia (Phillips, 1987; Huang and Kozub, 1991). Although, light is not required for forming stipe initials, light of 390 nm is necessary for complete expansion of apothecia (Thaning and Nilsson, 2000, Hao *et al*., 2003). Apothecia puff-off ascospore (up to $2.3x10^6$ ascospores per apothecium) (Schwartz and Steadman, 1978) clouds during a sudden decrease in atmospheric humidity or pressure (Dickson and Fisher, 1923; Harthill and Underhill, 1976), and their release continues for at least 72–84 h (Clarkson *et al*., 2004). Knowledge of ascus/ascospore maturation process not only will increase our ability to predict the airborne inoculum levels in agricultural systems, but also improve techniques to produce large quantities of ascospores of similar age and genetic background. Although, both ascospores and mycelium of *S. sclerotiorum* often are used as a source of inoculum in artificial inoculations (Whipps *et al*., 2002, Zhao and Meng, 2003a), using ascospore inoculum is preferable for resistance screening because ascospore infection mimics the infections in the field (Whipps *et al*., 2002). Because of the difficulties in preparing a large number of uniformly aged ascospores, sporulating apothecia are usually used as inoculum source in small scale experiments (Hudyncia *et al*., 2000). For large scale experiments, large number of sclerotia are often buried in soil and appropriate conditions provided for production of apothecia and release of ascospores. Ascospores stored dry at low temperature are known to remain viable and infective for up to 2 years (Hunter *et al*., 1982). Even careful planning to collect large number of ascospores does not ensure the uniformity of the age of ascospores. Molecular studies on *S. sclerotiorum* so far have relied on studying the mutants or transformants of the mycelial phase for this genus (Godoy *et al*., 1990; Boland, 1992; Melzer and Boland, 1996; Zhou and Boland, 1997; Rollins and Dickman 1998, 2001; Deng *et al*., 2002; Rollins, 2003; Girard *et al*., 2004; Jurick *et al*., 2004), or analyzing gene functions by placing target genes into other fungi (Vautard-Mey *et al*., 1999; Vacher *et al*., 2003). Mutagenesis (including transformation) and screening for mutants at the mycelial stage are not efficient in this group of fungi because of the multinucleate nature of their hyphal cells (up to hundreds of nuclei per cell). Uniform ascospores are desirable for transformation because of their relatively identical and simple genetic background.

Pathogen diversity

Sclerotinia sclerotiorum [(Lib.) de Bary], unlike many fungal species, has been taxonomically well-defined by a wide range of criteria, including morphological features (Willetts and Wong, 1980), isoenzymes, DNA restriction fragment length polymorphisms (RFLP) (Kohn *et al.*, 1988), and random amplified polymorphic DNA (RAPD) (Sharma *et al.,* 2009c). Geographical isolates are known to have morphological variability and a high level of intraspecific phenotypic variability (Fig.1 n, o) (DRMR, 2010-11; Purdy, 1979). Morrall *et al.,* (1972) grew 114 isolates from 23 host plants in Saskatchewan, Canada on a glucose-salt agar medium and noted large variations in numbers, shape,

size and texture of sclerotia, and six other characteristics. Kohn (1995) also revealed genetic heterogenity, with numerous clones in *S. sclerotiorum* isolates from *B. napus*. Kohli *et al*., (1995) also reported that *S. sclerotiorum* isolates from *Ranunculus ficaria* showed remarkable phenotypic variability in mycelia growth rate, pigmentation, and amount of aerial mycelium, compared to the generally uniform appearance of isolates from canola in Canada and Norway. This study clearly showed that two populations isolated in Norway were quite different morphologically from populations found in canola crops of Canada and Norway regions.

Rational, systematic definition of intraspecific groupings based on factors such as pathogenicity, virulence, or geographic distribution has been difficult. This is not only because of problem with assay procedures, but also due to lack of independent criteria including mycelial or vegetative incompatibility or molecular-genetic marker, which characterize intraspecific heterogeneity. Once, such heterogeneity is identified, groups can be defined, and the phenotypes of these groups, influencing aggressiveness and host specificity, can be described. Mycelial compatibility, the ability of two strains of filamentous fungi to anastomose and form one continuous colony, is synonymous with vegetative compatibility (Fig.1m). A distinction must be maintained between vegetative and heterokaryon compatibility unless it is known that two strains not only anastomose but also form a stable heterokaryon. As an easy test for self-recognition, vegetative compatibility has been extremely useful in intraspecific strain comparisons. The deployment of vegetative and heterokaryon compatibility testing, as well as the mechanisms behind these phenomena, have been amply reviewed (Leslie, 1993; Glass and Kuldau, 1992; Glass *et al*., 2000, Saupe, 2000) and continue to be very interesting areas of research. Kohn *et al.,* (1990) reported existence of a high level of mycelial incompatibility among strain and genetic heterogeneity within the species of *S. sclerotiorum*; mycelial compatibility-incompatibility interaction may be an effective method of categorizing this heterogeneity. There is no physiological specialization found in this species (Mordue and Holliday, 1976), but strains can differ in their reaction to various hosts and one strain can be highly virulent to some hosts but mildly so against the other. Differentiation of *S. sclerotiorum* strains is based generally on morphological differences in sclerotia, mycelial growth, and ascospoers (Anderson *et al.,* 1987). Cultural variability revealed that potato dextrose agar medium was the best medium for both mycelia growth and production of maximum number of sclerotia. Differences in the morphology of isolates have been observed by Li *et al*., (2003) and Garrabrandt *et al*., (1983) who also identified isolates producing tan coloured sclerotia. Very few reports exist describing darkly-pigmented isolates of *S. sclerotiorum*, such as those reported from Canada and South-Western region of the USA (Lazarovits *et al*., 2000; Sanogo and Puppala, 2007). Reliable methods for identification and differenation of strains are locking. Molecular biology approaches revealing similarity and differences between different modifications of DNA analyses seem to be effective. Using RAPD analyses, Sharma *et al.* (2013) observed high polymorphism among seventeen geographical isolates (Fig.1p, r).

Determining morphological variability through mycelium compatibility among *S. sclerotiorum* isolates, Akarm *et al*. (2008) observed the combination with antagonheristic reactions with each other by forming a thin band of living or dead mycelia. After demonstrating that MCG and DNA fingerprint were linked in clonal populations of *S. sclerotiorum*, several researchers deployed MCG typing in population studies of both *S. sclerotiorum* and *S. minor* (Kohn *et al*., 1991; Kohli *et al*., 1992; Kohli *et al*., 1995; Cubeta *et al*., 1997; Carpenter *et al*., 1999; Carbone and Kohn 2001; Hambleton *et al*., 2002, Phillips *et al*., 2002; Durman *et al*., 2003; Hollowell *et al*., 2003; Kull *et al*., 2004; Atallah *et al*., 2004; Sexton and Howlett, 2004). Genetic diversity among *S. sclerotiorum* isolates not related to the pathogenicity of the pathogen has also ben reported (Cubeta *et al*., 1997; Sun *et al*., 2005; Malvarez *et al*., 2007). Utilising microsatellite markers, Sexton *et al*. (2006) demonstrated genetic diversity among *S. sclerotiorum* isolates from oilseed rape in South-East Australia.

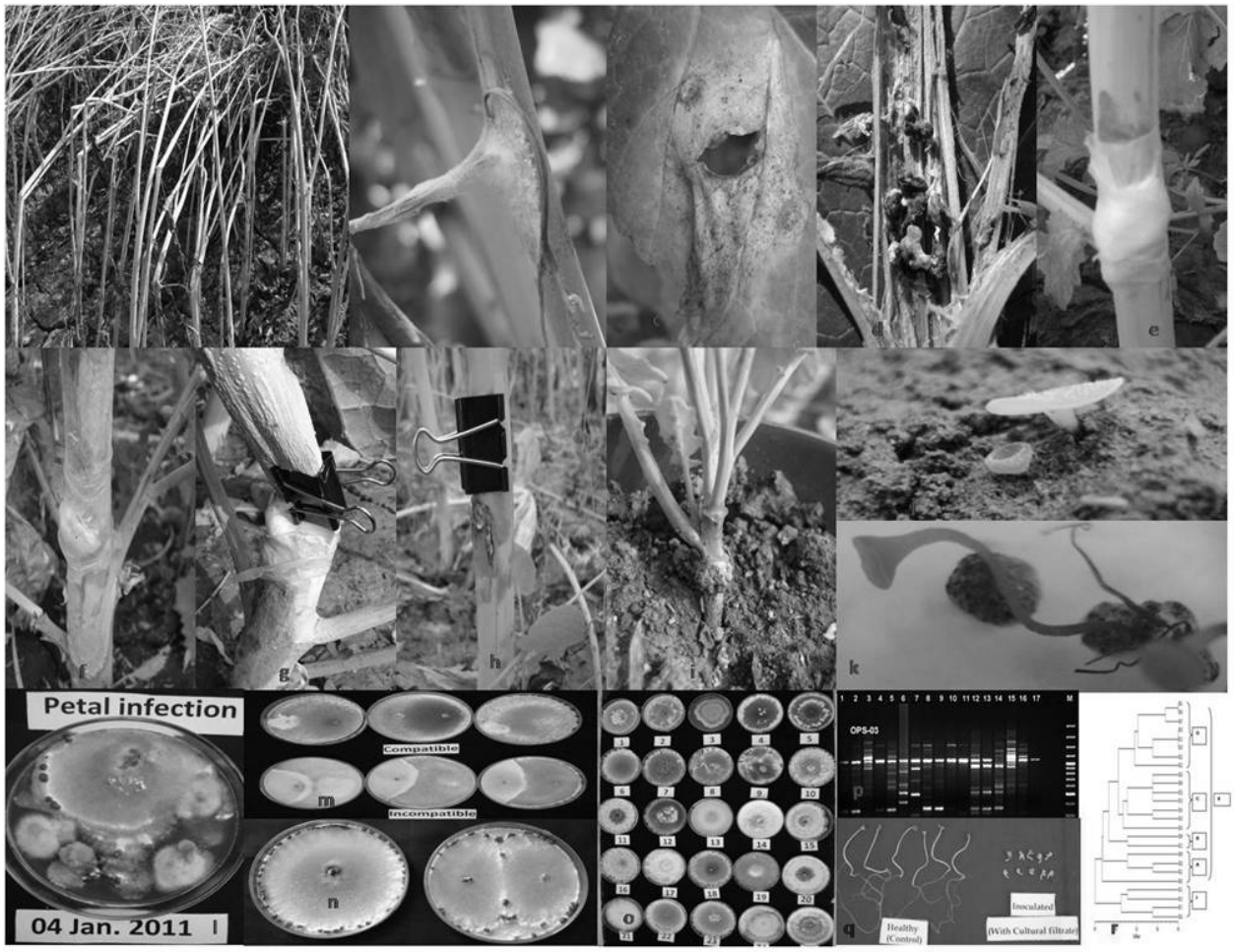

Fig.1. a: *Sclerotinia* infected sick field; b: symptoms on stem; c: shot hole symptom on leaf; d: sclerotia formation in stem; e: stem inoculation technique; f: disease progress on inoculated stem; g: highly susceptible *Brassica* accession; h: tolerant reaction; i: tolerant reaction on wild *Brassica*; j: apothecia formation in field; k: apothecia formation *invitro*; l: petal infection with ascospores; m: compitable and incompitable (MCG) reaction; n: sclerotia formation; o: morphological variability; p: molecular diversity; q:effect of culture filtrate on seed germination and seedling vigour; r: dendogram showing grouping of isolates (Photo: Dr. Pankaj Sharma, DRMR, Bharatpur)

Hypovirulence of *Sclerotinia*

Hypovirulence refers to the reduced ability of select isolates, within a fungal plant pathogen population to infect, colonize, kill, and (or) reproduce on susceptible host tissues (Elliston, 1982). This may also be associated with other phenotypic characters including reduced growth rate or sporulation, and altered colony morphology or color. Hypovirulence has been reported to occur in *S. sclerotiorum*, *S. minor* and *S. homoeocarpa* and, to varying degrees, has been associated with the presence of dsRNAs. One hypovirulent *S. sclerotiorum* isolate containing varying numbers of dsRNA elements, grew slowly in culture, developed typical colony morphology, produced significantly smaller lesions on celery than virulent isolates (Boland, 1992). Treatment with cyclohexamide and (or) heat, followed by hyphal tip subculturing, to recover an isolate that was free of dsRNA were not successful. The hypovirulent phenotype and

dsRNA were transferred to vegetatively compatible recipient isolates through hyphal anastomosis, and recipient isolates developed the hypovirulent phenotype with dsRNA. Other isolates of the pathogen also contained dsRNA, but there was no correlation between the presence of dsRNAs and reduced virulence (Boland, 1992; Zhou and Boland, 1998). Therefore, associations between dsRNAs and hypovirulent phenotypes are specific to individual dsRNAs and preclude any general observations on the presence or absence of dsRNA and their association with fungal phenotypes.

Symptoms

Based on the symptoms, the disease has been named white blight (Roy and Saikia, 1976), white rot (Rai and Dhawan, 1976), stem blight, stalk break, stem canker, or rape canker, and Sclerotina rot (AICRP-RM, 2009). Generally, the stem is affected more frequently under natural conditions, though all above-ground parts are also attacked. The initial sign of the mycelial infection is the appearance of elongated water-soaked lesions at the base of the stem that expand rapidly (Fig. 1b). The lesions usually become bleached and necrotic and subsequently develop patches of fluffy white mycelium, which is the most obvious sign of mycelial infection (Bolton *et al*., 2006). When the stem is completely gridled and covered by a cottony mycelial growth, the plant wilts and dries (Fig 1a). Mycelial infection in the field is generally very patchy. Carpogenic germination of sclerotia (production of apothecia and liberation of ascospores) under the dense, cool and wet plant canopy mostly occurs at flowering. Ascosporic infection in the field is mostly uniform, not patchy as mycelial infection, and often occurs at the leaf axil where petals lodge; infection also occurs on leaves and produces shot hole symptoms (Fig. 1c). Sometimes, the infection is restricted to a smaller area of the pith, which results in slow stunting of the plant and premature ripening rather than the sudden collapse of the affected plants. Such plants under field conditions can be easiely identified from a distance because of premature ripening. The affected stem tends to shred and numerous grayish-white to black, spherical sclerotia appear either on the surface or in the pith of the affected stem (Fig.1d).

When the crop is at seed maturation stage, the plants tend to lodge, touching the siliquae to the soil level. Such plants, though remaining free from stem or aerial infection throughout, show rotting of the siliquae with profuse fungal growth, along with sclerotial bodies just above the soil level. In an early stage of infection, damping-off, root rot and death of the whole plant may be observed. Some times during ascospore liberation, the infection is also observed on leaves as shot hole symptoms. Lesions on leaves are greyish, irregularly shaped and often associated with adhering petals (Fig.1c). These ascospores are also carried by the petals, which normally fall in the axil of the leaves where they germinate and cause aerial infection resulting into drying of the infected branch. Such branch normally produced the shrivelled siliques (Saharan *et al*., 2005; Saharan and Mehta, 2008). Occasionally, gray mould (*Botrytis cinerea*) is associated with stem rot, covering infected areas with brown to gray fluffy mycelium.

Disease Cycle

The life cycle of *S. sclerotiorum* is relatively simple and various sexual and asexual forms help in the spread of the diseases year after year. The pathogen primarily survives in the soil through sclerotia. Sclerotia get mixed with the soil through infected plant debris after the crop is harvested, or when contaminated seeds are sown in the soil. Seed samples have been found to contain up to 432 sclerotia per kilogram of the seed. Sclerotia are reported to remain viable and virulent upto 7 years assuring pathogen availability when a host crop is planted (Williams and Stelfox, 1980). Viability, however, depends on the type of sclerotia and several other environmental factors. Viability decreases with degree of abnormality; small sclerotia have less reserve food and soil organisms more easily destroy these than large sclerotia. The newly formed sclerotia do not germinate. Sclerotia become distributed throughout the tilled levels of soil, and when conditions become favourable, they germinate to form either a mycelium or apothecia. During harvesting and threshing operations, sclerotia remain on the soil surface with the crop debris, and some are buried in the soil by subsequent tillage operations. Survival of the

pathogen is also possible through infected seeds in the form of mycelial infection of the testa (Neergard, 1958).

There are reports that the fungus can also survive either through mycelium or through ascospores in dead or alive plants (Newton and Sequeria, 1972; Hims, 1979a; Willets and Wong, 1980). Mycelium of the pathogen grows saprophytically and overwinters on diseased stalks. Residues of the crop thus may provide host tissue for development of mycelium in a situation where moisture and temperature become favourable.

Mycelial production by sclerotia is negligible unless an exogenous source of energy is available for host infection; mycelial infection occurs at or below the soil level. Large quantities of ascospores forcibly discharged into the air and carried by air currents for distances ranging from a few centimetres to several kilometres. However, according to Steadman (1983), honeybees efficiently distribute spores to the site of initial infection. If the ascospores discharge before flowering, and unless the senescent tissues are available, the spores can remain viable on plant or on the soil surface for only two weeks. However, once the blossom is colonized, the mycelium can remain viable for more than a month. When in contact with the susceptible host tissue, the ascosporic mycelium produces an appressorium and penetration occurs either by mechanical rupture of the host cuticle, or through the natural openings. Secondary infection occurs when green, healthy tissues come in contact with the infected tissues, but no secondary infective propagulus are produced. Several initial infections tend to be self limiting because the dying plant no longer has the canopy to provide the necessary microclimate for further infection. Continuous germination of sclerotia and discharge of ascospores from apothecia ensures adequate infection potential over a 3-4 week period. The mycelium produces sclerotia externally on affected plant parts and/or internally in stem pith.

The pathogen has been reported to survive in the form of ascospores to some extent if favourable temperature and RH are present under field, as well as, under green house conditions. Since, ascospores survive for a longer period, they are helpful as a source of inoculum in some specific situations (Saharan and Mehta, 2008). Hims (1979a) first reported the role of wild plants acting as a source of primary inoculum in the form of ascospores for rapeseed infection in the United Kingdom. Some wild plants which may carry over the pathogen are hog weed (*Heracelum sphondylium*), cow parsley (*Anthriscus sylvestris*) Holfm, *Chenopodium* spp., and *Asphondilia* spp. (Saharan and Mehta, 2008).

Plant infection

Sclerotinia is characterized as a necrotrophic fungus meaning that it lives on dead and dying plant tissue. Some of the earliest studies on the disease showed that the pathogen kills cells ahead of the advancing mycelium. *Sclerotinia* produces toxic oxalic acid which is partly responsible for plant cell death. Ascospores discharged from the apothecia at the base of the plants in soil constitute an important source of primary infection. Mycelium in soil, or arising from the germinating sclerotia, is less important as primary source of infection, due to its low competitive saprophytic ability (Newton and Sequeira, 1972). On germination, ascospore gives rise to infection hyphae and initial penetration of the host tissue takes place either directly by mechanical pressure through the cuticle or through the natural openings; hyphae may also penetrate already wounded or injured tissues. After entrance of the fungus into the host, the mycelium ramifies inter-or-intracellularly colonizing tissues by enzymatic dissolution of cell wall in advance, and cells die some distance ahead of the invading hyphae. Pectiolytic enzymes are responsible for tissue macearation indirectly damaging the cell membrane, and resulting in subsequent death of the cells (Morrall *et al*., 1972). In addition, the oxalic acid creates an acidic environment in and around the side where many degradative enzymes are most active. Myceliogenic germination of sclerotia results in direct mycelial infection of the host plant (Le Tourneau, 1979). Mycelium can penetrate the cuticle of the host plant using either enzymatic or mechanical means by producing appressoria, unless appressorial penetration occurs through stomata (Lumsden, 1979).

Epidemiology

Knowledge of the host–pathogen interaction and environmental conditions that trigger SR outbreaks provided enough information to assess disease risk and predict the time of disease onset. Continuous moisture for about 10 days is required for apothecial development and even a slight moisture stress prevents apothecial formation. No apothecial initials are produced at either 30 or 5 ºC. Approximately 48-72 h of continuous leaf wetness is required for infection by ascospores. Bom and Boland (2000b) evaluated environmental variables for forecasting SR of canola in Canada and indicated close association between soil moisture and development of the disease especially when petal infestation was high. Their work suggests that petal infestation and soil moisture levels are the best indicators to use in a predictive model. Sclerotinia rot was positively correlated with increase in soil moisture and relative humidity at flowering period (Sharma *et al.*, 2009b). Ascospore germination, mycelial penetration and growth as well as initiation and development of lesion are impotant factors for disease epidemics (Abawi and Grogan, 1975). Air borne ascospores and soil borne hyphae are important primary inoculum for epidemic of Sclerotinia rot. Sharma *et al*., (2010) also observed close association between petal infestation with ascospores and rainfall during full bloom stage in *B. juncea* (Fig.1l). Since ascospores on the leaf surfaces failed to germinate, petals infestation is considered very important for initiation of infection (Jamaux *et al*., 1995; Sharma *et al.,* 2010). Mwiindilila and Hall (1990) reported significant correlation between incidence of white mold and bean canopy coverage during the entire bloom period; amount and distribution of rainfall during this period were very critical for the development of dense plant canopy favorable to the disease. Hall and Mwiindilila (2000) further provided quantitative information on the duration and magnitude of factors, such as flowering, canopy density, moisture, and the presence of apothecia within the field. The risk map for *S. sclerotiorum,* presently in use in the Pairie provinces of Canada, was developed to help farmers in making fungicide spray decisions. To produce these maps, daily precipitation, minimum and maximum air-temperatures, as well as initial soil moisture in the upper 10 cm of soil, and estimated soil moisture as a per cent of field capacity in the upper 12 cm were collected; global Positioning System (GPS) locations, elevation, soil type, and the sclerotia in soil were also taken in to consideration (Bourdôt *et al*., 2001). Nitrogen fertilization is known to increase the incidence of the disease. Increasing the level of N (60-90Kg/ha) in soil leads to increase Sclerotinia rot of mustard from 26.3 to 37.7 per cent (Gupta *et al*., 2004). Based on disease incidence and 10 independent weather variables, a multiple linear regression for per cent Sclerotinia rot incidence = -11.2351 + 0.9529BSSH + 4.93924Eva + 3.83308pH + 0.60885RF (mm) - 0.406458RH 720 + 0.524095RH1420 + 0.17386Soil moisture (%) - 0.30461T max - 0.677744T min - 2.19556WS) has been described (DRMR, 2009-10).

Disease Forecasting and Prediction models

The need for forecasting SR of rapeseed has been recognized in countries such as Denmark, Germany and Canada. The possibility of forecasting SR of rapeseed based on petal infestation (PI) with the pathogen was first suggested by Gugel and Morrall (1986) and later refined by Turkington *et al*. (1991a). In a study of inoculum disease relationships, a strong relationship between disease incidence and percentage plant infection (PPI) at early bloom stage has been established. In general, ascospores produced within the field are often considered as the main source of inoculum. Nordin *et al*. (1992) found no indication that airborne ascospores of *S. sclerotiorum* from neighbouring fields contributed to increase disease levels in the observed rapeseed fields. In contrast, other studies indicated that ascospores of *S. sclerotiorum* from neighbouring fields could be an important source of inoculum for SR of canola in western Canada (Gugel and Morrall, 1986; Morrall and Dueck, 1982; Williams, 1981; Williams and Stelfox, 1979).

Extensive research from Canadaian researchers on effect of high matric potential, soil moisture, herbicides, seeding dates, plant canopy density, and canola cultivars (Cerkauskas *et al.,* 1983, 1985; Teo *et al.,* 1985, 1987, 1988, 1989) on carpogenic

germination and importance of environmental factors and petal infestation in both initiation and development of disease (Gugal and Morrall, 1986; Turkington and Morrall 1993; Turkington *et al.,* 1991b; Bom and Boland, 2000b) have provided enough information for development of a very successful forecasting model. A petal testing method for predicting SR of canola in western Canada, showing a positive relationshiop between disease incidence and the level of petal infestation by *S. sclerotiorum* ascospores at early bloom has an overall success rate of 73% (Turkington *et al.,* 1991b). Although, the forecasting model based on early bloom-petal-infestation was relatively accurate when disease risk and incidence were low, but it was less reliable when disease risk and incidence were moderate to high; rainfall and crop canopy density, which affect ascospores production and relaease, significantly influenced the relationship between petal infestation and disease incidence (Gugel and Verma, 1986; Turkington and Morrall, 1993). Jamaux and Spire (1994) developed a serological test for detection of *S. sclerotiorum* on petals, and the antibody developed was later used by Lefol and Morrall (1996) to study the relationship between petal infestation calculated by immune detection and by an agar plating technique. Turkington and Morrall (1993) prepared a manual containing colour photographs illustrating the difference between *S. sclerotiorum* and common saprophytes, which develop from rapeseed petals. Using these photographs and a key in the manual, about 45 growers from across western Canada successfully used a modified kit to set up petal tests and read the results. The manual used by growers in 1990 has been modified to correct minor problems that occurred in its use, but it is clear that growers can successfully use this kit to conduct their own petal tests. It is recommended that growers consider upto three successive petal tests during flowering to account for fluctuations in PI, but unless PI remains low, only two are usually necessary. Petal testing has several advantages over other methods of forecasting stem rot of rapeseed. It is applied on an individual crop basis. It is superior to search for apothecia in accounting for sources of inoculum that are aggregated or extrinsic to the crop. Finally, in the disease cycle, infested petals are a few steps closer than apothecia to the forecast target, namely diseased plants, thus, there is less potential for environmental intervention between forecast and reality. However, petal testing will never prevent unnecessary fungicide applications when a high disease risk is not translated into high disease incidence because of dry weather after flowering. Factors such as ascospore clumping and viability were found to affect the nature of the relationship. A risk-point table was developed by Thomas (1984) and provided an estimate of disease risk based on past field history, current crop, and weather conditions as well as the presence of apothecia in or around the field. Twengstrom *et al*. (1998a,b) described a forecasting model as a risk-point table for SR of rape (*B. napus* and *B. rapa*) that incorporated the number of years since the last rape crop, disease incidence in the last host crop of *S. sclerotiorum*, crop density, precipitation in the 2 weeks prior to flowering, the weather forecast, and the regional risk for apothecial development. The risk for infection of canola by ascospores of *S. sclerotiorum* was 13 times greater in fields with a history of previously high infestations of the pathogen compared with fields with no previous infestations (Twengstrom *et al*., 1998a). This deficiency is applied to all forecasting systems for SR. However, according to Bom and Boland (2000b), the model that include petal infestation and soil moisture predicts more fields correctly than the model using petal infestation alone, but the accuracy of both are affected by the timing of soil moisture measurements in relation to petal infestation and threshold values in discriminating categories of soil moisture and petal infestation. Bom and Boland (2000a) evaluated three polyclonal-antibody-based immunoassays for detection of *S. sclerotiorum* on canola petals as part of a prediction model for stem rot of canola. The accuracy of serological methods for quantifying petal infestation for practical uses requires further assessment. A simple forecasting system with fairly good reproducibility for evaluation of the risk of attacks of *Sclerotinia* in rape has been developed in Denmark (Buchwaldt, 1986). The risk-point system developed in Sweden was relatively simple. Factors that affect infection by *S. sclerotiorum* are expressed as risk points which, in turn, for each

specific field were summarized and compared to predetermined threshold values. According to this system, the most important factors are pre-crop of rape in a long period, development of rape, level of infection in the previous year, amount of rainfall two weeks before flowering and at the flowering time, weather forecast, and germination of sclerotia. Different risk indicators have been compared to establish the most important and suitable ones for the growers.

Agro meteorological computer models have also been developed based mostly on the weather data and growth stages of rape. The ScleroPro system, available to growers and advisors, is easy-to-handle, fully computerized and based on the weather and field-site-specific data (Koch *et al*., 2007). Makowski *et al*. (2005) have analysed systems that are based on determination of rape flowers infection and on mathematical models. The researchers have found that percentage of infected flowers is more accurate than the calculated logrithm, which is based on the development of rape and weather conditions, but the former method, is costly and time consuming (Makowski *et al*., 2005). The extent of carpogenic germination of sclerotia was a critical factor in the forecast for each region, and the model predicted the need for fungicide application with a high degree of accuracy. This forecasting model is similar to other previously reported risk point tables (Ahlers, 1989; Jakobsen, 1991), but differs in performance with respect to specificity and sensitivity. Based on the following information, the SR forecast have been developed:

a. Accumulated number of germinated sclerotia in depots, including the number of sclerotia with active apothecia (turgid, light brown).

b. The frequency of apothecium occurrence in rape fields selected at random and in fields with previous attack of *S. sclerotiorum.*

c. The growth stage of the oilseed rape as compared with the development of the fungus.

d. Rainfall (and temperature) at localities with depots of sclerotia.

e. Weather prognosis for 5 days at the time of the forecast.

f. High apothecial development only takes place after a rainfall of a minimum of 30 mm within a period of 7-14 days. On the other hand, this precipitation does not necessarily cause a high germination because of evaporation or an unfavourable microclimate.

g. To cause any serious damage, the germination of the sclerotia must have started 7-14 days before initial flowering (Growth stage 4.1). Apothecia formed after this time will come too late to do any damage.

h. Preliminary experience seems to indicate that there is a risk of attacks when the accumulated number of germinating sclerotia in the depots is over 30 per cent at the time of the forecast. Besides this, the majority must have active apothecia.

i. After a rainfall of a minimum of 30 mm, naturally occurring apothecia can be found-within a period of 7-14 days, especially in fields with previous attacks of *S. sclerotiorum.*

j. During the week after a rainfall of a minimum of 30 mm apothecia are formed and become visible.

k. One week without rainfall prevents or delays the formation of new apothecia and dries out those already produced.

Ghasolia and Shivpuri (2005) observed that Sclerotia on upper soil surface produced more apothecia. The prediction model developed for white stem rot as under:

$$Y= -19.14 – 4.59\ X_1 + 2.53\ X_2 + 1.02\ X_3 – 0.19\ X_4 - 0.00\ X_5 +0.63\ X_6,$$

where X_1=Temp. Max.; X_2= Temp. Min.; X_3=RH (Mor.); X_4=RH (Eve.); X_5=Sunshine; X_6=RF. Aghajani *et al*. (2010) from Iran reported that Gompertz model with a mean R^2 of 94.69 was selected as most appropriate model for determining

Sclerotinia stem rot progress in the field. The forecasting models for the prediction of Sclerotinia stem rot has also been reviewed by Mehta, (2014).

Sources of Resistance

One of the most effective, economical and environment friendly approach to control plant diseases is the development of genetically resistant cultivars. Since *Sclerotinia sclerotiorum* exhibits very little host specificity (Purdy, 1979), it is important to understand the diversity of this pathogen for the development of effective screening strategies to identify and deploy host resistance. Host resistance offers the only economic and sustainable method for effectively managing this disease (Zhao *et al*., 2004; Li *et al*., 2006). Because of its very wide host range, breeding for resistance against *S. sclerotiorum* is very challenging. *Brassica napus* and *B. juncea* cv. Rugosa have been reported to possess resistance against white stem rot both in the field as well as in the green house conditions (Singh *et al.,* 1994). Partial resistance has also been identified in some *B. napus* and, to a lesser extent, in *B. juncea* genotypes from China (Li *et al*., 1999, 2006, 2008; Zhao *et al*., 2004), Australia (Li *et al*., 2006, 2008), and India (Singh *et al*., 2008). Although, a significant number of partially resistant genotypes have been identified, breeding to increase the levels of resistance against Sclerotinia disease in *B. napus* and/or *B. juncea* has been ineffective. This is mainly because resistance to *S. sclerotiorum* in existing cultivars of *Brassica* and in other cultivated germplasm appears to be of a complex nature, *i.e.,* it can either be monogenic and/or polygenic depending on the plant species and materials under investigation (Abawi *et al*., 1978; Baswana *et al*., 1991; Zhao and Meng, 2003b; Zhao *et al*., 2006). Complete resistance has not been identified in canola. Partial field resistance has been identified in the Chinese variety Zhongyou 821 (Li *et al.,* 1999; Buchwaldt *et al*., 2003). A cultivar Zhongshuang No. 9, claimed to be better than Zhongyou 821, was reported in 2003 (Wang *et al*., 2003). The next most resistant *B. napus* genotypes that have been previously reported included 06-6-3792 (China), ZY004 (China), and RT 108 (Australia) with mean stem lesion lengths of <3.0 cm (Li *et al*., 2008). The genotypes of *B. juncea* namely, EC 597328 (Montara), EC 597329 (Berry) and EC 597331 (Ringot I) of Chinese origin were also found tolerant (Sharma *et al.*, 2009a). In addition, the levels of resistance previously reported in genotypes *B. juncea* JM06018 and JM 06006 with mean stem lesion lengths of 4.8 cm (Li *et al*., 2008) was far lower as compared to the *B. napus* genotypes. However, the situation has begun to improve due to introduction of new sources of *Sclerotinia* resistance from Chinese native cultivars. For example, the double-low restorer line of the elite cv. Zhongshuang 9 was released as *Sclerotinia* resistant (Wang *et al*., 2004). Glucosinolates present in Brassicaceae plants have fungicidal and bactericidal properties which can also give resistance against fungal pathogens (Zukalová and Vasák, 2002). Since significant differences in lesion lengths were detected, it is possible that isolates of *S. sclerotiorum* have a different pathogenic ability, as reported by Pratt and Rowe (1991) and Liu (1996). Li *et al*., (2001) found that Zhongyou 821 produced more indolic glucosinolates in the leaves after inoculation. Moderate *Sclerotinia* resistance has been reported in several related species, including *B. nigra*, *B. juncea* and *Sinapis alba* (Morrall and Dueck, 1982; Kolte, 1985). Novel approaches to the introduction of increased *Sclerotinia* resistance into oilseed rape include the development of transgenic plants expressing oxalate oxidase activity (Thompson *et al*., 1995) capable of degrading oxalic acid, a pathotoxin of the fungus. Induced mutagenesis has also proved to be a successful technique for introducing disease resistance genes into crop varieties (Konzak, 1956; Maluszynski *et al*., 1995); it is widely accepted that micro-mutations in minor genes, causing small quantitative changes in phenotypic characters, occur at much high frequencies (IAEA, 1977). Induced mutants exhibiting increased partial (quantitative) disease resistance have been isolated from small populations (Varghese, 1985; Worland and Law, 1991; Kinane and Jones, 1996). Against a non-specialised necrotrophic pathogen such as *S. sclerotiorum*, any resistance would probably be quantitative in nature. Successful isolation of *Sclerotinia*-resistant mutants from small M2 populations of oilseed rape could introduce potentially valuable genes into the *B. napus* gene

pool. In plant-pathogen interactions, development of new pathogenic races and break down of resistance are limiting factors in resistance deployment against plant diseases. The pathogen's life history characteristics and evolutionary potential are major factors leading to the pathogen overcoming host resistance (García-Arenal and McDonald, 2003; Coletta-Filho and Machado, 2002; McDonald and Linde, 2002). Therefore, knowledge regarding the population genetic structure and evolutionary potential of the pathogen will provide insight into the most suitable breeding strategy for durable resistance (McDonald and Linde, 2002). Nine genotypes viz., Cutton, ZYR-6, PSM-169, PDM-169, Westar, PYM-7, Parkland, Tobin and Candle showed resistance to stem rot in India (Shivpuri *et al*., 1997). Four genotypes viz., PCR-10, RW-8410, RW- 9401 and RGH-8006 had better resistance against *S. sclerotiorum* as compared to susceptible check (Pathak *et al.,* 2002). Wang *et al.* (2009), isolated genes from *B. napus* known as *B. napus* mitogen-activated protein kinase, (BnMPK4), and found that *BnMPK4* along with *PDF* 1.2 are inducible in resistant line Zhongshuang 9, but both were consistently suppressed in susceptible line 84039 after inoculation with *S. sclerotiorum*. Transgenic oilseed rape over expressing *BnMPK4* markedly enhanced resistance to *S. sclerotiorum*. Further, it was observed that transgenic plants inhibited growth of *S. sclerotiorum* and constitutively activated *PDF1.2*, but decreased H_2O_2 production and constitutively suppressed *PR-1* expression. Treatment of roots of the transgenic plants with H_2O_2 solution resulted in enhanced susceptibility to the two pathogens. It was suggested that *MPK4* positively regulates jasmonic acid-mediated defense response, which might play an important role in resistance to *S. sclerotiorum* in oilseed rape. Sharma *et al.* (2009a) evaluated indigenous and exotic genotypes of *B. napus* and *B. juncea* in pathogen-infested field and observed that genotype EC-597328 of *B. juncea* is tolerant to this disease (Fig.1h). Sharma *et al*. (2012) evaluated seventy germplasm from India, Australia and China by artificial stem inoculation (Fig. 1 e) and observed that none of the germplasm was resistant (Fig. 1 f,g) while partial tolerance was observed in lines BLN 3630 (EC597274) of *B. napus* and Berry (EC597329) of *B. juncea*.

For evaluation of resistance to rape SR, Sang *et al.* (2013) transferred the *MSI-99m* gene (modified *MSI-99*) into Chinese rape variety Zhongyou 821 using *Agrobacterium*-mediated method. Nine transformed lines carrying a *MSI-99m* expression vector were detected by polymerase chain reaction (PCR), among which seven lines expressed *MSI-99m* gene according to qRT–PCR analysis. Disease resistance analysis consistently showed that the high-level expression of *MSI-99m* increased resistance to *S. sclerotiorum* in transgenic rape lines. It was demonstrated that *MSI-99m* gene might be applied as a resistant gene resource in rape for the improvement of rape varieties.

Callogenic response to culture filtrate (CF) of the pathogen was evaluated and observed that MS medium supplemented with 1-naphthaleneacetic acid (NAA) and 1-benzylaminopurine (BAP) were suitable for callus induction; MS medium supplemented with CF of *S. sclerotiorum* @ 15 % was found inhibitory to callus production in *B. juncea* cv. RH-30. The activity of CF in callus production was lower in *B. carinata* cv. HC-9002 and *B. napus* cv. GSH-1 as compared to *B. juncea* (Watpade *et al.,* 2012). The biochemical changes occurred during the development of callus in the presence of CF also suggests that the total soluble protein in *B. napus* and *B. carinata*, and activity of polyphenol oxidase and peroxidase increased in resistant calli; catalase activity decreased in resistant/tolerant calli as compared to susceptible calli (Watpade and Mehta, 2012). Total soluble sugars and reducing sugars were higher after selection with *S. sclerotiorum* culture filtrate in calli of *B. napus* cv. GSH-1 and *B. carinata* cv. HC-9002, but other species showed lesser sugar contents. Generally, level of phenol, flavanol and total soluble protein were higher after selection in all tolerant calli (Watapde and Mehta, 2013). Culture filtrates of 25 geographical *S. sclerotiorum* isolates reduced seed germination and length of radical and plumule (Fig. 1q)in *B. juncea* cv. Rohini (Sharma *et al.,* 2014).

Lack of effective resistance to Sclerotinia disease in cultivated species has stimulated the interest of researchers towards exploitation of wild crucifer species to diversify the existing gene pool. Higher levels of resistance against Sclerotinia have already been reported in the secondary gene pool of bean (Abawi *et al.,* 1978; Gilmore *et al.,* 2002; Schwartz *et al.,* 2006). Introgression of genomic segments responsible for resistance against *Sclerotinia* from wild to cultivated species of sunflower has also been attempted (Ronicke *et al.,* 2004). Although, Brassicaceae family contains a wide array of different species, to date, only two wild crucifers, *Capsella bursa-pastoris* (Chen *et al.,* 2007) and *Erucastrum gallicum* (Lefol *et al.,* 1997a; Seguin-Swartz and Lefol, 1999), have been reported to show high level of resistance against Sclerotinia. Although introgressive hybrids were successfully obtained between *B. rapa and B. napus* species and *Capsella bursa-pastoris* (Chen *et al.,* 2007), it remains to be confirmed if the introgression of resistance against *S. sclerotiorum* from *E. gallicum* into cultivated species has in fact been accomplished (Lefol *et al.,* 1997a,b; Seguin-Swartz and Lefol, 1999). Garg *et al.,* (2010) developed Introgression lines following hybridization of three wild crucifers (viz. *Erucastrum cardaminoides, Diplotaxis tenuisiliqua* and *E. abyssinicum*) with *B. napus* or *B. juncea*; introgression lines had much higher levels of resistance (Fig 1i).

Oxalate oxidase (OXO): A common strategy for combating *Sclerotinia* is to degrade OA, the plant toxin and *Sclerotinia* pathogenicity factor secreted from the pathogen. There are three classes of known enzymes that can catabolize OA, namely oxalate oxidase (OXO) (EC 1.2.3.4) (Lane *et al*., 1991), oxalate decarboxylase (EC 4.1.1.2) (Mehta and Datta, 1991), and oxalyl-CoA decarboxylase (EC 4.1.1.8) (Lung *et al*., 1994). The bacterial oxalyl-CoA decarboxylase gene could be used for oxalate degradation and engineering *Sclerotinia* resistance in plants (Dickman and Mitra, 1992). However, both fungal and bacterial oxalate decarboxylases convert oxalate into CO_2 and formic acid, which might have a toxic effect on plant cells. Therefore, scientists have been focusing on OXO, which releases CO_2 and H_2O_2 from O_2 and OA. This enzyme was first isolated and characterized from barley and wheat (Lane *et al*., 1993; Kotsira and Clonis, 1997). The transgenics greatly reduced disease progression and lesion length following cotyledon and stem inoculation with *S. sclerotiorum,* indicating that OXO conferred resistance to the stem rot (Donaldson *et al*., 2001). Grison *et al*., (1996) introduced a hybrid endochitinase gene under a constitutive promoter by *Agrobacterium* mediated transformation into a winter-type oilseed rape inbred line.

Quantitative Trait Locus (QTL): Genetic studies have shown that the resistance to *Sclerotinia* can be either monogenic or polygenic, depending on the plant species and materials under investigation (Abawi *et al*., 1978; Baswana *et al*., 1991). Little is known about how many genes in rapeseed are involved in *Sclerotinia* resistance. Quantitative Trait Locus mapping has proved to be a powerful approach in understanding the genetic basis of quantitative traits (Tanksley, 1993; Young, 1996). Quantitative Trait loci associated with some important traits in rapeseed have been mapped, including resistance to black-leg (Ferreira *et al*., 1994; Dion *et al*., 1995; Chevre *et al*., 1997), to club-root (Matsumoto *et al*., 1998), to white rust (Ferreira *et al*., 1995), to glucosinolate content (Toroser *et al*., 1995; Uzunova *et al*., 1995), and to other important agronomic traits (Teutonico and Osborn, 1994; Song *et al*., 1995; Butruille *et al*., 1999). Results from mapping and genetic analysis of *Sclerotinia* resistance show that QTLs in rapeseed would be very useful for marker-assistant selection and durable resistance cultivar breeding. It has been reported that oxalate played an important role in the pathogenesis of *Sclerotinia* (Maxwell and Lumsden, 1970; Godoy *et al*., 1990; Cessna *et al*., 2000). *Arabidopsis* has also been used to investigate *S. sclerotiorum* pathogenesis, although *S. sclerotiorum* mutants deficient in oxalic acid (OA) production have been found to be non-pathogenic to *Arabidopsis* (Godoy *et al*., 1990). Dickman and Mitra (1992) initiated investigation of *Sclerotinia-Arabidopsis* pathosystem. Zhao and Meng (2003a) identified three quantitative trait loci (QTLs) on the linkage groups, N3, N12 and N17 of the A- and C-genomes

of *B. napus* involved in the control of resistance to SR at the seedling stage, although, three QTLs on N7, N10 and N15 control resistance at the adult plant stage. On the other hand, Zhao *et al*. (2006) identified eight regions on N2, N3, N5, N12, N14, N16 and N19 affecting resistance to this disease. Thus, it is apparent that at least 11 of the 19 *B. napus* chromosomes carry QTLs for resistance, and the phenotypic variation explained by these QTLs varied from 5.9% to 39.8%. One of these loci was linked to a 3-indolylmethyl glucosinolate locus (Zhao and Meng, 2003a). Molecular markers linked to these QTLs would allow screening for resistance at the genotypic level, thereby increasing the effectiveness of selection for this trait. Variation for resistance to *Sclerotinia* was continuous among the DH lines, suggesting polygenic control of this trait in this population. Zhao and Meng (2003b) and Zhao *et al*. (2006) identified several QTLs for this trait in *B. napus*. Similarly, polygenic control of resistance to SR has also been reported in the C-genome of *B. oleracea* (Baswana *et al*., 1991). To date, complete resistance to this pathogen has not been identified, although partial resistance was recently reported in *B. napus* cv. Zhongyou 821 (Buchwaldt *et al*., 2003; Li *et al*., 1999).

Defense Signaling Pathways: When plants are challenged by pathogens, a complex and integrated set of defense responses, both constitutive and induced, are triggered (Dong, 1998; Kunkel and Brooks, 2002). These include wax layers, preformed antimicrobial enzymes, secondary metabolites, and toxic compounds under the "constitutive responses", and systemic acquired resistance (SAR), as well as, induced systemic resistance, under the "induced responses". A series of events are initiated in plants during interactions between plants and pathogens including the production of signaling compounds such as jasmonic acid (JA), ethylene (ET), salicylic acid (SA), or reactive oxygen species (Kunkel and Brooks, 2002). Signaling pathways, mediated by JA/ET and SA, are important components of plant defense systems (Dong, 1998; Feys and Parker, 2000; Martinez *et al*., 2001; McDowell and Dangl, 2000), and depending on the system between host/pathogen, they are regulated and can act independently, synergistically, or antagonistically (Kachroo and Kachroo, 2007). In *Arabidopsis*, as revealed by microarray analysis, five out of 41 genes responding to JA are those involved in its biosynthesis, indicating the existence of a positive feedback regulatory system for JA biosynthesis (Sasaki *et al*., 2001). Mehta *et al.* (2010a) has demonstrated that application of SAR chemicals (Isonicotinic acid or Salicylic acid @ 100 ppm) as seed treatment result in significant reduction in seedling mortality, lesion length, disease incidence, and disease intensity.

Physiological Specialization

Variability in pathogen with respect to cultural, morphological, physiological and pathogenic characters play an important role in distinguishing various races of the pathogen. Variation in pathogenicity has also been associated with the production of pectolytic enzymes, hemicellulase and oxalic acid (Noyes and Hancock, 1981). However, a variation in these factors does not appear to be the primary determinant of pathogenicity. Price and Calhoun (1975) reported wide variations in different *S. sclerotiorum* isolates. The isolates of *S. sclerotiorum* collected from rapeseed- mustard have also shown variation in virulence (Rai and Dhawan, 1976). Dhawan *et al*. (1981) reported variation in virulence amongst four Chinese mustard isolates. Willets and Wong (1980) reported that pathogenicity and morphologically differed *S. sclerotiorum* isolates produced more protopectinase than a normal strain, but the normal isolates secreted more virulent toxin. Little information is available on additional mechanisms that affect virulence among isolates of *S. sclerotiorum.*

Clonal variability within and among field populations of *S. sclerotiorum* isolated from canola petals in western Canada has been determined by analysis of two independent criteria, mycelial compatibility and DNA fingerprinting (Kohli *et al.,* 1992); strains belong to the same clone, with compatible mycelia, have identical DNA fingerprints. Thirty-nine clones were identified among 66 strains from several locations in Alberta, Saskatchewan and Manitoba. The most widely distributed clone accounting for 18 per cent of the isolates has been recorded in all three provinces; seven other clones were also identified

in these provinces. In 33 out of 35 mycelial compatibility groups (MCGs), each MCG has a unique DNA fingerprint; the remaining two MCGs include strains with one of two fingerprints and interpreted as two clones. A comparison of strains from western provinces with those of two fields in Ontario showed that the one clone identified in both Ontario fields was also present in Manitoba and Saskatchewan. This study demonstrates that field populations of *S. sclerotiorum* in canola in Canada contain more than one clone. Analysis of monosporous siblings from homothallic sexual reproduction in each of the two clones showed no mitotic segregation for determinants of either mycelial compatibility or DNA fingerprints. Therefore, intake clonal genotypes can potentially be dispersed as ascospores.

According to Errampolli and Kohn (1996), Electrophoretic Karyotypes (Eks) of 83 isolates were variable within agricultural and natural populations of *S. sclerotiorum* as well as among *S. minor* and *S. trifoliorum*. Molecular techniques have been used to characterize different field isolates of S. *sclerotiorum*. Chromosomal DNA by pulsed field gel electrophoresis, (PFGE) revealed that *S. sclerotiorum* contains at least 16 chromosomes ranging from 1.5 to 4.0 Mb; size of the haploid genome estimated to be 43.5 Mb.

Comparison of 594 unique DNA fingerprints of *S. sclerotiorum* from Canadian canola, revealed that no fingerprint was shared among Canadian, North Carolina and Louisiana populations. DNA fingerprints from the North Carolina sample was found distinct from those of the Canadian and Louisiana samples with significantly more hybridizing fragments in the 77 to 18-kilobase range. Forty-one mycelial compatibility groups (MCGs) and 50 unique DNA fingerprints from the North Carolina sample, and three MCGs and three fingerprints from the Louisiana sample were identical. From the North Carolina sample, 32 MCGs were associated with a unique DNA fingerprints and 11 clones were identified (i.e. cases in which two or more isolates belong to the same MCG and share the same DNA fingerprint). Six clones samples from two or more fields represented approximately 29 per cent of the total sample (74 to 84 isolates), with six clones recovered from fields 75 kb apart. There were 10 cases in which one MCG associated with more than one DNA fingerprint, and two cases in which one DNA fingerprint associated with more than one MCG. The small sample from Louisiana was strictly clonal. The North Carolina sample has a clonal component, but deviates from one to one association of MCG with DNA fingerprint to an extent consistent with more recombination as transposition than the other two populations sampled.

Disease Management

Management of *S. sclerotiorum* is a major challenge faced by plant pathologists. Management is difficult, inconsistent and uneconomical due to the presence of wide host range and long-term survival of the resting sclerotia. Since no single method can effectively control *S. sclerotiorum*, the best approach to control the pathogen is by the integration of various eco-friendly measures.

Cultural control

Cultivation of crops susceptible to *S. sclerotiorum* will lead to the build up of sclerotial inoculum in the field. The degree of field infestation by *S. sclerotiorum* ranges from 0 to 85% (Tu, 1986). Hence, the knowledge on the level of sclerotial population in the field will be very useful in designing any cultural control measures. Sclerotia in the top 2-3 cm soil often germinate carpogenically. The sclerotial populations deteriorate faster by the mycoparasites that dwell in the top soil. But if the soil is ploughed, the sclerotia are buried deeper in the soil and survive for several years. A significant negative relationship exists between sclerotial viability and depth of burial, and between sclerotial viability and populations of colonizing bacteria under zero tillage condition (Duncan, 2003). The zero tillage, thus, not only reduces the inoculum load of the sclerotia, but also the infection of host plants by the pathogen. Cultivation of non-host crops, although, reduces inoculum load (Adams and Ayers, 1979), three to four years' of crop rotation was not effective enough to reduce the incidence of stem rot of canola in Canada (Morrall and Dueck, 1982); a minimum of 5 year rotation of two non host crops is essential to decrease the infection level by the

pathogen (Gulya *et al*., 1997). Cultural practices including wider row spacing or lower plant populations that reduce the microclimate favorable for disease development are used in some crops. A significant positive relationship between SR incidence and seeding rate was found (Jurke and Fernando, 2006). Sanitation practices including vegetable production, and clean seed programs to keep sclerotia out of seed lots are also useful practices in some crop production systems. Although, cultural practices can reduce the impact of *Sclerotinia* infection, they are often not sufficient to control the pathogen. Since, increased level of P, K, Ca and sulphur reduced disease severity significantly in mustard, a balanced fertilizer application of macro and micronutrient should be adopted (Singh and Tripathi, 1993). The persistent nature of sclerotia and the wide host range of this pathogen generally render cultural practices ineffective (Williams and Stelfox, 1980; Boland and Hall, 1994).

The type of soil and amount of frequency of irrigation significantly affect sclerotial germination and apothecial development. Maximum number of apothecia were recorded in the sandy loam than in the sandy soil (Mehta *et al.,* 2009). Further, flooding of the field (once a week) prior to sowing resulted in least disease incidence and minimum lesion length; optimum irrigation applied once in 3 or 7 days intervals produced lower disease intensity as compared to the control (Mehta *et al.,* 2009).

Pereira *et al*. (1996) evaluated integrated effect of vermi-compost, soil solarization, herbicide (EPTC), fungicide (procyniodene), *Trichoderma harzianum* and *Bacillus subtilis* for the control of *S. sclerotiorum*. Soil solarization through coverage of transparent polythene (0.1 mm) for 45 days is a good strategy for management of soil-borne pathogens. Solarization of soil with transparent thick plastic sheets (60-micron) for two months significantly reduced (76%) the incidence of lettuce drop (Gepp *et al*., 2001). Herbicide EPTC treatment significantly increased the level of control irrespective of the burial depth of the sclerotia in soil. Similarly, *T. harzianum* in the presence of vermi-compost combined with EPTC treatment also reported to be very promising control strategy for *S. sclerotiorum*.

Augmentation of soil with organic or inorganic compounds helps in increasing microbial population capable of suppressing germination of sclerotia. Soil application of compost with higher microbial populations inhibited carpogenic germination of *S. sclerotiorum* and reduced infection (Couper *et al*., 2001). Amending soil with formulated products, S-H and CF-5 mixtures promoted growth of *Trichoderma* sp., soil-borne bacteria and actinomycetes which controlled carpogenic germination of sclerotia (Huang *et al*., 1997). Timely sowing, field sanitation, burning of stubble, seed treatment and deep ploughing followed by crop rotation with irrigated rice cultivars was found effective in managing Sclerotinia rot in Indian mustard (Singh, 2001).

Leaf extract of neem (*Azadirachta indica*), datura (*Datura stramonium*), mehndi (*Lowsonia inermis*) and clerodendron (*Clerodendrum inerme*) inhibited *in vitro* mycelial growth and sclerotial formation; Bougainvillea (*Bougainvillea spectabilis*), neem (*A.indica*) and garlic (*Allium sativum*) leaf extracts were most effective (Mehta *et al.,* 2011). Soil amendment with Bougainvillea leaves was also effective in reducing seedling mortality, number of apothecia production, lesion length and disease intensity in mustard. Soil amendment with mustard cake, sesame cake, jamun (*Syzygium cuminii*) seed powder and poultry manure also found effective in reducing seedling mortality, number of apothecia p roduction, lesion length and disease intensity in mustard (Mehta *et al.,* 2010b).

Biocontrol

In the light of present day concern about the environment, human health and development of resistance to funcicides, biological control is an attractive alternative for plant disease management. Biocontrol is the reduction of the amount of inoculum or disease-producing activity of a pathogen accomplished by or through one or more organisms other than man (Cook and Baker, 1983). Concerns associated with the use of chemical pesticides have made the biological control of *Sclerotinia* diseases an interesting area of research amongst plant pathologists (Bardin and Huang, 2001).

Sclerotinia sclerotiorum over-winters in the soil or on the plant debris as sclerotia. Soil microbial population plays an important role in reducing the inoculum build up of the soil–borne pathogens. Both fungi and bacteria near the soil surface play an important role in degrading the sclerotial bodies. Diurnal fluctuation of soil temperature, moisture and relative humidity cause cracks on the sclerotial rinds, resulting not only in leakage of the cell constituents, but also providing avenues of infection by the antagonistic microbes dwelling in the soil.

Fungal antagonists: In general, the use of biocontrol agent is restricted to controlled environment because they need stable environmental conditions for growth and successful establishment in the infection court (Whipps, 1994). The mycoparasitic fungi parasitizing sclerotia include *Coniothyrium minitans*, *Trichoderma* spp., *Gliocladium* spp. *Sporidesmium sclerotivorum*, *Talaromyces flavus, Epicoccum purpurescens, Streptomyces sp., Fusarium*, *Hormodendrum*, *Mucor*, *Penicillium*, *Aspergillus*, *Stachybotrys*, and *Verticillium* (Adams and Ayers, 1979; Makkonen and Pohjakallio, 1960; Singh and Kaur, 2001; Martinson and del Rio, 2001; Baharlouei *et al.,* 2011).

Soil application of *C. minitans* to different host crops reduced carpogenic germination and viability of sclerotia under wide range of temperature, relative humidity and soil moisture (McLaren *et al*., 1996; Sandys-Winsch *et al.,* 1993; Budge *et al*., 1995; Hedke and Tiedemann, 1998). Although, soil incorporation of *C. minitans* in microplots of oilseed rape reduced soil inoculum, it neither led to disease control nor yield improvement (McQuilken *et al*., 1995). Wettable Granule of *C. minitans* containing $1x10^9$ viable conidia per gram colonized and decayed sclerotia within 3 months of its application, and was effective against *S. sclerotiorum* infecting vegetables, ornamentals, oilseed rape and beans (Luth, 2001). Hannusch and Bolland, (1996) reported that change in air temperature by 4 ^oC, or relative humidity by 5%, adversely affected the *S. sclerotiorum*-suppressing ability of the fungi isolated from anthroplanes of bean and rapeseed. Singh and Kaur (2001) reported mycoparisitism of *T. harzianum* (Th38), *T. viride*, and *E. purpurescens* against *S. sclerotiorum*; *T. harzianum* controls *S. sclerotiorum* by hyphal mycoparasitism rather than by sclerotial parasitism. The isolates of *T. harzianum* 3, *T. harzianum* 4, *T. virens* and *T. viride* were most potent in reducing the linear growth of mycelium and apothecial production of *S. sclerotiorum in-vitro; T. harzianum* was most effective in reducing the lesion length and disease intensity when applied simultaneously, or seven days prior to the pathogen under the screen house conditions; antagonist applied at the rate of 15 g/Kg soil as wheat bran inoculum was superior in reducing SR of rapeseed-mustard (Mehta *et al.,* 2012). Srinivasan *et al.* (2001) reported that even the cultural filtrate of *T. viride* and *T. harzianum* significantly reduced the growth of *S. sclerotiorum in vitro* conditions. Pathak *et al*. (2001) also reported reduced SR incidence with soil application of *T. viride*, *T. harzianum* and *G. virens* combined with bavistin seed treatment. Soil-borne strains of *Gliocladium roseum*, *T. harzianum* and *Aspergillus* sp. also showed inhibitory effect against the pathogen (Rodriguez and Godeas, 2001). Application of Kalisena SL (*Aspergillus niger)* formulation in *S. sclerotiorum*-infested plots saved the cauliflower seedlings, and soil application of *A. niger* AN27 parasitized and killed the existing sclerotial population (Sen, 2000). *Streptomyces* sp isolate 422 significantly reduced SR incidence (Baharlouei *et al.,* 2011) by increasing the level of hydrolytic enzymes including chitinase and â-1, 3 glucanase in canola plants (Fernando *et al.,* 2007). Soil incorporation of sclerotial parasite *S. sclerotiorum* was also found effective up to 5 years in controlling Sclerotinia rot (Martinson and del Rio, 2001).

Considering the epidemiology of the disease, any biological control which only reduces primary source of soil inoculum by reducing sclerotial germination, apothecial development and discharge of ascospores will be of very limited importance for the control of SR in oilseed rape and mustard. This is simply because there is still enough sclerotial inoculum left in the soil to infect rapeseed-mustard plants at flowering stage. The potential mycoparasite, applied aerially, must also be effective in inhibiting germination of ascospores on petals. For successful control of

SR in oilseed rape and mustard the potential mycoparasite must therefore, be apllied both aerially and in the soil. In this regard, results of several studies by Meena *et al.,* (2009, 2011, 2013, 2014) showing significant effectiveness of *T. harzianumi* applied as soil inoculants, seed treatment and foliar spray, singly or in combination, in controlling SR in mustard is a step in the right direction. Soil inoculation of *T. harzianum* isolate GR and soil application of FYM- infected with *T. harzianum* isolate SI-02 reduced SR incidence by 69% and 60.8%, respectively (Meena *et al.,* 2009). Similarly, seed treatment combined with foliar spray of *T. harzianum*, and seed treatment with *T. harzianum* and foliar spray with garlic (*Allium sativum)* bulb extract, not only significantly reduced SR incidence, but also gave higher cost:benefit ratio (Meena *et al.,* 2011, 2013, 2014). The disease suppression was due to the effective saprophytic colonization of petals.

Bacterial antagonists: 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 including *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 white mold fungus still remains to be explored fully. Strains of *Bacillus* spp., frequently isolated from the sclerotia of *S. sclerotiorum* from North Dakota in the USA, not only reduced germination of infected sclerotia, but also adversely affected integrity and colour of medulla (Wu, 1988); fifty three per cent of sclerotial bodies of *S. sclerotiorum* recovered from the soils of North Dakota were infected by *Bacillus* species. *Bacillus* strains isolated from canola and wheat plants also showed antifungal activity to *S. sclerotiorum in vitro* (Zhang and Fernando, 2003). Apart from pre-colonization, several *Bacillus* spp. also produced the antibiotic Zwittermicin-A (Zhang and Fernando, 2004), which increased degradation and reduced germination of sclerotia of *S. sclerotiorum* (Nelson *et al*., 2001). *Erwinia herbicola* and *B. polymyxa* inhibited the growth of *S. sclerotiorum in vitro* (Godoy *et al*., 1990). Antagonistic *Pseudomonas* spp. (DF41) and *P. chlororaphis* (PA23) inhibited the germination of ascospores of *S. sclerotiorum* (Savchuk and Fernando, 2004). Application of DF41 and PA23 on to petals increased bacterial population after 24 h, but population 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). Results from these studies indicate that PA23 and DF41 are effective biocontrol agents against *S. sclerotiorum* (Savchuk and Fernando, 2004). The bacterial strains/ species viz., *B. subtillis*, *P. fluorescens* 132, *P. maltophila* and *P. fluorescens* M have been reported to be most effective by inhibiting mycelial growth and sclerotial formation against *S. sclerotiorum in vitro* conditions. Per cent mycelial growth inhibition of the pathogen was negatively correlated with the number of sclerotia formation. Regression equation developed revealed $Y = -1.9882x + 65.418$, with $R^2=0.819$ (Mehta and Hieu, 2014). The *P. chlororaphis* (PA23), *B. amyloliquefaciens* (BS6), and *Pantoea agglomerans* exert multiple mode of action and lead to the suppression of carpogenic germination and mycelial growth through the production of volatile and non-volatile antimicrobial antibiotics. Moreover, bacteria PA23 and BS6 trigger/ induce resistance via the production of defense related gene products. *P. agglomerans* degrades oxalic acid through the production of oxalate oxidase. The above-mentioned promising strains would pave the way for the management of *S. sclerotiorum* in both agricultural and horticultural crops (Fernando *et al.,* 2004). *P. chlororaphis* strain PA23 controlled ascospore germination, and stem rot of canola in both greenhouse and field studies (Fernando *et al*., 2007). Antibiotics extracted from PA23 caused inhibition of sclerotial and spore germination, hyphal lysis, vacuolation, and protoplast leakage in a number of plant pathogens, including *S. sclerotiorum* (Zhang *et al*., 2004). Presence of these antibiotics was confirmed by sequencing the PCR products and through BLAST search in the gene bank (Zhang and Fernando, 2004). *P. agglomerans* isolated from leaves and flowers of canola produces oxalate oxidase and degrades oxalic acid produced by *S. sclerotiorum*, the

pathogencity factor required for the successful establishment of the host-parasite relationship. Pre-colonization of infection court including blossoms and leaves by *P. agglomerans* would probably be very effective in preventing the infection process.

Fungicides

Sclerotinia continues to be a very difficult pathogen to control. Therefore, fungicides have been extensively used for the control of *S. sclerotiorum* in canola, as well as in other crops like soybean, dry bean and some vegetables (Bailey *et al*., 2000; Budge and Whipps, 2001). Fungicides are effective in reducing severe yield losses (Morrall and Dueck, 1982, Morrall *et al.,* 1983, 1984a,b; 1985) there was an increase in the percentage of UK oilseed rape crops sprayed to control *Sclerotinia* from 9% in 1991 to 44% in 1993 (Turner and Hardwick, 1995).

Seed treatment: *S. sclerotiorum* survives in infected seeds as dormant mycelia in testae and cotyledons for 3 years or longer (Tu, 1988). When infected seeds were sown, 88-100% failed to germinate. Seedlings produced from infected seeds subsequently died from white mold at an early stage, seeds that failed to germinate were rotted by *S. sclerotiorum*, and three to six sclerotia were formed in place of each seed (Tu, 1988). These sclerotia could become a source of inoculum. Captan and thiophanate-methyl used in seed treatment were 100% effective in eradicating the fungus from the infected seeds. Seed treatment with carbendazim also found effective in minimizing the Sclerotinia rot incidence (Sharma *et al.,* 2011).

Foliar application: In canola, fungicides are often applied at the full bloom stage to prevent infection of the senescing petals, which can fall on the leaf axils leading to infection of the stem (Kutcher and Wolf, 2006). The application of fungicides can be expensive and also has negative environmental impacts. Moreover, the development of resistance to fungicides in the pathogen population always remains a threat (Gossen *et al*., 2001). A benzimidazole fungicide, carbendazim (MBC), was widely used to control this disease routinely in China, but widespread control failures in 2001 and 2002 attributed to development of MBC resistance in *S. sclerotiorum*. Therefore, the use of MBC and related fungicides for controlling SR in China was compromised after more than 30 years application of MBC in Jiangsu Province (Pan 1998; Shi *et al*., 2000; Zhang *et al*., 2003). Fludioxonil at 100, 200 and 300 µg/ml provided susstantial disease control (Kuang *et al.,* 2011). Foliar spray of carbendazim at full bloom stage provided significant disease reduction and highest seed yield among the different treatments over control (Sharma *et al.,* 2011). In Europe, spray application of prothioconazole 250 EC (Proline) at the rate of 175g a.i./ha controls *Sclerotinia* in oilseed rape (Davies and Muncey, 2004). A significant reduction in disease development and lesion size has been observed by Dhawan (1980) with the spray application of Benlate and Topsin-M in *B. juncea*. The dicarboximide fungicides (*e.g.* iprodione, procymidone and dimethachlon) have also been widely used for the control of SR for more than 6 years in Jiangsu Province. However, it appears that repeated applications of dicarboximide fungicides have resulted in the emergence of resistant strains in several plant pathogens including *S. homoeocarpa* and *S. minor* (Jo *et al*., 2006; Smith *et al*., 1995). Shivpuri and Gupta (2001) have demonstrated that Bavistin, TPM and phenyl pyrrate had inhibitory effect on *S. sclerotiorum* growth *in vitro*. In a recent study, the occurrence of insensitivity to dimethachlon in *S. sclerotiorum* was reported in Jiangsu Province (Ma *et al*., 2009). One alternative to achieve and optimize disease control and minimize the risk of resistance development is to incorporate new fungicides with contrasting modes of action into a spray program (Staub, 1991). Boscalid (trade name 'Cantus' in China), is a new broad-spectrum fungicide belonging to carboximides class, inhibits succinate ubiquinone reductase (complex II) [also known as succinate dehydrogenase (SDH)], in the mitochondrial electron transport chain (Zhang *et al*., 2007). Analysis of the data set from official field trials of the German state extension service during 1991–2003 has shown that only 33% of fungicide treatment had been economically effective (Dunker and Tiedemann, 2004). Further, disease management through chemical control may also prove ineffective due to difficulty in timing of

the fungicide application with the release of ascospores (Bolton *et al*., 2006).

Extensive research on foliar-applied fungicides to control SR of canola has been reported from Canada (McKenzie and Verma, 1992a,b; McKenzie *et al.,*1988, 1989; Morrall and Verma 1987, 1988; Morrall *et al.,* 1983, 1984a,b;1985; Verma, 1982a,b,c;1984; Verma and Morrall, 1984, 1987;Verma *et al.,*1983, 1985, 1986, 1987; Verma and Mckenzie, 1982; Gugel and Verma, 1986). During the early eightees, SR of rapeseed/canola in Canada was managed by spraying fungicide Benlate with the use of a small aircraft (aerial application). In the absence of epidemiological information, especially regarding conditions favourable for carpogenic germination and importance of petals, in both initiation and development of infection, the control by aerial application of fungicides was erratic and often unsuccessful. Other problems with the aerial application of fungicides were unavailability of aircraft, poor crop coverage, and loss of fungicides through drift, because the aircraft can not fly close to the ground. Aerial application and application of fungicides by a tractor mounted sprayer (ground level application) were compared (Verma, 1984; Morrall and Verma, 1987, 1988; Morrall *et al.*1983, 1984 a,b; 1985). Compared to the aerial application, ground level application of fungicide was cheaper, provided better crop coverage and disease control, no loss of fungicides through drift, and easily available, because most farmers owned their own tractor-mounted sprayer. Loss of crops in tractor–driven area/track was minimal because most fallen plants sprung back and tractor-driven areas were not visible after a few weeks. In Canada, ground level application of fungicides is the most commonly used method for controlling SR of canola.

Based on the petal infestation forecast model a split-level fungicide application technology was studied (Morrall *et al.,* 1984b; 1985) where, only half the recommended dose of fungicides was applied if the risk of the disease incidence was low. If the weather turns favourable for high disease incidence after 2-3 weeks, the remaining half dose can be applied. Significant control of SR was obtained even with the half recommended dose, and in most cases, no further fungicide application was needed.

Future Strategy and Priorities for Disease Management

S. sclerotiorum is a major challenge faced by plant pathologists. Management is difficult, inconsistent and uneconomical due to the presence of wide host range and long-term survival of the resting structures. Biotechnological methods viz., gene transfer, *Agrobacterium tumefaciences* mediated transformation, protoplast culture, somatic hybridization and microplast techniques should be exploited for developing transgenic plants of crops with superior resistance to *Sclerotinia.* Several strategies including detoxification defence, activation and general inhibition have potential to engineer *Sclerotinia* resistance. It is necessary to understand disease epidemic in variable environmental conditions. The integrated disease management strategy including cultural, chemical, biological and host resisitance should be refined, retested and revalidated under changing environmental conditions. The main emphasis should be on the disease management aspect with the followings:

i. Effect of weather on different stages of pathogen/ pathogenesis/ disease cycle.
ii. Development of precise and quick diagnostic tools.
iii. Monitoring of occurrence, distribution and severity of disease.
iv. Analyses of host-pathogen-environment interaction for developing disease forecasting models.
v. Development of simple and functional disease forecasting models.
vi. Use of Information Technology (IT) to manage, storage, processing, analysis and presentation of data.
vii. Dissemination of disease management technology.
viii. Identification of multiple disease resistant sources.

ix. Analysis of genetic diversity in populations of host and pathogen.

x. Induced resistance and systemically acquired resistance (SAR).

xi. Identification of disease tolerance and partial resistance genes.

xii. Genetics of virulence and virulence spectrum.

xiii. Best use of IPM and IDM technology.

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

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