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REVIEW ARTICLE

Sclerotinia sclerotiorum – A threat and challenge for crops

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ABSTRACT: *Sclerotinia sclerotiorum* (Lib.) de Bary is a soil borne plant pathogen, capable of infecting more than 500 host plants worldwide. It is a major pathogen that plays a crucial role in reducing the yield in economically important crops. It causes stem & crown rot, wilt, leaf & petiole rot, blossom blight, fruit rot and damping off diseases in crops. The initial mycelia infection at the base of the stem is an appearance of elongated water soaked lesions that expand rapidly. Ascospore infection is quite general and occurs on the other plant parts. Effective pathogenesis by the *S. sclerotiorum* requires the secretion of oxalic acid and lytic enzymes. Germination of overwintered sclerotia, and release, survival and germination of ascospores are important factors for the development of disease. The capability of sclerotia to survive for more than 5 years, so it becomes very difficult to manage the *Sclerotinia* diseases. Management of *S. sclerotiorum* is a major challenge, and the best being the integration of various Measures like, cultural practices, chemical, biological and resistance varieties. The review summarizes current information on morphology, symptomatology, disease cycle and management of the disease. In addition, current research and management strategies to combat *S. sclerotiorum also* discuss.
Keywords: *Sclerotinia sclerotiorum,* rot, management.

INTRODUCTION

Sclerotinia sclerotiorum is a ubiquitous necrotrophic pathogen that attacks a wide range of cultivated and wild plant species including canola (oilseed rape), mustard, alfalfa, soybean, field-bean, lentil, field pea, and sunflower. It results in damage of the plant tissue, followed by cell death and soft rot or white mould of the crop (Purdy, 1979). Initially the pathogen was first reported to infect sunflower during 1861 (Huang, 1983). It causes head rot of, leaf blight of canola, and pod rot of dry bean, blossom blight of alfalfa and lettuce drop. Most of the plants susceptible to the necrotrophic pathogen belong to *Solanaceae, Cruciferae, Umbelliferae, Compositae, Chenopodiaceae* and *Leguminosae*.

Systematic position: Acc. To Krik *et al.* (2008)

This pathogen classified as: Domain- *Eukarya,* Kingdom- *Fungi,* Phylum- *Ascomycota,* Sub phylum- *Pezizomycotina,* Class- *Leotiomycetes,* Order- *Helotiales,* Family, *Sclerotinaceae*, Genus - *Sclerotinia,* Species- *sclerotiorum.*

Several workers reported *S. Sclerotiorum on* various crops

Scientist	Crop	Year
Shaw and Ajerakar	Rapeseed and mustard	1915
Rai and Agnihotri	Gaillardia	1970
Sehgal and Agrawat	Fennel, potato, sunflower	1971
Pones *et al.*	Lettuce, cabbage and bean	1979
Huang	Sunflower	1983
Hall *et al.*	Grapes	2002
Chen *et al.*	Chickpea	2006
Prova *et al.*	Hyacinth bean	2014

Morphology

- Hyphae are hyaline, septate, multinucleate and thin walled.
- Sclerotia are round, semi spherical to irregular in shape. Sclerotia have light coloured interior called medulla. Covering with rind. The rind contains melanin pigment which are highly resistant to degradation.
- Apothecia (small mushroom) are cup shaped, light brown and vary in size from 2-11mm in diameter.
- Apothecia are formed on a slender stalk of 20-80 mm in length called stipe.

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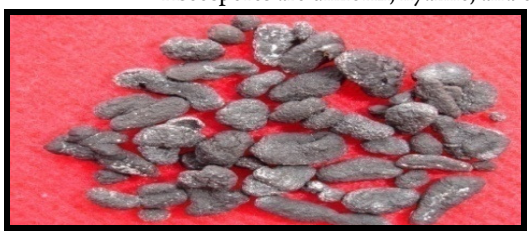
- Asci are cylindrical, narrow, rounded at apex with 8 ascospores per ascus.
- Ascospores are uniform, hyaline, and ellipsoidal with smooth walls.


Sclerotia


Apothecia with stipe

Economic importance

Sclerotinia sclerotiorum is omnipresent and has a very wide host range and causes economic losses in crops such as oilseeds, pulses, forage legumes, vegetables and ornamentals.

Crop	Losses	According to
Soyabean	19 %	Anderson and Tenuta (2001)
Canola	5-100%	Manitoba Agriculture, F.A.R.I. (2002)
Mustard	60 % & 30-90 %	Ghasolia & Shivpuri (2004)
Bean	92%	Schwartz and Steadman (1989)
Pigeon pea	60-80 %	Schwartz and Steadman (1989)
Brinjal	26-47 % PDI	Iqbal *et al.,* (2003)

Symptomatology

Symptoms differ among host species; however, there are a number of similarities as well. Common symptoms are the appearance of water-soaked irregular spots on fruits, stems, leaves, or petioles. These spots enlarge and a cottony mycelium covers the affected area. The fungus spreads and the plant turns into a soft, slimy, water-soaked mass. The cottony mycelium produces numerous sclerotia (black seed-like reproductive structures) after host death, which is a reliable diagnostic sign of *Sclerotinia* infection.

Symptoms on various crops

1. Sunflower (Head & Stem rot) - Appearance of water soaked lesion on the stem or receptacle of the head. Rot extends on both directions on the stem and head leading to rotting of the head and stem. Head of sunflower appears broom like. Cottony mycelium and sclerotial bodies are produced on varying shapes on the affected portions.

2. Mustard Stem rot - Initial symptoms of stem rot appear as water soaked spots on leaves or stems. Later the lesions on the leaf extend to petiole and infect the stem. Lesions on the stem appear as a pale grey to white lesions on the stem at or above the soil surface. As disease advances it spreads to upper branches including pods. Finally stem girdles at the point of infection, leading to wilting and death of the plant. Black sclerotial bodies are produced on or inside the hollow stem.

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3. Bean (White mould)- Symptom develops as pale colored, water-soaked lesions after full bloom on blossoms as white cottony growth. Leaves, stems and pods in contact with the colonized blossoms are infected. Infected tissue turns dry and has a chalky or bleached appearance. Pathogen produces black sclerotia on the affected host tissues. Finally the infected plants die off with severe yield reduction.

4.Soyabean (Stem rot)- Blossom infection leads to the development of water soaked symptoms on stem or pod, which often results from infected flowers. Few days after infection diseased stem are killed and become tan and eventually dries and shred. Infected plant parts have signs of the fungal pathogen as white, fluffy mycelium during humid conditions and sclerotia on the surface of the stem.

5. Cabbage (Head rot)- Symptoms first appear as water soaked spots on lower or upper cabbage leaves. Water soaked spots enlarge, infected tissue becomes soft, and outer leaves begin to wilt. As the disease progress a white cottony growth becomes evident on the leaves. Finally the entire cabbage head would be covered with white cottony growth followed by the development of sclerotia on the head.

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Disease cycle

Sclerotia of *S. sclerotiorum remain* viable in soil for many years. It imbibes moisture from moist soil and leads to germination of the sclerotia. Sclerotia germinate to produce apothecia (Carpogenic germination) or directly produce mycelium (Myceliogenic germination). Apothecia develop most rapidly when soils are saturated and temperatures are in the range of 10 to 20°C. Fungal infection and mycelial growth is maximized in the presence of free water on the plant surfaces. Apothecia liberate ascospores into the air and land on the petals. Infection was initiated via the senescing petals that serve as an initial source of nutrients for the germination of ascospores landing on petals. Upon establishment the fungus deploys two main pathogenicity determinants, the secretion of oxalic acid and a battery of acidic lytic enzymes released by the advancing mycelium. Stems and petioles are infected, vascular tissues are disrupted, and stems, pods, or leaves beyond the site of infection die. As nutrients are exhausted, fungal mycelia aggregate into sclerotia that form both inside and outside the plant stem. These sclerotia then fall to the ground and over winter for years (Schwartz and Steadman, 1978). During the favorable environmental conditions resting structures germinate and initiate the disease cycle again.

Pathogenicity factors

Sclerotinia sclerotiorum secretes multiple pathogenicity factors. Degradation of plant cell wall, its components and tissue maceration occur by the concerted action of several extracellular lytic enzymes. Effective pathogenesis by *S. sclerotiorum requires* the secretion of pathogenicity factors like oxalic acid (Cessna *et al.,* 2000), extracellular lytic enzymes such as cellulases, hemicellulases and pectinases, aspartyl protease, endopolygalacturonases and acidic protease (Riou *et al.,* 1991). 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. Reduction in extracellular pH, activate the production of cell wall degrading enzymes (Marciano *et al.,* 1983). OA directly limits host defense compounds by suppressing the oxidative burst. In conjunction, plant cell wall-degrading enzymes, including cellulolytic and pectinolytic enzymes, cause maceration of plant tissues, necrosis followed by plant death. Thus the release of an array of lytic enzymes and the oxalic acid from the growing mycelium is the pathogenicity factors that are required for the establishment of the host-parasite relationship.

Pre disposing factors

Temperature and soil moisture are key factors affecting sclerotial germination. Continuous moisture and 15-17 °C temp. (10 -14 days) are required for apothecial development. No apothecial formation at 30 °C or 5 °C. Approximately 2-3 days of continuous leaf wetness is required for leaf infection by ascospores. Increase nitrogen fertilizer also increase the Scleotinia disease incidence. Several factors also favor the disease: Over irrigation, Mono culturing, High seed rate & impure seed mixed with sclerotia, Imbalanced fertilizer application (Urea), infected stubbles left in the fields, Unawareness in farmer's about disease.

Management

Management is very difficult due to wide host range and long survival of sclerotia. The best approach to control the pathogen is by integration of various ecofriendly measures:

Cultural practices

Adopt various cultural practices like: Deep summer ploughing, Crop rotation, Wider row spacing, Proper field sanitation, Flooding of field, adopt quarantine measures, Use sclerotial free and certified seeds, Destruction of weeds, Use balanced fertilizer in proper manner, Proper irrigation, Prediction based management advisory to farmers developed for well time application of pesticides to control disease.

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Bio control/ecofriendly management

- The mycoparasitic fungi: *Coniothyrium minitans, Trichoderma spp., Gliocladium spp.* etc. (Adams and Ayers, 1979).
- *Coniothyrium minitans* occurs naturally in soil as a mycoparasite of *S. sclerotiorum.* It decline the viablilty of sclerotia and suppresses the ascospores release.
- Seed treatment @ 10 g /kg, soil application @ 2.5 kg/ha and two foliar spray @ 0.2 % at 50 and 70 days after sowing *Trichoderma viride* against *Sclerotinia* rot of Indian mustard.
- Soil application of Zinc @25 kg/ha with mustard cake @ 2 ton/ha against SR of mustard. (Sharma, *et al.,* 2011).
- Seed treatment with garlic clove extract (1%) or neem powder (6g/kg seed) against stem rot of chickpea (Pandey *et al.,* 2011).
- Seed and foliar applicaion with gralic clove extract (10 %) against *Sclerotinia rot* of mustard. (Sharma, *et al.,* 2016).
- Seed and foliar application of garlic or eukalyptus (5%) against SR of corainder (Fagodiya *et al.,* 2017).
- *Sclerotinia sclerotiorum hypovirulence associated DNA virus 1* is a single stranded DNA virus with a circular genome that infects the fungus *Sclerotinia sclerotiorum* (Zhang *et al.,* 2009).

Chemical control

- Carbendazim (0.1 %) as seed treatment and foliar spray (65 DAS) against *Sclerotinia* rot of mustard. Seed treatment with carbendazim + mancozeb (0.1%) was equally effective as carbendazim (Ghasolia and Shivpuri, 2008).
- Seed treatment with carbendazim (0.1%) and foliar spray (at 50 DAS) against SR of mustard (Sharma *et al.,* 2017).
- Seed dressing with carbendazim, mancozeb, thiram and bayleton @ 2 g/kg seed against *Sclerotinia* rot of mustard (Pathak and Godika, 2002).

Use of Resistance sources

There is no known commercial plant resistance to this pathogen so management measure depend on disease avoidance.

Few resistant/tolerance sources reported on different crops

Crop	Resistance/tolerant varieties
Sweet potato	Bearegard
Cucumber	Ganfeng 2, zhongnong 2, CI, A 15
Bean	Arka vijay, rajani
Safflower	AC sunset, RHA439
Mustard	Norin-9, BOH-2600, omni nature, jet neuf, cutton, RRN-505,Hyola-401, Kiran, RH-492, PAB-9511
Linseed	Antares, norlin
Pea	Dark skin perfection, perfection-132, PI-155109, PI-263027
Brinjal	V-1198, V-1200, V-1687, V-1740, V-1755
Sunflower	Sunstar 277, HA-390, RHA-391, RHA-392CM-361, CM-497

IDM module against *Sclerotinia* rot of mustard (POP of zone III B Rajsathan)

- Destruction of diseased crop debris of previous crop.
- Deep summer ploughing & leveling of fields for proper drainage.
- Crop rotation with non-host crops like wheat, barley, rice, maize etc.
- Soil incorporation of *Trichoderma* based product @ 2.5kg/ha pre-incubated in 50 kg FYM/ha at the time of field preparation.
- Follow optimum sowing time (16-31 Oct.).
- Apply recommended dose of fertilizers i.e. N: P: K: S – 80:40:40:40.
- Seed treatment with garlic extract @ (2%) or Seed treatment with *Trichoderma* @ 10 g/kg.
- Appropriate seed rate (4 kg/ha) to maintain the optimum plant population in field
- Sowing of healthy, certified and clean seeds free from sclerotial bodies.
- Use of Tolerant varieties – RRN- 505.
- Removal of collateral host and weeds viz; Chenopodium sp., *Asphodelus* (pyazi).
- At 50% flowering need based foliar spray of *Trichoderma* @ 0.2% or 0.05 % carbendazim towards stem soon after disease appearance at 20 days interval may help to check the spread of disease.

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- Excessive irrigation should be avoided (follow sprinkler irrigation).
- Removal of infected plants and stubbles from fields as soon as possible

Future perspectives

- Utilization of disease resistance sources.
- Development of disease prediction system.
- Mode of action of individual partial genes is being investigated.
- Possible resistance mechanism includes rapid localization of the invading pathogen and altered toxin response.
- Sclerotial germination could be predicted on the basis of rainfall and temperature data and readily incorporated into a forecasting system.
- Genetic variation for resistance/avoidance.

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CONFLICTS OF INTEREST
"The authors declare no conflict of interest".