International Journal of Botany Studies
www.botanyjournals.com
ISSN: 2455-541X
Received: 29-11-2021, Accepted: 15-12-2021, Published: 30-12-2021
Volume 6, Issue 6, 2021, Page No. 1471-1475

Sclerotium rolfsii: A soil-borne fungal menace to crop production

Ritesh Kumar[1*], Nidhi B Thakur[2]
[1] Department of Plant Pathology, MSSSoA, Centurion University of Technology and Management, Odisha, India
[2] Department of Agricultural Economics and Extension, SoA, Lovely Professional University, Punjab, India

Abstract
Cultivation of agricultural crops are largely affected by several biotic factors. Among that, collar rot causes significant damage to variety of crops and ultimately responsible for yield loss. Collar rot is caused by Sclerotium rolfsii, a soil inhabitant and non-specific facultative parasite that found in almost all types of agro-ecological systems. Wide variation among isolates and adaptability of the pathogen to high temperature suggesting to develop improved strategy for management. The problem associated to this fungus may be increased in future, particularly under changing climate. Development of fungicide with multiple mode of action can be a reliable option to manage this pathogen.

Keywords: collar rot, *Sclerotium rolfsii*, soil-borne, variability

Introduction
Sclerotium rolfsii Sacc. is a well-known fungal plant pathogen having a wide host range, which is one of the most destructive soil inhabiting plant pathogens and causing collar rot, that results in heavy loss in different crops across the globe as well as in India. The fungus infects over 500 plant species, mostly comprised of dicotyledonous and few monocotyledonous plants. It induces symptoms like crown and root rot, collar rot, foot rot, stem rot, stem canker, damping off, southern wilt or blight or southern stem rot (Punja, 1985) [30]. The fungus was first detected by Rolfs in 1892 as one of the reason for tomato blight in Florida. Later, Saccardo (1911) [35] named the fungus as *Sclerotium rolfsii*. Higgins (1927) [17] studied about the physiology and host range of the pathogen. Its perfect stage was first studied by Curzi (1931) [11] and he proposed generic name as *Corticium*. Mundkur (1934) [25] successfully isolated the perfect stage of *S. rolfsii*. McClintock (1917) [22] and Butler and Bisby (1931) [6] reported that collar rot disease caused by this fungus for the first time from USA and India, respectively.

Distribution and economic importance
Collar rot disease is wide spread and cause serious losses in countries like Bolivia, China, Egypt, India, Taiwan, Thailand and USA (Bowen *et al.*, 1992) [5]. In India, the disease occurs regularly in areas of states like Maharashtra, Gujarat, Madhya Pradesh, Karnataka, Andhra Pradesh, Orissa and Tamil Nadu. *Sclerotium rolfsii* is predominantly distributed in tropical and subtropical countries. It is common where high temperature exist during the rainy season. It is estimated that over 5, 00, 000 ha of fields were infected by this pathogen and yield losses of over 25% have been recorded in several crops (Mayee and Datar, 1988) [20]. Reddy *et al.* (1971) carried out survey during 1969-70 in Karnataka and found that, the losses due to collar rot in wheat crop were about 5%. In a survey on foot rot of wheat during *rabi* 1978-79 and *rabi* 1979-80 in Malaprabha, the maximum disease incidence of 10.20% and 5.20% in rainfed and irrigated fields, respectively (Nargund, 1981) [26]. Palakshappa (1986) [28] reported maximum disease incidence of 30% in betel vine in Chikkodi taluka of Belgum district. Harlapur (1988) [15] observed incidence about 9.85% and 4.66% under rainfed and irrigated conditions, respectively of foot rot of wheat caused by *Sclerotium rolfsii*. Sharma and Pathak (1994) [38] conducted a field experiment at Sriganganagar in India during 1982-84 to investigate yield and sucrose losses in the sugarbeet caused by *Sclerotium rolfsii*. They recorded the reduction of yield and sucrose by 46.50% and 62.20%, respectively. Mehan *et al.* (1995) [23] reported that the disease caused by *S. rolfsii* affected groundnut in many countries. The reduction in yield by 10-25% and pod loss of more than 50% in heavily infected fields. Hanumanthegowda (1999) [14] carried out survey on stem rot of groundnut during *kharif* 1998-99 and rabi/summer, 1998-99 in Dharwad, Belgaum and Havier districts. He reported a maximum disease incidence of 12.57% and 8.68% in rained and irrigated fields, respectively. Southern blight of soybean caused by this pathogen was first reported in Nigeria with yield reduction of about 59% under severe conditions (Aken and Dashiell, 1991) [2].

Symptoms caused by the pathogen
Symptoms like pre-emergence as well post-emergence death of seedlings was reported by Agrawal and Kotashane (1971) [1]. The disease generally develops on isolated plants, scattered throughout the field wherever the inoculum is present. The spread to adjacent row can be rapid under favourable conditions. A sudden yellowing, browning and wilting of the entire plant are the first symptoms (Fig 1). The most characteristics sign of the disease is white, fan like mat of fungal mycelium that forms on stem bases, leaf debris and the soil surface around the infected plants. The mycelial mat may extend several centimeters up to the stem above the soil line (Fig 2). The symptoms of stem rot involve mycelium covering the plant stem near the soil surface and produced organic acids, which were toxic to living plant tissue (Wilson, 1953) [44]. This followed the necrosis of plant cells. The mycelium invaded the stem,

gynophores and also pods and caused rotting of the tissues. Beattie (1954) [3] also observed same symptoms on infected plants. Wheeler (1969) [43] reported the symptoms as young plant killed rapidly, but older plant turned yellow and wilted subsequently. The white mycelium often appeared on stem base of drying plants. Siddaramaiah (1979) observed foot rot symptoms accompanied by girdling of younger plants and later such plants were succumbed to death. The infection on older plants was also observed on roots as well as pegs at harvest. The rotted pegs broke off and left the pods in the soil, there by resulting in considerable yield losses. The infected seedlings became stunted with chlorotic leaves and ultimately withered and died. Nyvall (1989) [27] also described the symptoms as wilting of the plants due to infection of stem at base level. Further, soil level near the infected stem was covered with white mycelium. The wilted plants remained upright. The infected areas of stem shredded and covered with numerous sclerotial bodies. Stem base was covered with elongated and overcrowded brown lesions. These formed reddish colour. During dry whether brown lenticular lesions occurred on stem just below the soil surface. The peg infection caused light to dark brown lesions (0.05–2.00 cm long), which resulted in tissue shredding and pod loss. Lesions on young pods of Spanish peanuts were orange yellowish to light tan in colour. Severely decayed kernels were shriveled and covered with mycelium.

Fig 1: Symptoms of *Sclerotium rolfsii* in isolated plants, scattered in the field of chickpea.

Fig 2: Mycelial mat of *Sclerotium rolfsii* extended to the stem above the soil line

Pathogenicity

Differences in morphology and pathogenicity in the isolates of *S. rolfsii* was reported by Edson and Shapvalov (1923) [13] from North Carolina and Arkansas. One of those two isolates was more virulent in potato and seed decay. Sengupta and Das (1970) [36] conducted the cross inoculation studied of isolates of *S. rolfsii* from groundnut, wheat, potato, guava and Bengal gram. They reported that, Bengal gram was the most susceptible host of *S. rolfsii*. But, all the isolates were most virulent to their appropriate hosts. The specialization was not demonstrated conclusively. Datar and Bindu (1974) [12] proved the pathogenicity of *S. rolfsii* on sunflower by soil inoculation method under glass house conditions. The inoculum was prepared by growing the fungus on sterilized maize bran medium and mixed with the sterilized soil one week before sowing. Typical symptoms were produced within a week of germination which was identical to those produced in the field. Mishra and Bais (1987) [24] used 15 days old fungal culture grown on sand corn meal medium for proving pathogenicity of root rot of barley caused by *S. rolfsii* by mixing upper 4-5 cm layer of soil with inoculum at the rate of one flask per pot. Siddaramaiah and Chandrappa (1988) [41] proved the pathogenicity of *S. rolfsii* on cardamom in pot culture studies by inoculating 25 days old sclerotial cultures which was grown on sand corn meal medium and observed the symptoms a week after inoculation.

Morphological and Cultural variation in *Sclerotium rolfsii*

Variation is a rule in most of the root infecting fungi. The variation may arise following change in crop cultivation, genetic modification of hosts, physical or chemical modification of the soil, environment or accidental introduction of new genetic material into a region or local gene pool. It may also be a way of survival of the pathogen under adverse conditions. Typical characters of *Sclerotium rolfsii* Sacc was described by Subramanian (1971) [42]. The fungal mycelium was very floccose, not ropy, producing numerous sclerotia. Sclerotia were pinkish buff to olive brown to clove brown in colour, globuse shape, 82. 5 mm in diameter. *S. rolfsii* formed hymenium which was aerolate, putty colored, 30-40 µ thick. Basidia were ovoid, 79 × 4-5 µ, each bearing 2 or 4 parallel or divergent sterigmata and 2.5 × 4-6 µ long. Basidiospores were elliptical to obviate, hyaline, smooth, rounded or apiculate at base measuring 6-7 × 3.5-5 µ. Kim (1974) [18] grouped the isolates of *S. rolfsii* on the basis of difference in morphology, mode of growth in culture and pathogenicity. The mycelium of *S. rolfsii* was very floccose, snow white, thick, cottony and grown rapidly all over the blotting paper during blotter tests while, sclerotia were the resting infective propagules of the fungus and can be seen on the mycelial strands. They were globose to ellipsoid, pinkish, buff to olive brown to clove brown. Young sclerotia appeared white, producing characteristics exudes droplets around them, which slowly become dark as aged and resemble to mustard seeds, 1-2 mm in diameter when mature. Manjappa (1979) [19] found variation among the isolates of *S. rolfsii* from different crop (sunflower, groundnut, wheat, red gram, tomato, lucerne and tamarind). All the eight isolates showed marked differences in the rate of growth on both solid and liquid media and time taken for sclerotial initiation. The isolates also differed with respect to size, number and weight of sclerotia and the virulence of pathogen. Radwan *et al.* (1987) [31] have also observed variation in mycelial colour and sclerotial production in five isolates collected from different fields. Prithviraj *et al.* (1996) [29] found two types of sclerotia, *viz.*, 'small' and 'large'. Hernandez and Ysla (1997) [16] evaluated eight isolates of *S. rolfsii* for cultural and morphological characteristics and found variability in their mycelial density, number and diameter of sclerotia, mycelial density, presence of rhizomorphs and duration of sclerotial formation.

Effect of different fungicides on *Sclerotium rolfsii*

Shalby (1997) [37] studied the effect of benlate, vitavax, Rhizolex T and Tecto TBZ (thiabendazole) on disease severity and as seed treatments on soil fungi causing sesame root rot and concluded that benlate and vitavax were the most effective seed dressings against the *Macrophomina phaseolina* under both laboratory and green house conditions. Rao *et al.* (1998) [32] reported that Seed treatments consisting of carbendazim WP, carbendazim SD + captan, carbendazim + thiram, captan, thiram, mancozeb, tolclofos-methyl, pyroquilon, TCMTB, carbedazim-Jkstein, quintozene, captafol, carboxin and triadimenol were tested for the management of these fungi under artificial infection of the seed. The seed treatments were found to control the damage due to these fungi. Among the treatments, carbendazim SD (0.05% + captan 0.125%) was the best in reducing seed rot and pre and post-emergence seedling blight and in increasing pod yields significantly in all the three seasons of field experimentation, followed by treatment with captafol (0.25%). Choudhary *et al.* (2004) [10] observed the effectiveness of four fungicides, *i.e.* Bavistin, Antracol, Indofil M-45 and Ridomil MZ applied at 300, 400, 500 and 1000 ppm, in inhibiting the mycelial growth of *Macrophomina phaseolina*, the causal agent of stem and root rot of sesame, was studied *in vitro* using the poisoned food technique. All fungicides dose-dependently inhibited mycelial growth compared with the untreated control, with Bavistin being the most effective.

Adaptability of pathogen to higher temperature regimes

Climate change in terms of enhancing global temperature will change the pattern of disease through changes in host distribution and phenology, changes in plant associated microflora and direct biological effects on rapidly adapting pathogens. Atmospheric CO_2, a major greenhouse gas, has increased by nearly 30%and temperature has risen by 0.3 to 0.6°C (Chakraborty *et al*., 1998) [8]. Evaluation of the limited literature in this area suggests that the most likely impact of climate change will be felt in three areas: in losses from plant diseases, in the efficacy of disease management strategies and in the geographical distribution of plant diseases. Change in temperature could have positive, negative or no impact on individual plant diseases.

According to Chakraborty *et al*. (2000) [9] changes in temperature regimes due to climate change may alter the growth stage, development rate and pathogenicity of infectious agents, and the physiology and resistance of the host plant. Increase in temperature has been also reported to cause a shift in the geographical distribution of host pathogens (Mboup *et al*., 2012) [21]. Models suggested by Boland *et al*. (2004) [4] predict that expected climate change will significantly affect the occurrence of plant diseases in agriculture and forestry in the coming years. Direct, multiple effects on the epidemiology of plant diseases are expected, including the survival of primary inoculum, the rate of disease progress during a growing season, and the duration of epidemics. These effects will positively or negatively influence individual pathogens and the diseases they cause. Changes in the spectra of diseases are also anticipated. Abiotic diseases associated with environmental extremes are expected to increase, and interactions between biotic and abiotic diseases might represent the most important effects of climate change on plant diseases.

Disease assessment due to pathogen

The assessment of disease is vital to our interpretation as to whether disease management practices are successful. Relative magnitudes in success of disease management are judged on a comparative basis by growers and scientist through disease assessment. A slightly different approach to measuring disease incidence reliability was taken by Shokes *et al*. (1987) [39] in the assessment of foliar diseases; however, the approach is equally applicable for collar and root diseases. The goal was to provide a measurement of agreement among several disease evaluators to estimate the importance of disease evaluation. Campbell and Neher (1994) [7] stated that cost is a factor of primary importance in planning the assessment of any disease. Cost of effort must be balance against the need for a certain amount of information. According to Campbell and Neher (1994) [7] the general goal of disease assessment will remain constant for all types of studies. The goal is to provide reliable estimates of the amount of diseases in an area (plot, field, farm, country, region etc.) based on the specific symptoms and signs, which are known to be the characteristics of the disease at the lowest reasonable cost with known confidence.

Conclusion

A number of crops are cultivated, which share a large contribution in total agricultural production and their lower productivity is attributed by many constrains including pathogenic stress, among them collar rot is a major problem, which is caused by *Sclerotium rolfsii*. The wide host range, prolific growth and ability to produce persistant sclerotia enable this pathogen for the potential economic loss in agricultural production system. Pathogens exhibits variation in terms of morphological, biological, chemical and their pathogenic character Wide variation among isolates of pathogen indicates genetic exchange in mycelia among the isolates is occurred in nature. This hypothesis has a vivid point because the sexual stage of this fungus is rare in nature and its role in the life cycle of the fungus is unknown. Further, adaptability of the pathogen to high temperature suggests for development of 'new' modules of management for this pathogen in the field scale under scenario of changing climate associated with elevated temperature. Additionally, the forecasting models of *S. rolfsii* can be developed based on temperature adaptability. Each pathogen has got its own cardinal temperatue and understanding the temperature of the pathogen will help to standarize the management practices. But the adaptability of the pathogens towards higher temperature in the arena of global warming and climate change will disturb various models of crop protection so from the management side, these needed to be upgraded regularly.

References

1. Agrawal SC, Kotashane SR. Resistance in some of soybean varieties against *Sclerotium rolfsii* Sacc. Indian Phytopathology,1971:24:401-403.
2. Aken CN, Dashiell KE. First report of southern blight caused by *Sclerotium rolfsii* on soybean in Nigeria. *Plant Dis,*1991:75:537.
3. Beattie JH. Growing peanuts United States Department Agriculture. *Farmers Bull*, 1954, 552.
4. Boland GL, Melzer MS, Hopkin A, Higgins V, Nassuth A. Climate change and plant diseases in Ontario. *Can. J. Plant Pathol*,2004:26:335-350.
5. Bowen KL, Hagan AK, Weeks SR. Seven years of *Sclerotium rolfsii* in peanut field; yield losses and means of minimum. *plant dis*,1992:76:982-985.
6. Butler EJ, Bisby GR. Fungi in India. *Science Monograph*, Indian Council of Agric. Res. New Delhi, 1931, 552.
7. Campbell CL, Neher DA. Estimating Disease Incidence and Severity. Epidemiology and management of root disease,1994:2:36-45.
8. Chakraborty S. Potential impact of climate change on plant diseases of economic significance to Australia. Australas. Plant Pathol,1998:27:15-35.
9. Chakraborty S, Tiedemann AV, Teng PS. Climate change: potential impact on plant diseases. *Environmental Pollution*,2000:108:317-326.
10. Choudhary CS, Singh SN, Prasad SM. *In vitro* effectiveness of chemicals to control *Macrophomina phaseolina* (Tassi.) Goid, causing stem and root rot of sesame. *J. Appl. Biology*,2004:14(1):46-47.
11. Curzi M. Studies on *Sclerotium rolfsii*. *Patol veg roma*, Ser,1931:2(11):306-373.
12. Datar VV, Bindu KJ. Collar rot of sunflower, A new host record from India. *Curr.Sci*,1974:43:496.
13. Edson HA, Shapvalov M. Parasitism of *Sclerotium rolfsii* on Irish potatoes. *J.Agric.Res*,1923:23:4-46.
14. Hanumanthegowda B. Studies on stem rot of groundnut caused by *Sclerotium rolfsii* Sacc. M.Sc. (Agri). Thesis, Uni. of Agric. Sci, Dharwad, 1999, 37-38.
15. Harlapur SI. Studies on some aspects of root rot of wheat caused by *Sclerotium rolfsii* Sacc. M.Sc. (Agri). Thesis, Uni. of Agril Sci, Dharwad, 1988, 64.
16. Hernandez MCA, Ysla LH. Variability among *Sclerotium rolfsii* Sacc. Isolates in cultural, morphological and pathogenic characteristics. *Fitopathologia*,1997:32(3):182-186.
17. Higgins BB. Physiology and parasitism of *Sclerotium rolfsii* Sacc. *Phytopathology*,1927:17:417-448.
18. Kim K. Studies on *Sclerotium rolfsii* Sacc. isolated from *Mangolia kabus* in Korea. Korean J.Pl. Prot,1974:13:105-133.
19. Manjappa BH. Studies on the survival and variation in *Sclerotium rolfsii* Sacc. M.Sc. (Agri) Thesis, Uni. of Agri Sci. Bangalore, 1979, 72-74.
20. Mayee CD, Datar. Diseases of groundnut in Tropics. *Rev. Trop. Pl. Path*,1988:5:85-118.
21. Mboup M, Bahri B, Leconte M, De Vallavieille-Pope C, Kaltz O, Enjalbert J. Genetic structure and local adaptation of European wheat yellow rust populations: the role of temperature specific adaptation. Evol. Appl,2012:5:341-352.
22. McClintock JA. Peanut wilt caused by Sclerotium rolfsii. J. of Agric. Res,1917:8:441-448.
23. Mehan VK, Mayee CD, McDonald D, Ramakrishanan N, Jayanthi S. Resistance in groundnut to *Sclerotium rolfsii* caused stem and pod rots. International J. of Pest Manag,1995:41:79-82.
24. Mishra BK, Bais BS. Studies on seedling blight and foot rot of barley caused by *Sclerotium rolfsii*. Indian phytopathology,1987:40:161-167.
25. Mundkur BB. Perfect stage of *Sclerotium rolfsii* Sacc. in culture. Indian J. Agric. Sci,1934:4:779-782.
26. Nargund VB. Studies on foot rot of wheat caused by Sclerotium rolfsii Sacc. in Karnataka. M.Sc. (Agri) Thesis, *Uni. of Agric. Sci*, Bangalore, 1981, 78-79.
27. Nyvall RF. Field crop Diseases Handbook, Second Edition, Published by Van Nartrand Renhold, New York, U.S.A, 1989, 817.
28. Palakshappa MG. Studies on foot rot of betelvine caused by *Sclerotium rolfsii* Sacc.in Karnataka. M.Sc. (Agri). Thesis, Univ. of Agric. Sci, Bangalore, 1986, 62.
29. Prithviraj B, Singh UP, Kobayashi K, Sugawara KM, Nishimura R, Sakaiwada A. Variability in sclerotial morphology in *Sclerotium rolfsii*: SEM and isoezyme studies. J. Plant Dis. Prot,1996:103:495-501.
30. Punja ZK. The biology, ecology and control of Sclerotium rolfsii. *Annu. Rev. Phytopathology*,1985:23:97-127.
31. Radwan IA, El-Wakil AA, Satour MM, Zayed MA. Comparative morphological and physiological studies on certain isolates of *Sclerotium rolfsii*. Egypt Res. Rev,1987:63:59-69.
32. Rao VV R, Rao KC, Shantaram MV, Reddy MS. Management of seed and seedling diseases of groundnut (*Arachis hypogaea L.*) through seed treatment. *Indian J. Pl. Protec*,1998:26(1):1-8.
33. Reddy MR, Fazalnoor K, Hedge RK. Screening of commercial varieties and genetic stocks of wheat against foot rot caused by *Sclerotium rolfsii* Sacc. Mysore J. of Agric. Sci,1971:5:252-256.
34. Rolfs PA. Tomato blight some hints. Bulletin of Florida Agricultural Experiment Station, U.S.A, 1892, 18.
35. Saccardo PA. Notae Mycologiae. *Annual Mycology,1911:9:249-257.*
36. Sengupta PK, Das CR. Studies on some isolates of *Sclerotium rolfsii*. *Z. pflackranth P. Fl Schutz*,1970:77:582-584.
37. Shalby SIM. Effect of fungicidal treatment of sesame seeds on root rot infection, plant growth and chemical components. Bulletin of Faculty of Agril. Univ. of Cairo,1997:48(2):397-411.
38. Sharma BS, Pathak VN. Yield and sucrose losses in sugarbeet due to root rot. Indian Phytopathology,1994:47:408-411.
39. Shokes FM, Berger RD, Smith DS, Rasp JM. Reliability of disease assessment procedure; a case study with leaf spot of peanut. *Oliageneaux*,1987:42:245-251.
40. Siddaramaiah AL. Stem, sheath and leaf rot disease of cardamom caused by *Sclerotium rolfsii* from India. *Curr. Res*,1988:16:82.
41. Siddaramaiah AL, Chandrappa HM. New collar rot disease on *Desmodium uncimatum* and *Lutononis bainesii* from India. *Curr. Res*,1988:16:83.
42. Subramanian CV. Hyphomycetes, An account of Indian species, except *Cercospora*. ICAR, New Delhi, 1971.

43. Wheeler BEJ. An introduction to plant diseases. Published by the English language Book Society and John Wiley and Sons Ltd., London, 1969, 374.
44. Wilson C. Preventing the Diseases of Peanut. United state Department of Agriculture Year Book, 1953, 448-454.
45.