Ecology of *Sclerotinia* Species

P. B. Adams and W. A. Ayers

Plant pathologist and microbiologist, respectively, Soilborne Diseases Laboratory, Plant Protection Institute, Federal Research, Science and Education Administration, U. S. Department of Agriculture, Beltsville, MD 20705.

Approximately 90% of the life cycle of *Sclerotinia* spp. is spent in soil as sclerotia. At certain times of the year, depending on the inherent nature of the fungus and various environmental factors, the sclerotia germinate and form either mycelium which can infect a host or form an apothecium. Ascospores that develop within the apothecium, are disseminated by air currents and initiate infection when they contact susceptible tissue. After thorough invasion of host tissue by mycelium, sclerotia are formed. These new sclerotia eventually return to the soil environment, survive adverse periods, and await the next susceptible crop.

That portion of the life cycle involving the development of apothecia, host infection, and pathogenesis will be dealt with in other presentations of this symposium. This presentation will focus on dissemination of the fungus and its existence and survival in soil.

Our review of the literature for this presentation revealed references indicating that various species of *Sclerotinia* exist in nearly all sections of the world including Europe, Middle East, Far East, South Pacific, Africa, and South and North America.

DISSEMINATION

Sclerotinia spp. become established and are spread from field to field, and from one geographical area to another, by several means. Windblown ascospores can be a major means of field-to-field spread. This aspect will be discussed in more detail in the epidemiology section of this symposium. *Sclerotinia* spp. also may be disseminted from field to field in soil adhering to seedlings, farm equipment, animals, or man (10,29) in the form of sclerotia or as mycelium in infected host tissue. On farms where diseased plant tissue is used as cattle feed or bedding, the spreading of manure on fields has been shown (10) to be a likely means of introducing the pathogen to uncontaminated fields. In this connection, Brown (4) showed that less than 2% of the sclerotia of *S. sclerotiorum* fed to sheep passed through the digestive tract in a viable condition. Thus sheep, and possibly other animals, fed diseased plant refuse and then turned out to pasture, could spread the pathogen to *Sclerotinia*-free fields. Irrigation also has been shown to be involved in the spread of *Sclerotinia* species from field to field. Steadman et al (30) showed that sclerotia of *S. sclerotiorum* could be collected from waterways and in irrigation runoff from fields in Nebraska. Such sclerotia remained viable for at least 10–21 days in flowing water. Patterns of movement of the sclerotia were correlated with previous or current season infection of bean plants.

Probably the greatest potential for long distance dissemination of *Sclerotinia* spp. is either by seed infected with mycelia or by seed contaminated with sclerotia. *Sclerotinia*-infected or infested seed has been reported for sunflower (35), cabbage (22), cauliflower (22), kale (22), clover (10), bean (29), lupins (7), peanut (26), rape, and barley (J. Dueck, *personal communication*).

NATURAL INOCULUM DENSITIES

Although much has been written about the biology and ecology of *Sclerotinia* spp., very little has been reported on natural populations of sclerotia in soil. Henderson (13) reported finding from zero to 20 sclerotia of *S. sclerotiorum* per 929 cm^2 (1 ft^2) of field soil to a depth of 5.1 cm (2 in.). This is the equivalent of 0 to three sclerotia per kilogram of soil. Working with sunflower, Hoes and Huang (14) found approximately two to three sclerotia of *S. sclerotiorum* per kilogram of nonrhizosphere soil. However, they found about 24 sclerotia per kilogram of soil in the rhizosphere of diseased plants. Abawi and Grogan (1) determined the inoculum density of *S. sclerotiorum* at various depths in a bean field in New York. At depths of 0–2.5, 2.5–10, and 10–17.5 cm the number of sclerotia were approximately 7, 2, and 0.5 sclerotia per kilogram of soil, respectively, before plowing and near zero at all depths after planting. The inoculum densities of *S. sclerotiorum* in bean fields in western Nebraska were shown to range from 0.1 to 6.2 sclerotia per kilogram of soil (28). On the basis of this limited amount of information, it appears that the levels of sclerotia in natural soils range from zero to less than 10 sclerotia per kilogram of soil in a field ready for planting.

There appears to be no published information on the natural sclerotium densities of *S. minor*, *S. borealis* or *S. trifoliorum*. We have determined inoculum densities of a number of the fields in the eastern United States (P. B. Adams, *unpublished*). In three New York State muck fields, with a history of severe lettuce drop, the inoculum density of *S. minor* ranged from 160 to 820 sclerotia per kilogram of soil; in 11 mineral soil fields in New Jersey, the inoculum density of *S. minor* ranged from 0 to 230 sclerotia per kilogram of soil. In a field with a history of severe Sclerotinia peanut blight in Virginia, we found the inoculum density to range from 35 to 100 sclerotia per kilogram of soil. Thus, in fields with a history of losses due to *Sclerotinia* species the inoculum density of

S. minor was found to be 10 to 100 times greater than that of *S. sclerotiorum*.

The sclerotial inoculum density of *Sclerotinia* species can be increased in two ways: (i) by the production of secondary sclerotia in soil, and (ii) by the production of sclerotia on their hosts. Williams and Western (34) and others have shown that sclerotia of *S. sclerotiorum* and *S. trifoliorum* are capable of forming secondary sclerotia in soil in the absence of a host. Although these two species, as well as *S. minor* (Adams, *unpublished*), can form secondary sclerotia and thereby increase their numbers, very little is known about the extent of this phenomenon in nature, what factors affect its occurence, or its significance in relation to survival or to disease levels.

Anyone who has observed crop losses due to *Sclerotinia* spp. knows the importance of the host in the production of sclerotia. Stevens (31) reported that if a plant bed 2.7 × 61 m (9 × 200 ft) containing 2,000 lettuce plants were diseased, as many as 17,000 sclerotia of *S. minor* would be produced and eventually worked into the soil. If thoroughly incorporated into the soil to a depth of 15 cm, this would increase the inoculum density by about 0.5 sclerotia per kg soil. Adams (2) reported that as many as 1,000 sclerotia of *S. minor* formed on a single, diseased, romaine lettuce plant. Assuming that each plant occupied 929 cm^2 (1 ft^2) of a field and each plant produced 1,000 sclerotia, the inoculum density of the field would be increased by 50 sclerotia per kilogram of soil when the diseased crop was worked into the soil to a depth of 15 cm.

An often neglected factor is the production of sclerotia on weed hosts. Schwartz (H. F. Schwartz, *personal communications*) compiled a host range of *S. sclerotiorum* (*S. minor, S. sclerotiorum,* and *S. trifoliorum*) in which 361 species in 225 genera among 64 plant families were listed as hosts. Weed hosts are a significant factor in the production of sclerotia in the field and should not be overlooked.

SURVIVAL IN SOIL

Sclerotia are the structures which allow species of *Sclerotinia* to survive for long periods of time under adverse conditions. Information on how long sclerotia usually survive in nature in a particular field is not readily available. The best information we have is based on the experience of farmers and is only an approximation. For sclerotia of *S. trifoliorum*, Dillon-Weston et al (10) estimated that sclerotia can survive for 6-8 yr. Tribe (32) reported that *S. trifoliorum* sclerotia buried at a depth greater than 6.4 cm persisted for about 8 yr.

Davis (9) was of the opinion that sclerotia of *S. sclerotiorum* near the soil surface do not remain viable for more than 1 yr. Young and Morris (35), however, reported that at least a 4-yr rotation was needed before sunflowers can be grown on a field with a history of Sclerotinia wilt. In the case of bean white mold, Starr et al (29) suggested a 3- to 5-yr period of nonhost crops. Cook (8) reported that rotating beans with corn and sugarbeets every 3rd yr was not an effective control practice in Nebraska.

In New Jersey, farmers usually can grow lettuce successfully for 3–4 yr before lettuce drop caused by *S. minor* becomes too severe. Experience has shown that if these fields are used for nonhost crops for 4 yr they can then be cropped with lettuce for another 3–4 yr (J. K. Springer, *personal communication*).

Based on information provided by growers' experience, sclerotia of *S. trifoliorum* survive in nature for about 8 yr, whereas sclerotia of *S. sclerotiorum* and *S. minor* survive for 4–5 yr.

Brown and Butler (5) reported that under favorably dry conditions sclerotia of *S. sclerotiorum* remain visible for at least 10 yr. This is in contrast to the estimated 4–5 yr of survival for sclerotia in field soil.

Factors affecting survival. Temperature and soil pH appear to be of minimal importance in affecting survival. In our studies (P. B. Adams, *unpublished*) in Maryland, sclerotia added to small field plots with differing soil textures and pH, survived well during the winter months. The results from laboratory studies show that normal soil temperatures (10–30 C) did not adversely affect survival. A constant soil temperature of 35 C for 3 wk or more, however, did reduce survival of sclerotia (2). This high temperature for a prolonged period of time would not be expected under natural field conditions in a temperate region. Moore (21) reported that nearly 100% of the sclerotia were killed when soil was flooded with water for 26–31 days. This phenomenon was thought to be due to activities of various members of the soil microflora.

The most significant component of soil affecting survival of sclerotia appears to be biological. More than 30 species of fungi and bacteria have been implicated by various workers as antagonists or mycoparasites of *Sclerotinia* spp. For all but a few, tests for parasitic or antagonistic activity were conducted exclusively in vitro on culture media or under sterile conditions. Thus, proof of antagonistic activity of most species under natural conditions generally is lacking. Fourteen species of fungi belonging to the genera *Acrostalagmus, Fusarium, Gliocladium, Hormodendrum, Mucor, Penicillium, Trichoderma,* and *Verticillium* were described as parasitic on sclerotia of *S. trifoliorum* (20) based on in vitro tests. Bacteria, particularly a low-temperature, fluorescent *Pseudomonas* sp., were implicated as exerting an important influence in suppressing the spread of *S. trifoliorum* on frozen red clover leaves in Finland (23, 24, 25). Erviö et al (11) studied the survival of sclerotia of *S. trifoliorum* on the surface of soil in the field. They observed destruction of sclerotia by snails, fungi, and bacteria; most sclerotia were destroyed during the first summer. They felt that bacteria were more important than fungi in causing destruction of the sclerotia.

Species of *Aspergillus, Penicillium,* and *Stachybotrys* isolated from decaying sclerotia were shown to be antagonistic to *S. sclerotiorum* in vitro by Rai and Saxena (27). They reported that sclerotia on the soil surface were strongly colonized by *Penicillium* spp. under field conditions. However, they did not provide information about the extent of the colonization or provide unequivocal evidence of mycoparasitism. Bedi (3) reported that all of the sclerotia mixed into nonsterile soil were killed after 3 wk after the addition of a potato-dextrose broth culture of *Aspergillus flavus*. The rapidity and completeness of this result suggest the effect may have been caused by toxic metabolites in the medium rather than by mycoparasitism.

Certain isolates of *Trichoderma* spp. appear to be parasitic on sclerotia of *S. sclerotiorum* in soil, but others do not. Jones and Watson (19) noted that four single-spore isolates of *T. viride* infected sclerotia in vitro, but only one of the isolates decayed sclerotia on moist sand and in soil. Makkonen and Pohjakallio (20) observed destruction of sclerotia of *S. trifoliorum* by *T. viride* on sterilized sand but did not test the parasitism in soil. Sclerotia of *S. sclerotiorum* were more resistant than those of *S. trifoliorum* to *T. viride* and susceptibility of sclerotia of *S. sclerotiorum* varied considerably. They found that one apparently resistant strain was attacked only if the sclerotia were first injured by freezing or by pricking. They later tested *T. viride* and other fungi as parasites of *S. trifoliorum* and *S. sclerotiorum* under field conditions and concluded that *Trichoderma* spp. played a small role in the natural destruction of sclerotia (11).

Tribe (32) noted that *T. viride* and *Gliocladium roseum* commonly appear when soil particles were placed on cultures of *S. trifoliorum* and that both soil fungi frequently overrun *S. trifoliorum* on agar medium, but that neither infected sclerotia in soil. On the other hand, Huang (15) observed destruction of 58% of the sclerotia in soil infested with an isolate of *T. viride.* Thus, although *Trichoderma* spp. frequently are observed to be surface colonizers of sclerotia removed from soil and placed under moist conditions or on culture media, only certain strains appear to parasitize sclerotia under natural conditions.

Coniothyrium minitans is well established as a mycoparasite of *Sclerotinia* spp. under natural conditions. This mycoparasite first was isolated from sclerotia of *S. sclerotiorum* in California and described as a new species by Campbell (6). He demonstrated in laboratory experiments that *C. minitans* parasitized sclerotia of *S. sclerotiorum* and produced pycnidia upon and within the sclerotia. Pycnidiospores were exuded from the pycnidia as a black liquid mass and could be used to infect healthy sclerotia.

Coniothyrium minitans also was isolated by Tribe (32) from

sclerotia of *S. trifoliorum* in England. He showed that *C. minitans* was parasitic to *S. trifoliorum* sclerotia in vitro and in nonsterile soil. In a loamy sand or a heavy clay soil from 85–95% of the introduced sclerotia were killed by the mycoparasite within 11 wk. The soils remained highly infective to *S. trifoliorum* for at least 14 mo.

Little is yet known of the physiology of the mycoparasitism by *C. minitans*. Jones and Watson (19) and Jones et al (18) described lysis of the pseudoparenchymatous tissue of the sclerotia of *S. sclerotiorum* which they attributed to exoenzymes, particularly β-glucanase and chitinase, produced by *C. minitans*. Ghaffar (12) speculated that melanolytic enzymes played a role in permitting entry through the sclerotial rind. Serial tissue sections showed that pycnidia developed on and inside of the sclerotia. Huang and Hoes (17) described penetration of hyphae of *S. sclerotiorum* by *C. minitans* hyphae without the formation of specialized penetration structures. The host cytoplasm disintegrated and cell walls collapsed as a result of infection. The ability of *C. minitans* to parasitize sclerotia of *S. sclerotiorum* inside plant roots and stems as well as those on the root surface of infected sunflower plants was demonstrated by Huang (16).

While studying the behavior of sclerotia of *S. minor* in soil, we have isolated three fungi which destructively infect sclerotia. The first, *Sporidesmium sclerotivorum* was described as a new species (33) and has been found in soil samples from fields in California, Louisiana, Maryland, New Jersey, New York, North Carolina, and Oregon. Under laboratory conditions in natural soil, this fungus infected 100% of the sclerotia in 6 wk and caused destruction of 95% or more in 10 wk. This mycoparasite is unique in that it grows through natural soil of various textures from an infected sclerotium to a healthy sclerotium producing conidia as it spreads through soil (W. A. Ayers and P. B. Adams, *unpublished*). The mycoparasitism by this fungus occurs in natural soils of varying textures at all agricultural pH levels, a wide soil moisture range, and is favored by soil temperatures in the range of 15 to 25 C (P. B. Adams and W. A. Ayers, *unpublished*). We suspect, based on a limited amount of data, that this mycoparasite is a component of the soil microflora that is responsible for the natural decline of sclerotia in soil. Another mycoparasite that we have isolated from New Jersey soil samples appears to belong to a new genus. A third mycoparasite has been found in soil samples from a California lettuce field. This fungus appears to be a new species of the genus *Teratosperma*. Little is known of these mycoparasites other than that they behave like *Sporidesmium* in infecting sclerotia and spreading through soil. These recent findings suggest that our knowledge of mycoparasites as natural agents of destruction of sclerotia is still in its infancy. Much progress in this area may be expected in the next few years.

CONCLUSIONS

It is quite surprising how little we know about the ecology of these important plant pathogens. In the area of dissemination of the fungus, we seem to know a great deal; yet, we usually do not know how new fields brought into cultivation become infested with *Sclerotinia* spp. Was this due to wind-blown ascospores, contaminated seed or by other means? Why is it that fields in North Carolina and Virginia where peanuts were grown for years with no evidence of Sclerotinia blight now sustain severe losses due to the disease: Could this be related to changes in the agronomic practices used for this crop?

Not much information is available on the natural inoculum densities of the various species of *Sclerotinia* in field soils. This kind of information is urgently needed, especially in fields used to grow vegetable crops. With sufficient information, one should be able to predict disease severity based on the inoculum density of the pathogen at the time of planting the crop. Such a disease-forecasting system could identify fields which need disease control measures and fields which do not. Such a disease-forecasting system would be very useful in an integrated pest management program.

Sclerotia of *Sclerotinia* species generally survive in soil for 3–8 yr. Soil temperatures, pH, and moisture appear to have little *direct* effect on their survival. The biological component of soil appears to be the major factor which determines the survival of the sclerotia in soil.

Many microorganisms in the soil have a detrimental effect on sclerotia. *Coniothyrium minitans* and certain *Trichoderma* spp. have been firmly established as destructive mycoparasites of *Sclerotinia* spp. In addition, we have recently found in our laboratory three mycoparasites of *Sclerotinia* spp. that may be responsible for the natural destruction of sclerotia in soil and may have potential as biological control agents. What about the rest of the soil flora and fauna? This work should be intensified for it may lead to practical biological control measures.

In the last 10 yr we have made much progress in our understanding of the ecology of *Sclerotinia* spp. It seems probable that much more will be learned about the ecology of *Sclerotinia* spp. in the next 10 yr.

LITERATURE CITED

1. ABAWI, G. S., and R. G. GROGAN. 1975. Source of primary inoculum and effects of temperature and moisture on infection of beans by *Whetzelinia sclerotiorum*. Phytopathology 65:300-309.
2. ADAMS, P. B. 1975. Factors affecting survival of *Sclerotinia sclerotiorum* in soil. Plant Dis. Rep. 59:599-603.
3. BEDI, K. S. 1961. Factors affecting the viability of sclerotia of *Sclerotinia sclerotiorum* (Lib.) deBary. Indian J. Agric. Sci. 31:236-245.
4. BROWN, J. G. 1937. Relation of livestock to the control sclerotinosis of lettuce. Phytopathology 27:1045-1050.
5. BROWN, J. G., and K. D. BUTLER. 1936. Sclerotiniose of lettuce in Arizona. Pages 475-506 in Ariz. Agric. Exp. Stn. Tech. Bull. 63.
6. CAMPBELL, W. A. 1947. A new species of *Coniothyrium* parasitic on sclerotia. Mycologia 39:190-195.
7. CHAMBERLAIN, E. E. 1932. Sclerotium-disease (*Sclerotinia sclerotiorum*) of tomatoes. N. Z. J. Agric. 45:260-268.
8. COOK, G. E. 1973. Some aspects of the epidemiology of white mold on dry beans cause by *Whetzelinia sclerotiorum* in western Nebraska. Ph.D. thesis. Univ. of Nebraska, Lincoln, 81 pp.
9. DAVIS, W. H. 1925. Drop of chinese cabbage and our common cabbage caused by *Sclerotinia sclerotiorum* (Lib.) Massee (*Sclerotinia libertiana* Fckl). Phytopathology 15:249-259.
10. DILLON-WESTON, W. A. R., R. A. LOVELESS, and E. R. TAYLOR. 1946. Clover rot. J. Agric. Sci. 36:18-28.
11. ERVIÖ, L.-R., A.-M. HALKILAHTI, and O. POHJAKALLIO. 1964. The survival in the soil of sclerotia of two *Sclerotinia* species and their ability to form mycelia. Adv. Front. Plant Sci. 8:121-133.
12. GHAFFAR, A. 1972. Some observations on the parasitism of *Coniothyrium minitans* on the sclerotia of *Sclerotinia sclerotiorum*. Pak. J. Bot. 4:85-87.
13. HENDERSON, R. M. 1962. Some aspects of the life cycle of the plant pathogen *Sclerotinia sclerotiorum* in Western Australia. J. West Aust. Roy. Soc. 45:133-135.
14. HOES, J. A., and H. C. HUANG. 1975. *Sclerotinia sclerotiorum*: viability and separation of sclerotinia from soil. Phytopathology 65:1431-1432.
15. HUANG, H. C. 1976. Biological control of *Sclerotinia* wilt in sunflowers. Pages 69-72 in Rep. Annu. Conf. Manit. Agron. 1976.
16. HUANG, H. C. 1977. Importance of *Coniothyrium minitans* in survival of sclerotia of *Sclerotinia sclerotiorum* in wilted sunflower. Can. J. Bot. 55:289-295.
17. HUANG, H. C., and J. A. HOES. 1976. Penetration and infection of *Sclerotinia sclerotiorum* by *Coniothyrium minitans*. Can. J. Bot. 54:406-410.
18. JONES, D., A. H. GORDON, and J. S. D. BACON. 1974. Cooperative action by endo- and exo-β-(1→3) glucanases from parasitic fungi in the degradation of cell-wall glucans of *Sclerotinia sclerotiorum* (Lib.) deBary. Biochem. J. 140:47-55.
19. JONES, D., and D. WATSON. 1969. Parasitism and lysis by soil fungi of *Sclerotinia sclerotiorum* (Lib.) deBary, a phytopathenic fungus. Nature 224:287-288.
20. MAKKONEN, R., and O. POHJAKALLIO. 1960. On the parasites attacking the sclerotia of some fungi pathogenic to higher plants and on the resistance of these sclerotia to their parasites. Acta Agric. Scand. 10:105-126.
21. MOORE, W. D. 1949. Flooding as a means of destroying the sclerotia of *Sclerotinia sclerotiorum*. Phytopathology 39:920-927.

22. NEERGAARD, P. 1958. Mycelial seed infection of certain crucifers by *Sclerotinia sclerotiorum* (Lib.) D By. Plant Dis. Rep. 42:1105-1106.
23. POHJAKALLIO, O., and A. SALONEN. 1950. On the appearance and significance of fungi and bacteria antibiotically affecting the fungus *Sclerotinia trifoliorum* Erikss. Maatalous. Aikak. 22:63-67.
24. POHJAKALLIO, O., A. SALONEN, and E. LAAKKONEN. 1949. Investigations into the bacteria antibiotically affecting the fungus *Sclerotinia trifoliorum* Erikss. Physiol. Plant. 2:312-322.
25. POHJAKALLIO, O., A. SALONEN, and E. RELANDER. 1953. Investigations into the micro-organisms limiting damage by the clover rot fungus. Acta Agric. Scand. 3:53-60.
26. PORTER, D. M., and M. K. BEUTE. 1974. *Sclerotinia* blight of peanuts. Phytopathology 64:263-264.
27. RAI, J. N., and V. C. SAXENA. 1975. Sclerotial mycoflora and its role in natural biological control of 'white-rot' disease. Plant Soil 43:509-513.
28. SCHWARTZ, H. F,, and J. R. STEADMAN. 1977. *Sclerotinia sclerotiorum* inoculum production in Western Nebraska. Bean Improv. Annu. Rep. 20:69-70.
29. STARR, G. C., H. J. WALTERS, and G. H. BRIDGMON. 1953. White mold (*Sclerotinia*) of beans. Wyo. Agric. Exp. Stn. Bull. 322.
30. STEADMAN, J. R., C. R. MAIER, H. F. SCHWARTZ, and E. D. KERR. 1975. Pollution of surface irrigation waters by plant pathogenic organisms. Water Resour. Bull. 11:796-804.
31. STEVENS, F. L. 1911. A serious lettuce disease. N. C. Agric. Exp. Stn. Bull. 217.
32. TRIBE, H. T. 1957. On the parasitism of *Sclerotinia trifoliorum* by *Coniothyrium minitans*. Trans. Br. Mycol. Soc. 40:489-499.
33. UECKER, F. A., W. A. AYERS, and P. B. ADAMS. 1978. A new hyphomycete on sclerotia of *Sclerotinia sclerotiorum*. Mycotaxon 7:275-282.
34. WILLIAMS, G. H., and J. H. WESTERN. 1965. The biology of *Sclerotinia trifoliorum* Erikss. and other species of sclerotium-forming fungi. II. The survival of sclerotia in soil. Ann. Appl. Biol. 56:261-268.
35. YOUNG, P. A., and H. E. MORRIS. 1927. *Sclerotinia* wilt of sunflowers. Montana Agric. Exp. Stn. Bull. 208.