Sclerotinia sclerotiorum: History, Diseases and Symptomatology, Host Range, Geographic Distribution, and Impact

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I am pleased to have this opportunity to discuss some facets of the accumulated information about *Sclerotinia sclerotiorum* deBary that comprise the present understanding of this most interesting fungus. It will be obvious that many contributions to the literature about *S. sclerotiorum* were not considered in the preparation of this paper. However, I hope that what is presented is historically accurate and that this initial paper of the Sclerotinia Symposium will help initiate a new focus on *S. sclerotiorum*.

HISTORY

In the beginning, Madame M. A. Libert described *Peziza sclerotiorum* (24); that binomial for the fungus stood until Fuckel (14) erected and described the genus *Sclerotinia*; he chose to honor Madame Libert by renaming *Peziza sclerotiorum* with a newly coined binomial, *Sclerotinia libertiana*. According to Wakefield (40), Fuckel apparently disliked the combination *S. sclerotiorum* and elected to establish the new one. Authors in the United States, and others elsewhere, accepted and used *S. libertiana* Fuckel until Wakefield (40) 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, but de Bary used it in his 1884 contribution (8). Thus, the proper name and authority for the subject fungus of this symposium seems to be *Sclerotinia sclerotiorum* (Lib.) de Bary. Although technically incorrect, the name *S. libertiana* occasionally is used in this paper to be consistent with its use in the literature prior to 1924.

It is literally impossible to consider the history of *S. sclerotiorum* without some comments about taxonomy and nomenclature, even though those subjects will be discussed in another paper of this symposium. My accounting of the history of the *S. sclerotiorum* will be sensu Purdy (31) in which I relegated to synonomy with *S. sclerotiorum* several species described from various hosts and locations. Since 1955 no new information has appeared that would justify alteration of my stated opinion. However, there are some points that provoke thought regarding speciation within the genus, such as: the type of mycelial germination of some small-sclerotia types described by Smith (35), Adams and Tate (1), and others; the mycelial interactions reported by Wong and Willetts (43); and the microanatomical investigations of various fungal structures proposed by Korf and Dumont (23), to name a few. To the best of my knowledge, Korf and Dumont (23) are correct in that prior to 1972 results of microanatomical studies or other critical anatomical studies do not exist if de Bary's work with sclerotia and other structures is not considered. However, de Bary was seeking to establish an understanding of events related to the life cycle of *S. sclerotiorum* rather than looking for differences in isolates of the fungus. Korf and Dumont (23) pointed out that taxonomic positions have been assigned on the basis of *gross* (my italics) measurement of asci, ascospores, paraphyses, apothecia, and sclerotia of the fungus, and I add, the associated host plant. These are facts and are accepted as such; however, Korf and Dumont (23) stated that transfer of epithets to *Whetzelinia* would be premature until the requisite microanatomical studies are undertaken. Although it was not stated, I infer their meaning to be that, pending the outcome of such studies, transfer of epithets to *Whetzelinia* may be made if justified by sufficient other evidence.

It is my opinion that eventually *Whetzelinia* Korf & Dumont, will be considered a superfluous name, that *Sclerotinia* Fuckel will be retained for the generic epithet, and that the type species (lectotype) will continue to be *Sclerotinia sclerotiorum* (Lib.) de Bary as proposed by Dennis (10) and Buchwald and Neergaard (4). *S. sclerotiorum* (Lib.) deBary certainly is well implanted in the literature, it is an important plant pathogen, and (in my opinion) nothing is gained by casting tradition aside if an alternative exists which would permit retention of the genus name *Sclerotinia* for *S. sclerotiorum* and *S. tuberosa*.

Eriksson (13) described the pathogen of clover stem rot as *Sclerotinia trifoliorum* Erikss. Then Wolf and Cromwell (42) suggested that clover stem rot may have been present near Berberbeck in Hesse, Germany in 1857. They also mentioned that the disease name may be synonymous with the name clover sickness, a disease known in England in the early 1800's and on which personnel at Rothamstead Experiment Station began work in 1849. However, the disease apparently was not attributed to *S. trifoliorum* until 1897.

According to Wolf and Cromwell (42) the first report of *S. trifoliorum* in the USA was published in Delaware in 1890. Wolf and Cromwell (42) stated that the sclerotia varied in size from 0.3 mm to 10.0 mm. They sent cultures of the clover stem rot pathogen from North Carolina to Prof. R. E. Smith who judged that the similarities of that isolate to *S. libertiana* (*S. sclerotiorum*), ".... leaves no doubt that the fungus really is *Sclerotinia libertiana*."

Although Smith (34) described a small-sclerotia form of *S. libertiana* that had caused serious losses of lettuce and other glasshouse crops, he considered it to be *S. libertiana* that had lost the ability to produce apothecia. Other workers observed a small-sclerotia type, but none except Jagger (18) considered it to be "... an undescribed species of Sclerotinia clearly distinct from *S. libertiana*." Jagger (19) described the small-sclerotia type (which had been isolated from lettuce, celery, and other crops in several

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locations in New York and from lettuce in Sanford, Florida) as *Sclerotinia minor* for which a range in sclerotial size of 0.5 to 2.0 mm was recorded. Valleau et al (38) suggested that *S. minor* and *S. trifoliorum* are identical, with *S. minor* occurring on a host (lettuce) not commonly recognized as a host for *S. trifoliorum*. Thus, associated host and size of sclerotia were used as the basis for speciation of Sclerotinia isolates. Additional species were named:

Sclerotinia intermedia Ramsey
Sclerotinia serica Keay
Sclerotinia trifoliorum Eriks. var. *fabae* Keay
Sclerotinia sativa Drayton and Groves

It is interesting that Dennis (9) included *S. sclerotiorum, S. trifoliorum* (also the variety *fabae*), *S. minor, S. serica,* and *S. tuberosa* but did not mention *S. intermedia* or *S. sativa*, which suggests that he did not recognize these latter two as valid species or that these species occur only in the new world. It appears that others share concepts or parts of concepts with Dennis, because *S. intermedia, S. sativa,* and *S. serica,* along with *S. trifoliorum* var. *fabae,* seem to have disappeared from the literature as if they were the "Piltdown men" of *Sclerotinia* spp.

Price and Calhoun (29) inoculated 11 hosts with 19 different isolates of *S. sclerotiorum* and reported differences in the extent of attack on individual hosts, as well as differences in host plant susceptibility to different isolates. They concluded that there was no evidence for physiological specialization of the pathogen. That conclusion was supported by Mordue and Holliday (26) in their Commonwealth Mycological Institute description of *Sclerotinia sclerotiorum*.

Cultural variants and geographic variation of *S. sclerotiorum* have been reported and races have been designated on the basis of the amount of mycelial growth, numbers of sclerotia, and apothecial production. Chivers (6) designated three races of *S. minor* based on cultural characteristics but indicated no relationship between cultural differences and pathogenic capability.

According to Keay (22) pathogenicity is valueless for establishing the systematic position of *Sclerotinia spp.* There is ample evidence in support of Keay's position in that many isolates from widely different hosts produce similar if not identical diseases, as was suggested by Valleau et al (38) for *S. minor* and *S. trifoliorum*.

Production of apothecia in different seasons in nature as suggested by Korf and Dumont (23) may have significance. The possibility for confusion already exists in that Valleau et al (38) and Williams and Western (41) observed apothecia of *S. trifoliorum* in both the fall and spring and apothecium production throughout the winter in southern Florida (November to March). A climatological analysis may be required for accurate species designation. Other parameters also may be essential; latitude, in particular, as well as elevation could add needed information. For example, is it possible that isolates from latitudes far from the equator (20) might behave in a manner similar to isolates from near the equator?

DISEASES AND SYMPTOMATOLOGY

What plant diseases are caused by *S. sclerotiorum*? A survey of the literature revealed more than sixty names used to refer to diseases caused by this omnivorous pathogen; alphabetically they range from banana fruit rot to wilt and admittedly some names may have been missed.

Certain diseases may serve as indicators of the history and symptomatology of diseases caused by *S. sclerotiorum*. To me, lettuce drop is classic. Smith (34) demonstrated beyond question that *S. sclerotiorum* (he used *S. libertiana*) caused "drop" and that *S. sclerotiorum* and *Botrytis cinerea* are different fungi. These two fungi produced similar diseases of lettuce, but the true drop caused by *S. sclerotiorum*, was more common on glasshouse lettuce in Massachusetts in the late 1890's than was the disease caused by *B. cinerea* (34).

According to Stevens and Hall (36), G. E. Stone reported that J. E. Humphrey had lettuce drop under observation in 1888 or 1889, but did not state what caused the problem. Drop was recorded in 1892 by Humphrey and, in 1895, L. H. Bailey pictured a lettuce-drop-affected plant that might have been infected by *S. sclerotiorum*. Also, according to Stevens and Hall (36), Rolfs (of *Sclerotium rolfsii*) (in a letter to Stevens) reported in 1896 that Sclerotinia drop was severe on lettuce in Gainesville, Florida, and that some fields of lettuce were destroyed almost completely.

Stevens and Hall (36) report that lettuce drop occurred in Massachusetts in 1890, in Florida in 1896, in North Carolina in 1897, and in Wisconsin in 1904. In contrast, Burger (5) described the occurrence of lettuce drop in the vicinity of Gainesville in 1896 and in North Carolina in 1897. But it was not until 1900 that this classic disease was attributed to *S. libertiana* (*S. sclerotiorum*) in Massachusetts (34).

Smith (34) discussed his observations of the pathogen of the "real drop" many of which suggest that he was observing the disease caused by a small-sclerotia form of *S. sclerotiorum*. He observed direct infection by mycelium from sclerotia in soil, spread (in the greenhouse) from infected to healthy plants by growth in (or over) soil, mycelia produced by sclerotia placed on sand (more copious growth from older than from younger sclerotia), and the formation of secondary sclerotia. Drop caused by a small-sclerotia type of *S. sclerotiorum* was more common than that caused by a large sclerotia type according to Smith (34).

Jagger (18) stated that the fungus Smith (34) had described was the same one he had observed associated with lettuce in several areas of New York and suggested that this fungus was an "... undescribed species of *Sclerotinia* ...". Thus, Jagger (19) described the small-sclerotia type as *Sclerotinia minor*.

According to Beach (2), Smith, in 1900, reported that this small-sclerotia form could renew mycelial growth, but that it had lost the ability to produce apothecia. Jagger (19) reported the induction of apothecia, their measurements, and measurements of asci, ascospores, and sclerotia, and other parameters considered to be of taxonomic importance at that time. Adams and Tate (1) reported infection of lettuce directly from sclerotia (via mycelium) without an intermediate source of nutrients (ie, organic matter). In contrast, I reported that to achieve infection of lettuce from sclerotia of the large-sclerotia type nonliving or detached lettuce leaves, must be in contact with sclerotia and the lettuce stem (30).

I am aware of very few reports of direct isolation of *S. trifoliorum* from lettuce, but several individuals have demonstrated pathogenicity to lettuce by inoculations with isolates considered to be *S. trifoliorum*. Held and Haenseler (17) suggested that severe attacks of lettuce drop in fields planted for the first time following clover or lucerne (alfalfa) may be caused by *S. trifoliorum*. Because lettuce is not a forage legume, isolations of any so-called large-sclerotia type of *Sclerotinia* appears to have been reported as *S. sclerotiorum* and small-sclerotia types as *S. minor*. For the most part, *S. minor* has been reserved for small sclerotial isolates obtained from lettuce, but as Valleau et al (38) point out, this situation exists because lettuce is not considered to be a host for *S. trifoliorum*.

Inasmuch as Eriksson (13) did not present measurements for sclerotia of *S. trifoliorum*, the range of sclerotial sizes (0.3 to 10 mm) reported by Wolf and Cromwell (43) for *S. trifoliorum* is as reliable as any other measurements reported for the species. It is obvious that sclerotia of *S. trifoliorum* (0.3-10.0 mm) may be smaller than sclerotia of *S. minor* (0.5-2.0 mm) (19) or within the extremes of sclerotial size of *S. sclerotiorum* (1.0-30.0 mm) (31). Apparently species designation for isolates of *Sclerotinia* frequently has been based on host association, size of sclerotia in culture or on the host, and personal preference.

It has been suggested that infection of lettuce by *S. sclerotiorum* can originate directly from sclerotia in soil (1,30,34) but Coley-Smith and Cooke (7) disagree. Infection also results from ascospores as reported by Newton and Sequeira (27).

Symptoms of lettuce drop have been recorded by many individuals and may be reiterated here as a composite of various descriptions as follows: only the outer (wrapper) leaf may wilt, giving the plant a one-sided appearance of stress; as more outer

leaves become infected they wilt and lay flat on the soil surface; heart leaves remain erect and the entire plant may appear light in color. A soft watery decay follows, and subsequently white mycelium and black sclerotia are produced on, in, and around infected plants. Infection takes place often at the leaf base and on the stem at or near the soil line.

Stem rot of clovers and other forage legumes invariably has been attributed to *S. trifoliorum*. As stated previously, the disease was known in Germany in 1857. Gilbert and Bennett (15) report that Eriksson credited Herman Hoffman with the first report in 1863 that clover sickness was caused by what he (Eriksson) later named *S. trifoliorum*. Gilbert and Bennett (15) as well as Wolf and Cromwell (42) stated that Rhem in 1872 designated the pathogen of clover stem rot as *Peziza ciborioides* Fries, a name considered to be untenable by Eriksson (13) who described the pathogen as *S. trifoliorum*.

A liberal interpretation of *S. trifoliorum* by some individuals seems to have been made because almost all isolates of *Sclerotinia* spp. obtained from forage legumes have been designated *S. trifoliorum*.

The first report of clover stem rot in the United States was in 1890 (42); subsequently many reports have been published about this disease and the pathogen that causes it.

Infection of leaves of red clover is reported to originate from ascospores from apothecia produced in the fall (38). However, Wadham (39) observed apothecia as early as 31 August and as late as 20 October. Valleau et al (38) and Williams and Western (41) observed apothecia in the spring that they considered to have been produced from sclerotia of *S. trifoliorum*. Wadham (39) also reported that disease symptoms were apparent in November and December. The pathogen advances from plant to plant by mycelium, but primary inoculum is, according to several reports, ascospores.

Symptoms of clover stem rot are a wilting of leaves and stems. The infected leaves become yellow, die, and turn brown. They fall to the soil surface and from them a white mycelial growth develops that infects stems; the stems progressively decay and a watery soft rot develops. White mycelia may develop on stems, leaves, and on soil where these plant parts lay, and sclerotia form in the mycelial mass. Roots near the soil surface also may be affected. Dijkstra (12) reported that freezing infected plants or parts of such plants, enhances the spread of the pathogen from infection sites.

According to Jones (21) stalk rot of sunflower beyond doubt is caused by *Sclerotinia libertiana* (*S. sclerotiorum*). He suggested that confusion reigned because some thought the pathogen to have been *Sclerotium compactum*, which later was shown to be the sterile condition of *Sclerotinia libertiana*. About 1920, the disease became important in sunflower production in the United States. Morris and Swingle (25) reported that the disease was caused by a large-sclerotia type that was found in Montana. They suggested that infection probably occurs from ascospores and also from sclerotia in soil. Often symptoms first are observed at the soil line where a scaly lesion develops. The bark around the crown and roots is killed, a white mycelium develops and sclerotia are produced on the plant surfaces and also in the pith cavity of the stalk (stem). The leaves become yellow and die, infection spreads up the stalk and the entire plant dies, often only vascular tissue and disorganized parenchyma remain. Flowers and sometimes the entire flower head are susceptible to infection by ascospores (25).

Although Sclerotinia blight of peanut had been reported in China in 1935 and in Argentina in 1950, the disease was not reported in the United States until 1974. Beute et al (3) reported that infection of peanut plants was initiated in the tap root near the soil line, in lateral branches along soil contact points, and in pegs at the soil line from which the fungus advances to lateral branches. Lesions on branches are light tan with distinct demarcation between diseased and healthy tissue. Lesions turn dark brown followed by severe shredding of tissues. These observations are clear testimony to mycelial infection originating from direct (eruptive) germination of small-type sclerotia in soil. Beute, et al (3) also state that organic debris is essential for successful infection.

In addition to the forage legume stem rot disease, several other diseases develop following infection by ascospores that are the primary inoculum. Smith (33) discussed cottony rot of lemons that develops through dead petals, into twigs and into the fruit. Fruit rot is the phase of the disease reported by most observers. Smith (35) described the life history of *S. sclerotiorum* in relation to green fruit rot disease of apricot that results from ascospores germinating, growing on the calyx that adheres to the fruit, and then invading the fruit. Taubenhaus and Ezekiel (37) reported that limb blight of fig was traced to infection via ascospores. Dickson (11) reported a wilt of greenhouse tomatoes and confirmed the pathogen to be *S. libertiana* (*S. sclerotiorum*). Purdy and Bradin (32) described the role of ascospores in the tomato (wilt) stem rot disease. Of course, there are other examples, but these serve to illustrate diseases of aboveground plant parts resulting only from infection by ascospores.

White mold of beans is a disease usually initiated by air-borne ascospores. White mold is a disease of significance in the field and in postharvest environments. Infection of bean plants can occur any time after seedling emergence. Although young seedlings may be attacked by mycelium from soil-borne sclerotia resulting in a damping-off symptom, this problem occurs infrequently. Infection usually is more abundant after blossoming begins. Concomitantly, apothecia are produced within the bean field or in adjacent fields, and ascospores borne by wind alight on petals that are already nonfunctional or soon will become so. These senescent or dead petals are a source of nonliving organic matter through which infections result. According to Harter and Zaumeyer (16) the first field symptom is the appearance of irregularly shaped watersoaked spots on stems followed by similar spots on branches and leaves. These spots or lesions enlarge followed by a soft watery rot of affected plant parts. Cottony mycelium spreads over affected parts and sclerotia develop. Leaves distal to lesions become yellow, turn brown, and often abscise. Multiples stem lesions can kill plants.

Pods are invaded by mycelium that advances from infected branches, by contact with the soil in which mycelium from infected abscised leaves infect them, or by mycelium originating from ascospores that germinate and advance through nonliving plant parts into the pods.

Infected, but often nonsymptomatic, pods are included in shipping containers. Such pods provide inoculum that spreads so rapidly that the entire contents of the container may be destroyed by the time the destination is reached. This condition is referred to as nesting.

My concepts of a generalized life cycle for *Sclerotinia sclerotiorum* and for generalized symptomatology are presented here.

Life cycle of *S. sclerotiorum*. Infection of susceptible host plants can occur from mycelium that originates from eruptive germination of sclerotia in soil. That type of germination is associated with small-sclerotia types. Hyphal germination of sclerotia causes infection by first invading nonliving organic matter and forming a mycelium which is an intermediate necessity for mycelial infection. Apothecia develop from sclerotia located either upon or buried in the soil, and eject ascospores which became airborne and alight on nonliving or senescent plant parts, germinate, ramify the nonliving plant part, and invasion of healthy plant parts results from mycelium that developed from the ascospore. If conditions are not suitable for germination, the ascospore may remain viable for a short time and germinate when conditions again become favorable. There are some reports in the literature that ascospores may penetrate healthy host tissue directly and establish infection. Such reports are few, however, suggesting that this type of infection is rare.

Abundant white mycelium may develop when environmental conditions are favorable, and subsequently, sclerotia are produced externally on affected plant parts and internally in stem pith cavities or fruit cavities, or between plant parts or tissues (bark and xylem). Sclerotia eventually reach the soil where they remain on the soil surface or are buried during farming operations that disturb the soil surface. Apothecia are produced after sclerotia have been "conditioned" or mycelium may develop from sclerotia in soil, thus completing the cycle.

Symptomatology. Penetration of the host cuticle is achieved by mechanical pressure; there is no evidence of pre-penetration dissolution of the cuticle. Following penetration, tissues rapidly become disorganized as a result of enzymatic processes that affect the middle lamella between cells. The first symptoms that often develop on leaves or young stems are watersoaked spots that may enlarge and become a watery soft rot in most hosts. In other hosts, relatively "dry" lesions are produced on stems, stalks, branches, or twigs with a sharp demarcation between healthy and diseased tissues. Such lesions enlarge and the plant part is girdled. Distal leaves become yellow and then turn brown followed by death of the isolated portion of plant. Continued fungal activity results in almost total destruction of parenchymatous tissues and the remaining vascular and structural elements of stems, stalks, branches, and twigs have a characteristic shredded appearance.

Infection of plant parts before harvest often results in postharvest disease from spread of the fungus from diseased to healthy tissue in storage or shipping containers.

Signs of the fungus in association with almost all hosts are copious amounts of white cottony mycelium when environmental conditions are suitable and the subsequent production of black sclerotia of variable size and shape.

HOST RANGE

Sclerotinia sclerotiorum appears to be among the most nonspecific, omnivorous, and successful of plant pathogens; Little can be said about its host range that cannot be envisioned quite adequately by a few numbers. P. B. Adams (*personal communication*) has compiled a host range in which hosts of *S. sclerotiorum* occur within taxonomic groups as follows: 64 plant families, 225 genera, 361 species, and 22 other (cultivars, etc.), for a total of 383 species and other categories.

The 64 plant families are listed in Table 1 along with the numbers of host genera and species.

Some points of interest are that four Gymnosperm hosts in the family Pinaceae are included; two narcotic-producing plants are hosts, *Papaver somniferum* L. and *Cannabis sativa* L.; there are 62 host species in 39 genera in the family Compositae, and the majority of hosts are herbaceous.

GEOGRAPHIC DISTRIBUTION

That *S. sclerotiorum* occurs in relatively cool and moist areas of the world is a generalized statement made by many individuals. However, *S. sclerotiorum* also occurs in localities generally considered to be hot and dry. When the temperature approaches the freezing point (O C) or when temperatures of more than 32 C prevail, *S. sclerotiorum* is much less active than at temperatures between these generalized extremes. The generalizations continue in that reports of occurrence or presence in various political units (countries, states, provinces, regions, etc.) are interpreted to mean that *S. sclerotiorum* occurs within that unit. The precise locations and the environmental conditions prevailing at the time the disease or fungus were observed were not specified. It is reasonable to assume that in locations such as Florida *S. sclerotiorum* occurs when crops are grown, such as tomatoes during the winter months (November to mid-March) when south Florida is relatively cool, even cold at times. There are a few crops in the field in Florida during the summer months and some of these are known hosts. Reports of *S. sclerotiorum* in summer are rare in Florida but are common in many other states, such as California, Nebraska, and New York.

S. sclerotiorum has been reported from many countries located in all continents. It is probable that the fungus occurs somewhere in almost every country.

IMPACT OF *SCLEROTINIA SCLEROTIORUM*

According to reports in the literature, crop losses attributable to *S. sclerotiorum* range from 0–100%. Crop losses lead to investigation of diseases. By conducting research we hope to contribute to the understanding of the host–*S. sclerotiorum* relationship or association, and that with adequate understanding we can develop effective control strategies and tactics. There is no question in my mind that if losses caused by *S. sclerotiorum*

TABLE 1. Plant families and numbers of genera and species within which hosts of *Sclerotinia sclerotiorum* have been reported [a]

Plant Family	Genera (No.)	Species (No.)	Plant Family	Genera (No.)	Species (No.)	Plant Family	Genera (No.)	Species (No.)
Actinidiaceae	1	1	Aizoaceae	1	1	Amaranthaceae	2	3
Annonaceae	2	2	Apocynaceae	1	2	Araliaceae	2	2
Aristolochiaceae	2	2	Asclepiadaceae	2	2	Begoniaceae	1	1
Berberidaceae	1	1	Boraginaceae	4	8	Campanulaceae	2	4
Capparidaceae	1	1	Caryophyllaceae	3	3	Chenopodiaceae	3	3
Compositae	39	62	Convolvulaceae	1	1	Cruciferae	18	32
Cucurbitaceae	3	6	Dipsacaceae	2	3	Euphorbiaceae	2	4
Fagaceae	1	1	Fumariaceae	1	1	Gentianaceae	1	1
Geraniaceae	1	2	Gesneriaceae	1	1	Gramineae	7	7
Hippocastanaceae	1	1	Iridaceae	3	4	Labiatae	4	4
Lauraceae	1	1	Leguminosae	21	52	Liliaceae	5	8
Linaceae	1	2	Malvaceae	5	9	Martyniaceae	1	1
Moraceae	2	4	Musaceae	1	2	Myoporaceae	1	1
Myrtaceae	1	1	Oleaceae	2	4	Onagraceae	2	2
Orobanchaceae	1	2	Papaveraceae	3	4	Passifloraceae	1	1
Pinaceae	4	4	Plantaginaceae	1	1	Polemoniaceae	1	2
Polygonaceae	3	3	Portulacaceae	1	1	Ranunculaceae	7	14
Rosaceae	7	14	Rutaceae	1	8	Saxifragaceae	1	1
Scrophulariaceae	6	6	Solanaceae	10	19	Theaceae	1	1
Tilliaceae	1	1	Tropaeolaceae	1	2	Umbelliferae	13	14
Urticaceae	3	5	Valerianaceae	1	1	Violaceae	1	1
Vitaceae	1	1	Misc. (unclassified)	1	2			

Totals	
Plant families	64
Genera	225
Species	361
Other	22
Total no. of entries	383

[a] Personal communication from P. B. Adams.

consistently were around the 100% mark when and where it occurs, there would most certainly be a greater effort to lessen its impact. If we could demonstrate more realistically and accurately the magnitude and frequency of loss, it is quite possible that we could generate greater support for research than now is possible.

Losses of some crops from disease caused by *Sclerotinia sclerotiorum* are shown in Table 2. If the data presented in Table 2 are accurate, millions of dollars are lost annually as a result of this fungus; ie, directly from loss of yield and indirectly from lessened quality (loss in grade). There are no data to illustrate the loss of expenditures for attempts to control diseases caused by *S. sclerotiorum* when attempts are either effective, or noneffective. There also is another form of loss caused by *S. sclerotiorum*, the production lost due to the abandonment of fields for growing preferred crops for less lucrative ones or to noncrop plants, weeds or fallow.

Almost all loss figures in the literature and in Table 2 are based on estimated ranges of disease incidence; apparently few accurate counts of diseased plants have been made. Beach (2) seems to be an exception because the data he presented are not the usual estimates. He reported measured losses from "lettuce drop" during a 3-yr period in Philadelphia County, Pennsylvania, as follows:

Year	Month	% Disease
1918	June	18
	October	75
1919	June	8
	October	17
1920	June	40
	October	17

These losses indeed, were, significant.

Poole (28) reported losses due to *S. sclerotiorum* in celery which were based on data obtained from several states. The data presented about Florida-grown celery illustrates the magnitude of the losses. In 1919, nine cars of celery were shipped and at the destination were examined for pink rot. Results were that one car had 50% pink rot, four cars had 20%, three cars had 5%, and the contents of one car apparently were unaffected.

TABLE 2. Crop production losses caused by *Sclerotinia sclerotiorum*[a]

Crop	Location	Crop loss because of disease	Dollar loss
Alfalfa	North Carolina	trace-9%/last 3 years	93,000 at 9% loss
	New Jersey	0.1% annual average	29,000
Bean-dry	Florida	5-10% annual average	48-50,000
and snapbean		6% loss in grade	...
	Michigan	1% annual average	1,000,000
	New York	1200 acres/year	600,000
	California[b]	0.5% in 1963	134,000
	U.S.[c]	3.5% annual (dry)	...
	U.S.[c]	2.0% annual (snapbean)	...
Celery	California[b]	2.5% in 1963	665,300
Lettuce	California[d]	5.0% annual average	185/ha
	New Jersey	10.0% annual average	450,000
	Orange County, NY	0.5% annual average	2,000
	Osewego, NY	25-30% annual	1,000,000
	California[b]	1.0 in 1963	...
	U.S.[c]	<1.0	...
Peanut	North Carolina	0.5% 3 year average	...
		2.0% in 1976	1,900,000
	Virginia	3.0% annual average	...
		5.0% in 1976	3,000,000
Potatoes	Florida	25-50% annual range	12-15,000,000
		40% loss in grade	4,000,000
	Washington	0.1-1.0% annual range	200,000-2,000,000
Spearmint	Washington	5% annual average	1,500,000
Sunflower	North Dakota	1.0% annual average	...
	U.S.	1.0% annual average	3,000,000
	New York	40 tons on 70 acres	...
Tomato	Florida	4-6% annual average	8-10,000,000
		5% loss in grade	2,500,000
Apricot	California[b]	1.0% in 1963	...
Cabbage	U.S.[c]	1.0% annual average	...
Eggplant	U.S.[c]	1.5% annual average	...

[a] Adams, P. B. 1978. (*Personal communication*).

[b] Ogawa, J. M., H. J. O'Reilly, A. D. Paulus, C. W. Nichols, D. H. Hall, and A. H. McCain. 1965. Estimates of crop losses and disease control costs in California, 1963. Univ. Calif. Agric. Expt. Stn. and Ext. Serv. 102 pp.

[c] Anonymous. 1965. Losses in Agriculture. U. S. Dep. Agric., Agr. Res. Serv., Agric. Handb. No. 291 120 pp.

[d] Macum, D. B., R. G. Grogan, and A. S. Greathead. 1977. Fungicide control of lettuce drop caused by *Sclerotinia sclerotiorum* 'minor'. Plant Dis. Rep. 61:555-559.

Other loss estimates from the literature support the trend established that disease incidence varies and the resulting crop loss also varies and is seemingly related directly to disease incidence, a high incidence produces a high loss. A total loss may be experienced for certain crops in certain years when "everything" is optimal. Fortunately, those optimal times do not come continuously.

INFERENCES

Publication of various types of information contributes to the development of knowledge or stimulates additional work about a particular subject. Such is the situation with *S. sclerotiorum*. Many excellent contributions have been published and many have stimulated individuals to seek additional knowledge. I doubt that a complete or total understanding of *S. sclerotiorum* with its many hosts in diverse environments will be forthcoming even in the distant future. Thus, it seems that any one contribution will not be the final answer, and that additional bits of new data will add knowledge in an evolutionary manner to the published information. To stimulate others to consider ones point of view brings reward, and their acceptance of ones conclusions is an even greater reward. Nonacceptance is an option that may result from a difference of opinion.

LITERATURE CITED

1. ADAMS, P. B., and C. J. TATE. 1975. Factors affecting lettuce drop caused by Sclerotinia sclerotiorum. Plant Dis. Rep. 59:140-143.
2. BEACH, W. S. 1921. The lettuce drop due to Sclerotinia minor. Pa. Agric. Exp. Stn. Bull. 165. 27 pp.
3. BEUTE, M. K., D. M. PORTER, and B. A. HADLEY. 1975. Sclerotinia blight of peanut in North Carolina and Virginia and its chemical control. Plant Dis. Rep. 59:697-701.
4. BUCHWALD, N. F., and P. NEERGAARD. 1973. A plea for the retention of Sclerotinia sclerotiorum as type species for the genus Sclerotinia Fckl. emend. Friesia 10:96-99.
5. BURGER, O. F. 1913. Lettuce drop. Fla. Agric. Exp. Stn. Bull. 116. 8 pp.
6. CHIVERS, A. H. 1929. A comparative study of Sclerotinia minor Jagger and Sclerotinia intermedia Ramsey in culture. Phytopathology 19:301-309.
7. COLEY-SMITH, J. R., and R. C. COOKE. 1971. Survival and germination of fungal sclerotia. Annu. Rev. Phytopathol. 9:65-92.
8. de BARY, A. 1884. Vergleichend Morphologie und Biologie der Pilze, Mycetozoen und Bacterien. (Comparative morphology and biology of the fungi, mycetozoa and bacteria. Translated by H. E. F. Garnsey, revised by I. B. Balfour, the Clarendon Press, Oxford. 1887). 525 pp.
9. DENNIS, R. W. G. 1956. A revision of the British Helotiaceae in the herbarium of the Royal Botanic Gardens, Kew, with notes on related European species. Commonw. Mycol. Inst., Mycological Pap. 62. 216 pp.
10. DENNIS, R. W. G. 1974. Whetzelinia Korf and Dumont, a superfluous name. Kew Bull. 29:89-91.
11. DICKSON, B. T. 1920. Sclerotinia wilt of greenhouse tomatoes. Phytopathology 10:500-501.
12. DIJKSTRA, J. 1966. Rotting by spread of mycelium from ascospore lesions of Sclerotinia trifoliorum. Neth. J. Plant Pathol. 72:279-283.
13. ERICKSSON, J. 1880. Om Klofverrotan med sarskildt afseende pa dess upptradande i vart land under aren 1878-79. Kgl. Landtbruks-Akad. Handl. Tidskr. 19:28-42.
14. FUCKEL, L. 1870. Symbolic mycologicae. Beitrage zur Kenntniss der Rheinischen Pilze. Jahrb. Nassauischen Vercins Naturk. 23-24:1-459.
15. GILBERT, A. H., and C. W. BENNETT. 1917. Sclerotinia trifoliorum, the cause of stem rot of clovers and alfalfa. Phytopathology 7:432-442.
16. HARTER, L. L., and W. J. ZAUMEYER. 1944. A monographic study of bean diseases and methods for their control. U. S. Dept. Agric. Tech. Bull. 868. 160 pp.
17. HELD, V. M., and C. M. HAENSELER. 1953. Cross-inoculation with New Jersey isolates of Sclerotinia sclerotiorum, S. minor, and S. trifoliorum. Plant Dis. Rep. 37:515-517.
18. JAGGER, I. C. 1913. The small lettuce Sclerotinia, an undescribed species. (Abstr.) Phytopathology 3:74.
19. JAGGER, I. C. 1920. Sclerotinia minor, n. sp., the cause of a decay of lettuce, celery, and other crops. J. Agric. Res. 20:331-334.
20. JAMALAINEN, E. A. 1954. Overwintering of cultivated plants under snow. FAO Plant Prot. Bull. 2:102-105.
21. JONES, E. S. 1923. Taxonomy of the Sclerotinia on Helianthus annus L. Phytopathology 13:496-500.
22. KEAY, M. A. 1939. A study of certain species of Sclerotinia. Ann. Appl. Biol. 26:227-246.
23. KORF, R. P., and K. P. DUMONT. 1972. Whetzelinia, a new generic name for Sclerotinia sclerotiorum and S. tuberosa. Mycologia 64:248-251.
24. LIBERT, M. A. 1837. Plante Crytogamicae arduennae (Exsiccati) No. 326. Published by M. A. Libert,
25. MORRIS, H. E., and D. B. SWINGLE. 1921. An important new disease of sunflower. Phytopathology 11:50.
26. MORDUE, J. E. M., and P. HOLLIDAY. 1976. Sclerotinia sclerotiorum. No. 513 in set 52 of Descriptions of Pathogenic Fungi and Bacteria. Commonw. Mycol. Inst., Kew, Surrey, England.
27. NEWTON, H. C., and L. SEQUEIRA. 1972. Ascospores as the primary infective propagule of Sclerotinia sclerotiorum in Wisconsin. Plant Dis. Rep. 56:798-802.
28. POOLE, R. F. 1922. The sclerotinia rot of celery. N. J. Agric. Exp. Stn. Bull. 359. 27 pp.
29. PRICE, K., and J. CALHOUN. 1975. Pathogenicity of isolates of Sclerotinia sclerotiorum (Lib.) de Bary to several hosts. Phytopathol. Z. 83:232-238.
30. PURDY, L. H. 1952. Infection of lettuce by Sclerotinia sclerotiorum (Lib.) de Bary. (Abstr.) Phytopathology 42:518.
31. PURDY, L. H. 1955. A broader concept of the species Sclerotinia sclerotiorum based on variability. Phytopathology 45:421-427.
32. PURDY, L. H., and R. BARDIN. 1953. Mode of infection of tomato plants by the ascospores of Sclerotinia sclerotiorum. Plant Dis. Rep. 37:361-362.
33. SMITH, C. O. 1916. Cottony rot of lemons in California. Calif. Agric. Exp. Stn. Bull. 265:235-258.
34. SMITH, R. E. 1900. Botrytis and Sclerotinia, their relationship to certain plant diseases and to each other. Bot. Gaz. 29:407-423.
35. SMITH, R. E. 1931. The life history of Sclerotinia sclerotiorum with reference to the green rot of apricot. Phytopathology 21:407-423.
36. STEVENS, F. L., and J. G. HALL. 1911. A serious lettuce disease (sclerotiniose) and a method of control. N. C. Agric. Exp. Stn. Tech. Bull. 8:85-145.
37. TAUBENHAUS, J. J., and W. N. EZEKIEL. 1931. A Sclerotinia limb blight of fig. Phytopathology 21:1195-1197.
38. VALLEAU, W. D., E. N. FERGUS, and L. HENSON. 1933. Resistance of red clovers to Sclerotinia trifoliorum Eriks., and infection studies. Ky. Agric. Exp. Stn. Bull. 341:113-132.
39. WADHAM, S. M. 1925. Observation of clover rot (Sclerotinia trifoliorum Eriks.). New Phytol. 24:50-56.
40. WAKEFIELD, E. N. 1924. On the names Sclerotinia sclerotiorum (Lib.) Massee, and S. libertiana Fuckel. Phytopathology 14:126-127.
41. WILLIAMS, G. H., and J. H . WESTERN. 1965. The biology of Sclerotinia trifoliorum Erikss. and other species of sclerotium forming fungi. I. Apothecium formation from sclerotia. Ann. Appl. Biol. 56:253-260.
42. WOLF, F. A., and R. O. CROMWELL. 1919. Clover stem rot. N. C. Agric. Exp. Stn. Tech. Bull. 16. 22 pp.
43. WONG, A. L., and J. H. WILLETTS. 1975. A taxonomic study of Sclerotinia sclerotiorum and related species: Mycelial interactions. J. Gen. Microbiol. 88:339-344.