Epidemiology of Diseases Caused by *Sclerotinia* Species

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Species of the genus *Sclerotinia* can function either as soilborne or airborne pathogens. Infections of aboveground plant parts result from ascosporic inoculum, whereas soil-line infection may result either from ascospores or sclerotia. Belowground infection, however, result from mycelial germination of soilborne sclerotia. Accordingly, the epidemiology of these two types of infections incited by *Sclerotinia* spp. is quite different and the effects of weather factors on their incidence and development differs considerably.

We propose that, generally, the large-sclerotia-type isolates of *Sclerotinia* (represented by *S. sclerotiorum* and *S. trifoliorum* as defined by L. Kohn in this symposium) function primarily by producing apothecia, and that mycelial germination from sclerotia contributes minimally, if at all, to the development of epidemics. In contrast, infections incited by the small-sclerotial-type isolates (*S. minor*) originate primarily through the eruptive mycelial germination of sclerotia, whereas production of apothecia under natural conditions occurs very rarely and thus is of minor importance. The different modes of infection exhibited by the large- and small-sclerotia-type isolates of *Sclerotinia* probably results from continued adaptation of these species to their ecological niches.

Although many reports that deal with diseases incited by *Sclerotinia* spp. have been published only a limited amount of detailed epidemiological information is available on a few important diseases. Furthermore, quantitative epidemiological data are essentially lacking. Because more information is available on white mold of beans incited by *S. sclerotiorum* and drop of lettuce caused by *S. minor*, these two diseases will be used to illustrate the distinct differences in the epidemiology of diseases incited by *Sclerotinia* spp. that infect from ascorsporic inoculum or by eruptive mycelial germination of sclerotia. The epidemiology of other important diseases incited by *Sclerotinia* spp. cannot be covered in this short presentation; nevertheless, we believe that their epidemiological patterns will closely resemble either that of white mold of beans or lettuce drop.

Most infection of beans occur on aboveground parts, and it seems unlikely that the large sclerotia per se of *S. sclerotiorum* are an important form of inoculum; they function essentially to produce airborne ascospores (2,12,30,31,35). Lettuce drop can be incited by either *S. sclerotiorum* or *S. minor* (24,25,28); these two species may occur together in the same lettuce field. However, *S. minor* usually is the predominate species on lettuce (7,24,25). Infection of lettuce with *S. sclerotiorum* almost always occurs at the ground level because it usually originates from ascosporic infection of senescent lower leaves. In contrast, infection with *S. minor* can occur either at the soil line through senescent lower leaves or below ground as deep as 10 cm through root and stem tissues. Production of apothecia, by at least some isolates of *S. minor*, has been reported (9,23) but there is only one report of their natural occurrence (18). Thus, the role of ascospores in lettuce drop epidemics caused by *S. minor* appears to be of minor importance. If involved, ascospores of *S. minor* probably would infect senescent lower leaves as does *S. sclerotiorum*.

This paper will attempt to: summarize the epidemiology of white mold of beans and lettuce drop diseases incited by *S. sclerotiorum* and *S. minor*, respectively, and discuss the use of epidemiological data in disease management.

EPIDEMIOLOGY OF WHITE MOLD OF BEANS

Source of inoculum. White mold epidemics of beans are initiated by ascospores produced by sclerotia of *S. sclerotiorum* (2,3,12,30,31,35). Only sclerotia in the top 2–3 cm of the soil are functional because apothecia with stipes longer than 3 cm rarely are produced under field conditions. Sclerotia present in and outside bean fields also can provide ascosporic inoculum for bean white mold epidemics. In New York, for example, ascosporic inoculum originates mainly from sclerotia outside of bean fields (2). Sclerotia often are found producing apothecia around the base of dandelion

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plants and wild clover, or near other host plants in hedge rows, uncultivated wooded areas, and fruit orchards. In dryer areas such as Nebraska and California, apothecia are produced in bean fields that are sprinkler- or furrow-irrigated (12,31). Under these conditions, water required for sclerotial germination is provided by irrigation, but apothecial production is nil prior to the development of a dense canopy of plants over the soil that decreases evaporation of water from the soil surface. As a consequence, low moisture tensions requisite for sclerotial germination are maintained long enough for apothecial production. In such areas, sclerotia may be distributed within and between fields in irrigation water (31).

Mycelium from sclerotia has been reported to infect beans (27); however, recent work (2) suggests that this occurs rarely under natural conditions. We observed that sclerotia placed in direct contact with bean tissues failed to infect, even after prolonged incubation under near optimum conditions. Instead of infecting directly by production of mycelium, the sclerotia produced an average of more than three apothecia per sclerotium and because a nonliving food base was not available, infection did not occur. Preconditioned (functionally mature) sclerotia of *S. sclerotiorum* usually produce very sparse mycelial growth when utilizing their own reserve energy. Such mycelium can infect beans only when a readily available energy source is present in direct contact between the sclerotium and bean tissues.

Requirements for production of ascosporic inoculum. Only preconditioned and functionally mature sclerotia are capable of producing ascosporic inoculum for white mold epidemics. The time required for preconditioning sclerotia varies for different isolates and optimum conditions required for preconditioning have not been determined precisely. In general, however, newly formed sclerotia require holding for various periods of time under cool moist conditions before attaining capability for carpogenic germination. Several factors are known to influence carpogenic germination of preconditioned sclerotia of *S. sclerotiorum* (11); however, prolonged high soil moisture is the most common limiting factor (2,11,15). Continuous moisture for about 10 days is required for apothecial development, and even a slight moisture tension (osmotic or matric or a combination of both) prevents apothecial formation. Field-collected sclerotia capable of carpogenic germination when sampled earlier, after exposure to extreme drying conditions on the soil surface of fields in New York, failed to produce apothecia when placed under near-ideal conditions in a growth chamber for as long as 3 mo. Thus, exposure to extreme drying and possibly high temperature had a prolonged detrimental effect on apothecial production; however, the sclerotia remained viable as indicated by consistent mycelial production on nutrient media. It was reported recently (14) that the optimum water potential for apothecium production by bean isolates from New York and Nebraska was −80 to −160 mb and −240 mb, respectively. This difference may reflect the relative adaptation of the isolates of *S. sclerotiorum* from Nebraska to reduced soil moisture tension. These data also indicate that maintaining soil water at near field capacity (−300 mb) for long periods of time is essential for carpogenic germination. Data on the effect of fluctuating soil water potential on apothecial production by sclerotia is not available. Soil water content, especially in the top 2–3 cm of soil, varies considerably and is affected by weather parameters such as relative humidity (RH), wind velocity, type and extent of plant canopy, and temperature.

Temperature also exerts a significant effect on apothecial production by *S. sclerotiorum* (2,11,30). Preconditioned sclerotia incubated in water at different constant temperatures produced the most initials and mature apothecia at 11 C; however, production at 15 C also was quite good. No apothecial initials were produced at either 30 or 5 C after 21 days of incubation. However, it is unlikely that temperature would be a limiting factor in most bean-producing areas of the USA. Many years of weather records in New York, for example, show that temperatures during the growing season are seldom too high or low for apothecial production. It is likely that moisture tension and temperature interact to affect apothecial production. Also both temperature and moisture tension fluctuates in nature, but most tests thus far have been done with constant moisture or temperature and combination affects have not been determined.

Dispersal and survival of ascospores. We believe that, generally, apothecia are produced and ascospores of *S. sclerotiorum* are discharged throughout the growing season if the moisture of the top 2–3 cm of soil is maintained near saturation. Under New York conditions, mature apothecia have been found as early as 20 April, provided that the snow has melted and soil temperature has reached 10 C for at least part of the day. When moisture is not a limiting factor, apothecia are produced throughout the bean-growing season in New York which begins with plantings in the first week of May and continues until harvest in mid-September (G. S. Abawi, *unpublished*).

Only limited information is available concerning the liberation, transport, and deposition of ascospores of *S. sclerotiorum* (35–37). It seems likely that ascospore liberation and transport in *Sclerotinia* spp. closely resembles that reported for other discomycetes (20,22). Each day, when subjected to a slight decrease in moisture tension, the mature asci forcibly discharge ascospores into the air to a distance of more than 1 cm. This height of discharge enables the ascospores to escape the still layer of air near the soil surface and to reach the more turbulent aboveground layers. It has been reported that the longest dispersal of ascospores of *S. sclerotiorum* is 25 m (35), several hundred meters (36), and several kilometers (10). Dispersal to the latter distances, in our opinion, is possible and probably not uncommon, but we have no substantiating data. It has been estimated that ascospore production by a single apothecium may be as high as 3×10^7 ascospores and a single sclerotium may produce 2.3×10^8 ascospores (31).

A mucilaginous material is discharged along with the ascospores that can cement the spores to host tissues or other objects encountered during flight. Ascospores deposited on bean tissues need not infect immediately, but can survive for a considerable time until the wet conditions and exogenous energy sources required for infection become available (15). Under laboratory conditions, the thin-walled ascospores of *S. sclerotiorum* survived for 21 days at 7% RH, but survival was less than 5 days at 100% RH. Ascospores atomized onto bean leaves under field conditions survived for as long as 12 days. However, the effect of fluctuating water potentials, temperature, and other weather elements on survival of ascospores needs further evaluation.

Requirements for host infection and disease development. Epidemics of white mold of beans occur only after flowering; however, a few infected plants have been observed occasionally in fields prior to blossoming. Ascospores of *S. sclerotiorum* require an exogenous energy source to infect healthy bean plants (2,3,12,29). Under field conditions, mature bean blossoms usually serve as an energy source. However, ascospores readily infected mechanically injured plants and also nonflowering beans with genetically induced necrosis or with necrotic lesions incited by other plant pathogens (5). This may explain the occasional occurrence of white mold prior to blossoming. Ascospores completely colonize mature and senescent blossoms within 2–3 days (3) and mycelial growth from these colonized blossoms produces infection by contact with leaf, stem, and pod tissues.

Numerous reports have stressed the importance of moisture in the development of white mold of beans (2,12,15,26,27,31). Infection of beans by *S. sclerotiorum* occurs only if free moisture is maintained for a relatively long period at the interface of bean tissues and the inoculum (2). Approximately 48-72 hr of continuous leaf wetness are required for infection by ascospores. Similarly, 16–24 hr and over 72 hr of leaf wetness are required for the infection of beans by moist, infected bean blossoms with actively growing mycelium and dry colonized bean blossoms, respectively. Furthermore, high relative humidity, even near 100%, is not sufficient for lesion initiation. For example, infection of detached leaves enclosed in plastic boxes with free water in the bottom usually fails unless the leaves are sprayed periodically with water. In addition, expansion of lesions also requires free moisture; lesion enlargment is stopped abruptly if the surface of infected tissues (except bulky stem tissues) becomes dry. However, the dry lesions can resume expansion when free water becomes available. Field inoculation studies and observations of white mold epidemics in New York and elsewhere also have substantiated the key role of moisture. These studies indicated that the duration of leaf wetness and frequency of rainfall or irrigation when inoculum is available is far more important than the total amount of water received (rainfall or irrigation) (2,31). Also, white mold of beans has been reported to be more prevalent in fields with heavy vegetative growth and in areas where air circulation is limited, such as low-lying fields and particularly those surrounded by uncultivated wooded areas (16,27). These conditions and locations probably exert an effect by increasing the duration of leaf wetness.

Ascospore germination and growth as well as lesion initiation and development are near optimum at constant temperatures of 20–25 C (2,37). Furthermore, lesions do not develop on inoculated leaves of plants incubated at 5 or 30 C. However, temperature per se (macroclimate data) does not appear to be a limiting factor in the development of white mold under New York conditions. However, until the temperature in the bean microclimate is studied, this conclusion must remain tentative. In the irrigated and hotter regions where beans are grown, higher temperatures may in some instances limit white mold incidence and development.

Role and fate of inoculum produced by primary infections. Under moist conditions, leaf, stem, and pod tissues in contact with infected blossom parts develop water-soaked lesions. These lesions continue to enlarge and within a few days become covered with a dense, white mycelial mat. Usually, numerous sclerotia are produced on the surface of the mycelium within 7–10 days. The fungus continues to grow and may invade the whole plant aboveground. Also under a dense canopy of foliage, fallen leaves, blossoms, and other plant parts become infected, with the result that mycelium grows over the soil surface and from infected plant parts aboveground.

Since asexual spores are not produced by *S. sclerotiorum*, plant-to-plant infection occurs only through direct hyphal growth from previously infected tissues. Newly produced sclerotia are capable only of limited hyphal growth unless provided with an exogenous food base and usually exhibit dormancy for carpogenic germination (2,11,12,30). Secondary spread of *S. sclerotiorum* on snap beans occurs only to a limited extent, and we believe that it plays a minor role in the development of epidemics. However, secondary spread by plant-to-plant mycelial growth may be more important on dry beans as a result of the longer period of susceptibility due to indeterminate flowering and the moist-chamber effect produced by the dense canopy of foliage. Mathematical analyses of white mold epidemics are not available at the present time; however, white mold appears to be a simple-interest disease as defined by Van der Plank (38).

Ecology of sclerotia and factors that affect their survival are covered in another paper of this symposium. However, it is interesting to note here that work in Nebraska has shown that the soil population of sclerotia did not increase even in fields where annual epidemics of white mold had occurred (31). Similarly, the soil population of sclerotia in a bean field in New York remained about the same even after three consecutive years of severe epidemics of white mold that resulted in complete loss of the crop each year (G. S. Abawi, *unpublished*). In contrast, it has been reported (31) that a 3-yr rotation with a nonhost crop did not significantly reduce the population of sclerotia. Thus, it appears that most sclerotia are ephemeral, but some are long-lived. These sclerotia that are persistent plus the availability of primary inoculum from outside bean fields seems to explain why no correlation has been found between white mold incidence and severity, and the previous cropping history of bean fields (16,27).

EPIDEMIOLOGY OF LETTUCE DROP CAUSED BY *S. MINOR*

In areas where both *S. minor* and *S. sclerotiorum* occur, drop of lettuce may be caused by either species and both may occur together in the same field. Usually, however, the occurrence of *S. sclerotiorum* is more sporadic; it may not be active one year, and yet may produce a widespread epidemic during the following year. In contrast, *S. minor* occurs more consistently year after year in infested fields and in some areas of New York and California that we have observed, drop in most years is caused almost entirely by *S. minor*. The literature concerning *S. sclerotiorum* and *S. minor* points out clear-cut differences in their epidemiology that seems to explain this differential behavior.

The available evidence indicates that the epidemiology of lettuce drop caused by *S. sclerotiorum* is similar to that of white mold of bean. Development of both diseases is influenced by factors involved with production of and infection by ascospores. Thus, this section is concerned primarily with the epidemiology of lettuce drop caused by *S. minor* and the comparison of it with that of *S. sclerotiorum*.

History of *Sclerotinia minor*. Drop of lettuce in Massachusetts caused by a small-sclerotia type of *Sclerotinia* was described by R. E. Smith in 1900 (32). Because of failure to induce apothecial production, he concluded that the fungus was a "degenerative form of *S. libertiana* that had become highly specialized as a vegetative parasite, able to renew growth in a vegetative manner by the direct production of mycelium." He reported also that the small sclerotia, when first formed, make no growth (remain dormant), but "after a period of rest and dryness, they send out mold-like growth which attacks plants. Its method of reproduction and spreading is strictly limited to soil." In a later publication, Stone and Smith (34) reported that covering the surface of soil with a few inches of sterilized soil completely controlled drop, but that drying of infested soil during August, September, and October resulted in a marked increase in the incidence of drop in the next crop.

In 1913, Jagger (23) reported lettuce drop in New York that was caused by a similar small sclerotial type of *Sclerotinia*. He was able to induce apothecial production in vitro and described a new species, *S. minor*. Beach (9) in 1918 reported the occurrence of lettuce drop caused by *S. minor* in Pennsylvania. He also induced the fungus to form apothecia, but did not find naturally occurring apothecia despite an extensive search during May, June, and July when apothecia of *S. libertiana* (= *S. sclerotiorum*) were abundant. Because of the apparent lack of natural apothecia of *S. minor*, Beach (9) tested dried sclerotia from infected lettuce tissues and found that when they were soaked in water and placed on lettuce stems, new mycelium developed within a few days. Thus, he agreed with Smith (32) that sclerotia of *S. minor* can renew growth vegetatively by direct production of mycelium after dormancy ends. Neither Smith (32) nor Beach (9) made definite statements whether mycelium from germinating sclerotia require a food base for infection of undamaged tissue. However, Beach (9) reported infection of lettuce heads that resulted from sclerotia buried in natural soil which suggests that a food base was not required. Beach (9) observed drop caused by both *S. minor* and *S. sclerotiorum* and noted the following differences in their characteristics and behavior: (i) *S. minor* produces smaller, but many more, sclerotia than does *S. sclerotiorum*. (ii) Cultivation or other conditions that reduce the duration of soil dampness prevent the production of apothecia by *S. sclerotiorum*, but do not prevent the vegetative growth of *S. minor* (thus, drop caused by *S. minor* can occur after a rainy period of too short duration for production of apothecia by *S. sclerotiorum*). (iii) S. minor tends to be more localized in distribution and spreads slower, but recurs more consistently year after year in infested fields. In contrast, *S. sclerotiorum* may occur with wide distribution in fields where very little or none had occurred in the previous year; the occurrence of *S. sclerotiorum* is associated with prolonged wet weather that is favorable for production of apothecia. (iv) Incidence of drop caused by *S. minor* is increased if infested soils become dry prior to planting. Recent literature (6–8,17–19,24,25) concerning the comparative epidemiology of lettuce drop caused by *S. minor* or *S. sclerotiorum* generally is in good agreement with Beach's conclusions that were published in 1918 (9). For example, Adams and Tate (7) showed that drop was increased by allowing soil moisture to fluctuate (and thus to dry) and that the number of infected plants was correlated with numbers of sclerotia added to the soil, but no apothecia had been produced during the incubation period. Thus, infection was due to mycelial germination of sclerotia. However, the numbers of sclerotia required to produce a high level of infection (500 sclerotia per 100 gm soil) seems excessively high and probably is because most of them had not overcome dormancy and did not germinate by eruptive mycelium production. This supposition is supported by a subsequent publication (8) in which the same authors showed a near-perfect correlation between numbers of germinable sclerotia and number of infected plants, which indicates that a single competent sclerotium is capable of causing infection if placed near susceptible tissues. Adams and Tate (7) reported that over 90% of all infections originate from sclerotia within the top 2 cm of soil, and Marcum et al (25) reported that about half of all infections originate from belowground sclerotia located in the upper 10 cm of soil. Thus, these reports are in general agreement with the report of Stone and Smith (34) that covering the soil surface with a few inches of sterile soil resulted in control of drop. It is not known, however, whether this is attributable to restricted spread of below ground lesions or failure of sclerotia to germinate at greater depths in the soil. Presumably plants that are infected on lower portions of the root could survive if spread of the lesions was restricted.

Source and production of inoculum. Two types of asexual (vegetative) germination, hyphal or mycelial, have been described for sclerotia of *S. minor* (8). Hyphal germination is characterized by the production of a few short hyphal strands that grow very little without an exogenous food base; hyphal germination of the large sclerotial isolates of *S. sclerotiorum* is similar. This type of inoculum will infect lettuce only through prior colonization of exogenous energy sources (8). In contrast, eruptive mycelial germination of sclerotia appears first as bulges in the sclerotial rind which eventually ruptures and exposes a massive and dense mycelium that utilizes stored food reserves in the sclerotium for growth as does *S. sclerotiorum* during carpogenic germination. The sclerotia of *S. minor* have a dormancy period prior to mycelial germination and the length of this dormancy varies among isolates and is affected by media composition, time after formation, drying, and other undetermined factors. Sclerotia that undergo mycelial germination are capable of infecting lettuce directly without the need for an exogenous energy source; thus the infective propagule for lettuce drop caused by *S. minor* is the sclerotium per se instead of ascospores as it is with *S. sclerotiorum*.

Production of apothecia by sclerotia of *S. minor* has been reported (9,18,23) and the role of ascospores in initiation of lettuce drop caused by *S. minor* has been suggested (24). However, a subsequent report (18) indicated that ascospores of *S. minor* generally play a minor role in the epidemic occurrence of lettuce drop caused by *S. minor*. In our opinion, if ascospores of *S. minor* were produced, they probably would infect lettuce in a similar way and would have similar infection requirements as ascospores of *S. sclerotiorum* on beans. In support of this, Beach (9) failed to induce infection with ascospores of *S. minor* and concluded that they require a food base for infection.

Inoculum density, dispersal, and survival. Under greenhouse conditions, a close correlation was reported between the incidence of lettuce drop and inoculum density of sclerotia of *S. minor* (7). An inoculum density of two to seven sclerotia per 100 g of soil caused about 10% lettuce drop. The incidence of lettuce drop was about 20 and 80% at inoculum densities of 31 and 250 sclerotia per 100 g of soil, respectively. The sclerotial populations of lettuce field soils in New Jersey varied from 0 to 20 and averaged 3.9 sclerotia per 100 g soil. Inasmuch as loss due to lettuce drop in New Jersey was estimated at 10% annually, it was concluded that field data substantiated the inoculum density data obtained under greenhouse conditions. However, these data are difficult to interpret because the percentage of sclerotia capable of eruptive mycelial germination was not reported. In a subsequent publication (8), the percent of infection was shown to coincide almost exactly with the percent of mycelial germination which indicates that each competent sclerotium, if in a location close to plant tissues, can incite an infection.

Many factors are known to affect survival and germination of sclerotia in soil (11). This will be discussed further in another paper in this symposium. However, survival of sclerotia varies greatly among different soils and at different soil moisture tensions (1,6). Lettuce planted in some soils consistently has little or no drop despite continuous cropping. The reason for the "suppressive" nature of such soils has not been determined.

Inoculum of lettuce drop is sedentary and spread between fields is slow and restricted. Belowground infection of roots and ground-level infection of senescent leaves generally occurs when leaves and roots are in direct contact or are only a few millimeters away from germinating sclerotia. It has been suggested that hyphal webs of *S. minor* and infected debris may become airborne and spread within and between fields (24). However, we believe that such spread is unlikely to play a significant role in the epidemiology of lettuce drop. Nevertheless, mycelial growth from plant to plant may occur, and sclerotia may spread within and between fields in irrigation water, machinery, etc.

Requirements for host infection and disease development. Infection of lettuce by *S. minor* results from mycelial germination of sclerotia (8,17,25); thus, inoculum density of germinable sclerotia in soil and the prevalence of conditions that favor sclerotial germination influence the incidence of lettuce drop. Lettuce drop is most severe when cool and moist weather conditions prevail (10,26) or irrigation is excessive during the growing season and especially near harvest time (D. B. Marcum and R. G. Grogan, *unpublished*). In addition, lettuce drop is more prevalent in low and poorly drained areas of the field. However, the incidence of lettuce drop was greater when soil moisture was allowed to fluctuate from 100 to 30% field capacity as compared to 100 to 80–90% field capacity (7). It was suggested that drying of sclerotia at or near the soil surface stimulates germination and infection when soil moisture is adjusted again to near field capacity. This observation agrees with reports by Smith (32) and Beach (9) that incidence of drop caused by *S. minor* was greatly increased in crops grown in infested soil that had been allowed to become dry prior to planting. Recent work in our laboratory showed that, although sclerotial germination is best at soil moisture near field capacity (−300 mb), considerable germination occurred at a soil water potential of −2 bars. Results of one experiment showed, for example, that at soil water potentials of 0, −0.05, −0.2, −1.0, −2.0, and −5.0 bars, sclerotial germination was 5, 95, 65, 36, 9, and 2%, respectively (G. S. Abawi, R. G. Grogan, and J. Duniway, *unpublished*). Nevertheless, free moisture still may be required for successful host infection as is the case with *S. sclerotiorum* on beans. It was demonstrated (17) under field conditions that significantly less drop occurred on a lettuce cultivar with an upright growth habit (Cos) than on the cultivars Buttercrunch, and Great Lakes. The lower leaves of the latter two cultivars are close to or in contact with the soil. All three cultivars are equally susceptible to infection by *S. minor* under greenhouse conditions when artificially inoculated. Thus, it was concluded that the lower leaves of both Buttercrunch and Great Lakes, in addition to being in contact with the soil, modify the soil microclimate by making it wetter and cooler and, thus, more inducive to sclerotial germination. In fact, germination of sclerotia was observed only a few centimeters from the base of the plant and only under the lower leaves (17). Thus, the escape of Cos lettuce under field conditions probably is due to reduced soil moisture and possibly to higher temperatures in the microclimate. Detailed information on the effect of temperature on incidence and severity of lettuce drop is not available. However, *Sclerotinia* spp. generally are favored by low temperatures with a favorable infection temperature of about 10–25 C.

Although infection of lettuce by *S. minor* may occur at any time during the growing season, most infection becomes evident by death of plants (drop) after head formation and as the crop approaches maturity (7,17,25). Mycelia produced by germinating sclerotia infect lower leaves and crown tissues and death usually follows within 1 wk. Similarly, belowground infections of the main root result in plant death within 7–14 days. However, infection originating on secondary roots progress more slowly and a 3-wk or longer incubation period may be required for symptom expression and plant death (D. B. Marcum and R. G. Grogan, *unpublished*).

Disease progress curves of lettuce drop incidence were determined statistically, analyzed for secondary spread, and plant-to-plant spread in nine lettuce plantings in New Zealand (24). Although the results were inconclusive and quite variable, it was suggested that secondary spread sensu Van der Plank (38) had occurred in addition to plant-to-plant spread. This suggestion was based on the significant occurrence of several adjacent infected plants along the rows. It also was postulated that primary infections are initiated by ascospores, whereas secondary infection are associated with mycelial inocula. Later work (17,19), however, suggested that ascospores are not likely to be the main source of inoculum in New Zealand. Additional research on the epidemiology of lettuce drop is needed to clarify the pattern of disease incidence and development. However, in our opinion, even though plant-to-plant spread may occur, it plays a minor role in the epidemiology of lettuce drop. Further research probably will show that lettuce drop is a simple interest disease sensu Van der Plank (38). Infections near the crown as compared with belowground infections away from the main root may be partially responsible for the variation in time for the appearance of lettuce drop under field conditions.

Role and fate of inoculum produced by primary infections. Under moist conditions, infected leaf and crown tissues become covered with the cottony mycelium of *S. minor*, especially near the soil line. Numerous sclerotia then are produced on the mycelial mat. Sclerotia are the main surviving structure as the mycelium probably is short-lived in soil. The newly produced sclerotia, after undergoing a dormancy period (8) must also undergo drying before they will germinate (D. B. Marcum and R. G. Grogan, *unpublished*). Functionally mature sclerotia in soil may germinate and produce infection or they may produce secondary sclerotia (6,9). However, the factors that favor or inhibit the production of secondary sclerotia in natural soil and their importance in epidemiology of lettuce drop are not known. Other factors that will be discussed in another paper of this symposium are known to influence survival and germination of sclerotia.

USE OF EPIDEMIOLOGICAL DATA IN DISEASE MANAGEMENT STRATEGIES

Detailed and quantitative epidemiological data are essential for the development of effective and economical control programs for diseases caused by *Sclerotinia* spp. Epidemiological data presently available on both white mold of beans and drop of lettuce, although not complete, have contributed significantly to the control of these important diseases and also to the development of integrated control strategies. Information on the source, nature, and availability of the primary source of inoculum and factors required for infection has improved timing of and application procedures for fungicide sprays both on lettuce and beans (21,25). Also, an effective procedure for screening for resistance to white mold of bean was developed using this information (4). Data on the critical role of moisture in the microclimate suggest that certain cultural practices as well as the genetic modification of plant architecture can be utilized to reduce disease incidence and development of both white mold and drop (13,33). Information on the role of moisture and other weather factors on the production of ascosporic inoculum and infection of beans are presently being used to forecast white mold in New York and Nebraska. Capability for forecasting white mold occurrence will help to determine the need, if any, for application of a fungicidal spray on beans and thus may reduce the cost of production and minimize pollution of the environment. Data on the mode of germination and inoculum density of sclerotia of *S. minor* in relation to the incidence of lettuce drop are essential for the development of a pest-management program for this disease (7). Soil indexing for viable sclerotia may allow the prediction of the potential for lettuce drop in a field and also will aid in determining the effect of cropping sequence, cultural practices, biological control strategies, etc on survival of sclerotia in soil. It appears, however, that data on numbers of sclerotia of *S. minor* must pertain to sclerotia that are competent to germinate by eruptive mycelial growth because this is the infecting propagule. Numbers of viable sclerotia as determined by ability to grow on nutrient media often correlate poorly with percent infection because sclerotia capable of only weak hyphal germination do not infect unless a nonliving food base is available.

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