Survival of Whetzelinia sclerotiorum and Initial Infection of Dry Edible Beans in Western Nebraska

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ABSTRACT

Sclerotia were the primary survival structures of *Whetzelinia sclerotiorum* in the North Platte Valley of western Nebraska. The fungus also overwintered as mycelium in bean seeds, but this was not an important source of initial inoculum. Nearly 75% of the sclerotia recovered after 3 years' burial at 5, 12.5, and 20 cm below the soil surface germinated and formed apothecia in culture. The majority of sclerotia that formed apothecial initials in soil were recovered at 5- and 10-cm depths. Sclerotial survival was not always correlated with carpogenic germination, but was adversely affected by high soil temperature and moisture. The capacity of sclerotia to form secondary sclerotia and their longevity in soil insures that inoculum will be present in a 3-year crop rotation. Ascospores were the primary source of initial inoculum and infected beans in the field and greenhouse after germination and colonization of senescent flowers, adhering to or in contact with other plant organs. Mycelium from sclerotia initiated less than 10% of the initial infections. Bean plants became infected when exposed to mature apothecia or ascospore suspensions. Initial infection in the field was correlated with senescence of flowers and apothecial production.

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White mold disease of dry edible beans *(Phaseolus vulgaris* L.) caused by the fungus *Whetzelinia sclerotiorum* (Lib.) Korf and Dumont [= *Sclerotinia sclerotiorum* (Lib.) de Bary (7)] has been present in western Nebraska for at least 20 years, but has not been considered economically important until recently. Presently, the disease is the most important problem in the North Platte Valley. The sporadic appearance and development of the disease in Nebraska, combined with erratic success of protective chemicals and lack of resistant varieties (15), has made control very difficult. Current control measures in Nebraska are based, to a great extent, on the studies of white mold on green beans (4, 5, 11, 20). Since there has not been an extensive investigation of the epidemiology of white mold of dry edible beans in Nebraska, studies were begun. The purpose of this study was to determine the mode and longevity of survival of the pathogen and the mode of primary infection on bean cultivar Great Northern.

MATERIALS AND METHODS.—*Laboratory trials.*—The effects of soil moisture and temperature on sclerotial survival were determined in the laboratory for sclerotia from two sources. 'Field' sclerotia were separated by hand from cull pile screenings obtained from bean processing plants in the North Platte Valley and stored at 22 C. 'Culture' sclerotia from three bean isolates of *W. sclerotiorum* were removed from 3-week-old potato dextrose agar (PDA) cultures, air-dried for 1 day at 22 C, and stored overnight at 5 C before placement in the soil. Small samples of all sclerotia were tested for mycelial

germination on PDA and for apothecial germination on 2% water agar (WA).

A moisture release curve for Tripp very fine sandy loam soil was determined using the techniques of Richards (12) and Veihmeyer and Hendrickson (18). From this curve, percentage of water (PW) for 15 bar (8.4), 1/3 bar (17.7), and near-saturated soil (36.4) was obtained. Appropriate amounts of water were added to each 538-g soil sample in a 15-× 38-cm plastic bag and mixed thoroughly. Each sample in its plastic bag was placed in a paper cup container and 25 'culture' or 'field' sclerotia were placed either on or 5 cm below the soil surface. A 5-cm length of Tygon tubing (ID = 0.24 cm) was inserted halfway into the open end of the plastic bag and secured. The tubing minimized evaporation while permitting air exchange. Containers were randomly placed in either a 5- or 27-C constant temperature incubator. Four containers representing each sclerotial depth, soil PW, and temperature were removed at 1-, 2-, and 3-month intervals. Soil also was removed from each container at these times, weighed, oven dried, and reweighed.

Sclerotia were recovered by washing soil from each container through a 0.85-mm (20-mesh) sieve with running tap water. Sclerotia were wrapped in cheesecloth and washed under running tap water for 12 hours. After drying, the sclerotia were placed on WA plates and incubated at 22 C for 2-3 weeks.

Field trials.—Survival of sclerotia also was studied in the field. Sacks to hold sclerotia were made by tying one end of an 8-cm length of nylon mesh hose (women's stocking) with monofilament. To each sack was added 100 'field' sclerotia and 60 to 80 g of soil from the appropriate depth. The open end of the sack was closed with monofilament and the sclerotia and soil were thoroughly mixed. These sacks were placed 61 cm apart at 0, 5, 10, 20, and 30 cm below the soil surface in a field previously cropped to dry beans and were removed 7, 11, and 17 months after burial. On each sampling date, four replications (initially totaling 400 sclerotia) were removed from each depth. After the sacks and their contents were soaked in water for 5 to 10 minutes, sclerotia were recovered from soil as outlined in the laboratory trial.

Isolations from plant residue and soil.—Soil and plant residue collected from fields with a previous history of white mold were examined for sclerotia and other propagules of *W. sclerotiorum*. Sclerotia were isolated by passing 1.0-kg soil samples through a 0.85-mm sieve and then incubating the collected debris from the sieve on WA. Plant debris was separated from samples by passing 1.5 to 3 kg of soil from each field through 0.85- and 0.25-mm (60-mesh) sieves. Debris collected from each sieve was washed under running tap water for 30 minutes, air-dried, and incubated at 22 C for 2 weeks on Hawthorne's semi-selective agar medium (HA) (B. T. Hawthorne, *personal communication).* HA contained cornmeal agar amended with 50 μg/ml streptomycin, 2 μg/ml chlorotetracycline and 50 μg/ml pentachloronitrobenzene. To detect fungal propagules other than sclerotia, 50-g soil samples were diluted 1:1,000 in 0.1% water agar, and 1.0-ml aliquots were incubated on HA for 2 weeks and examined for *W. sclerotiorum*.

Ascospore inoculations.—Ascospores were collected from two sources by the Millipore filter technique (14). Sclerotia collected from white mold-infected bean fields or PDA plates of *W. sclerotiorum* were buried in soil at a depth of 5 cm. After 8 weeks, sclerotia were removed from the soil, washed, and incubated on WA for 2 weeks. At this time mature apothecia had formed. Apothecia produced in a greenhouse soil bench were also used as a source of ascospores.

Young and senescent flowers, leaves, and cotyledons were collected from Great Northern (GN) bean plants, placed in petri dish humidity chambers, and immediately inoculated with ascospore suspensions. Humidity chambers were made by placing two pieces of Whatman No. 1 filter paper (9-cm diameter) in each petri dish, autoclaving, and saturating the filter with sterile water. Ascospore inoculum was prepared by backwashing filter paper from Millipore collections with distilled water, and adjusting the resultant suspension to a standard concentration of 4,400 ascospores per ml. Ascospore germination was determined on WA. Inoculations were made by placing 1.0 ml of ascospore suspension on one-half of the appropriate plant organ and 1 ml of water on the other half. A sample of the inoculum suspension was removed from the organs 2 days after inoculation and observed for ascospore germination. The inoculated plant organs were incubated at 22 C for 2 weeks and observed for white mold development. Isolations of *W. sclerotiorum* were made from organs exhibiting white mold symptoms.

Greenhouse infection was studied in a 2.1 × 2.1-m section of bench containing soil infested with sclerotia of *W. sclerotiorum* and planted with four rows of GN beans. After apothecia appeared in the soil bench, pots containing two or three 7-week-old GN #59 bean plants (midbloom) were placed between the bean rows so that plants in the pots were not in contact with plants in the soil bench. Each night, one pot was removed from the soil bench, placed in a dew chamber for 1 day, and then returned to the bench. Each pot of plants received three cycles of 8 days in the soil bench and 1 day of dew treatment. Both senescent and young leaves, flowers, and cotyledons were removed from the potted plants exposed to ascospores in the soil bench, and from potted plants in another greenhouse (control treatment). These organs were examined for symptoms of white mold and incubated in petri-dish humidity chambers.

TABLE 1. Effect of soil moisture on deterioration of sclerotia of *Whetzelinia sclerotiorum* placed at two soil depths and incubated at 5 or 27 C for 1 to 3 months

Incubation period (months)	Depth in soil (cm)	Deterioration of sclerotia (%)					
		PW = 8.4[b]		PW = 17.7[b]		PW = 36.4[b]	
		5 C	27 C	5 C	27 C	5 C	27 C
1	0	0	0	1	59	11	28
1	5	0	5	2	42	7	51
2	0	7	5	7	65	16	25
2	5	1	5	2	76	17	83
3	0	4	5	5	59	7	42
3	5	6	2	6	68	15	73

[a]Average of four replications totaling 100 sclerotia.
[b]PW = percentage of water = grams of water per 100 grams oven-dried soil.

TABLE 2. Percent of sclerotia of *Whetzelinia sclerotiorum* recovered with stipes after burial at five depths in soil for 7, 11, and 17 months

Depth of burial (cm)	Sclerotia with stipes (%)[a] after burial for			
	0 Months	7 Months[b]	11 Months[c]	17 Months[d]
0	0	3	1	1
5	0	58	6	34
10	0	17	4	27
20	0	0	0	4
30	0	1	1	2

[a]Average of four replications.
[b]Spring, 1972.
[c]Fall, 1972.
[d]Spring, 1973.

TABLE 3. Effect of incubation period and depth of burial in soil on formation of stipes from sclerotia of *Whetzelinia sclerotiorum* in culture

Depth of burial	Sclerotia (%)[a] that formed apothecial initials on WA after burial for			
(cm)	0 Months	7 Months[b]	11 Months[c]	17 Months[d]
0	0	2	43	39
5	0	71	38	78
10	0	73	39	86
20	0	83	51	69
30	0	83	38	84

[a]Average of four replications.
[b]Spring, 1972.
[c]Fall, 1972.
[d]Spring, 1973.

RESULTS.—*Sclerotial survival.*—The term deterioration refers to visible degradation of sclerotia, presumably by microbial activity. Although numerous fungi including *Trichoderma* sp., *Aspergillus* sp., *Coniothyrium* sp., and various unidentified nematodes and bacteria were isolated from and found in association with decomposed sclerotia, proof of parasitism was not determined.

Because the survival of 'field' and 'culture' sclerotia was affected similarly by the various temperature and soil moisture regimes, the data from both types of sclerotia were combined.

Few of the sclerotia kept at 5 C deteriorated during the 3-month incubation period (Table 1). Survival of sclerotia was not affected by placement, either on the soil surface or at the 5-cm depth, in soil kept at 5 C for 3 months. Changes in moisture content of the soil at 5 C was minimal over 3 months. Sclerotial survival in soil kept at 27 C with a PW of 8.4 was similar to that at the low and moderate moisture levels kept at 5 C. A high percentage of sclerotia, however, deteriorated during the first 2 months of incubation at 27 C in soils with initial PW of 17.7 and 36.4 (Table 1). Depth of burial had no effect on sclerotial survival in soil with a PW of 17.7, but in soil with a PW of 36.4 nearly twice as many sclerotia deteriorated at the 5-cm soil depth than on the soil surface. Although at 27 C loss of soil moisture ranged from 30 to 50% over 3 months, the comparison of dry, moderate, and wet soil remained valid.

Sclerotia incubated on or in soil germinated and formed the sexual stage in a few instances. Sclerotia kept in soil at 5 C for 3 months and then incubated on WA, germinated to form stipes. These sclerotia (representing 10% of the number originally placed in soil) were recovered from the surface and 5-cm depth in soil with initial PW of 17.7 and 36.4. Only four sclerotia formed stipes while buried in soil, however, and these were recovered after 3 months from soil with PW of 17.7 and 36.4 and kept at 5 C. Five of the sclerotia incubated in soil at 27 C formed stipes on WA and only after 3 months in soil. On PDA plates, mycelial germination of all sclerotia previously buried in any of the treatments was obscured by the rapid growth of other microorganisms.

Most sclerotia buried in nylon mesh sacks in soil at depths of 0, 5, 10, 20, and 30 cm were recovered after 17 months in soil. After the first winter 5, 8, and 17% of the sclerotia buried at depths of 0, 5, and 30 cm, respectively, had deteriorated. Of the sclerotia that remained intact at these three depths after the first winter 22, 28, and 7% respectively, deteriorated in the soil during the next 5 months. Very few of the sclerotia remaining intact after 11 months in soil, deteriorated during an additional 6 months in soil. After burial in the soil for 17 months, 60 and 75% of the remaining sclerotia were intact at depths of 5 and 30 cm, respectively.

Secondary sclerotia were produced on both the inside and outside of nylon mesh sacks recovered at each collection date. Secondary sclerotia developed at all soil depths, but not on the surface of the soil. In most cases, rind fragments of deteriorated sclerotia were adjacent to secondary sclerotia on the inside or outside of the sack.

Some sclerotia had stipes at the time they were recovered from soil. Throughout the burial period, more sclerotia at the 5-cm depth formed stipes than sclerotia at any other soil depth (Table 2). Only 6% of the sclerotia recovered from soil in the fall of 1972, after being buried at the 5 cm depth for 11 months, had formed stipes. However, 58 and 34% of the sclerotia recovered from soil in the spring of 1972 and 1973, after burial for 7 and 17 months, respectively, had produced stipes. A similar, but less pronounced, pattern was observed at the 10-cm depth. Regardless of depth of burial, none of the sclerotia formed stipes longer than 6 cm.

An average of 77 and 79% of the sclerotia buried below the soil surface for 7 and 17 months and recovered in the spring of 1972 and 1973, respectively, germinated to form apothecia on WA (Table 3). In contrast, an average of 42% of the sclerotia buried in the soil at the same depths for 11 months and recovered in the fall of 1972 germinated and formed apothecia. This pattern was not evident with sclerotia kept on the soil surface. After 7 months on the soil surface, very few of the sclerotia recovered in the spring of 1972, germinated to form apothecia. After nearly a year or more on the soil surface, 41% of the sclerotia germinated and produced apothecia in culture. Although the difference is small, sclerotia recovered from the soil surface formed fewer stipes (1-3 per sclerotium) than sclerotia recovered from soil at depths of 5, 10, 20, and 30 cm (4-7 stipes per sclerotium). Thus, after 17 months, sclerotia buried below the soil surface were more apt to form apothecia and in greater numbers than those remaining on the surface.

In another survival experiment, 'field' sclerotia were buried late in the fall of 1970 in a field plot containing 38-cm-diameter tiles inserted in the soil to a depth of 90 cm. Sclerotial recovery facilitated by the ring-shaped membrane technique of Boosalis (2) at 1, 2, and 3 years after burial at 0, 5, 12.5, and 20 cm was conducted as outlined in the previous burial trial. The results of this trial after 1 year confirmed those of the 11-month nylon sack burial trial with respect to sclerotial recovery and stipe formation at the various soil depths. After 2 years burial below the soil surface, an average of 78% of the sclerotia were recovered and 60% formed stipes on WA. Less than 5% had formed stipes in soil. No sclerotia were recovered from the soil surface. After 3 years of burial, three of the four sclerotia recovered from the soil surface germinated and formed stipes on WA. In contrast, 78% of the sclerotia were recovered after 3 years at the three soil depths, and 72% of these formed stipes on WA. Number of stipes per sclerotium ranged from 10 at 5 cm to 5 at 12.5 and 20 cm. At the 5-cm depth, 20% of the recovered sclerotia had formed stipes. Less than 5% of those recovered from other depths had stipes.

Isolations from plant residue and soil.—Studies were made to determine if the pathogen survived in soil and plant residue as sclerotia, ascospores, or mycelium. Sclerotia were readily recovered from soil in 6 of the 20 fields sampled during the 1972 growing season. Although mycelium or ascospores of *W. sclerotiorum* were not recovered from any of the soil samples, the pathogen was isolated as mycelium from 2% of the bean seed collected in the spring from four fields. The pathogen was not isolated from any plant residues screened from 24 fields. Thus, *W. sclerotiorum* did not survive on residues as mycelium or sclerotia, but did survive as mycelium in overwintered bean seed.

Initial infection.—Symptoms of white mold developed on more than 50% of the potted GN bean plants in the greenhouse. These plants had been placed on a bench containing mature apothecia that periodically discharged ascospores. Airborne ascospores detected in the greenhouse presumably settled on the plants and on the soil surrounding the stems. Initially, both abscissed and attached flowers and other plant organs were colonized by hyphae, presumably from germinated ascospores. Bean stems were invaded by mycelium from colonized senescent plant organs that were within 2 cm of the plant. In some cases senescent flowers on the plants were colonized, and from these the pathogen invaded the plant. None of the check treatment plants from an adjacent greenhouse developed white mold.

Presumably, ascospores which were deposited on the aerial parts of the host germinated and colonized the senescent organs. This was confirmed by removing senescent leaves, flowers, and cotyledons from plants exposed to ascospores discharged from apothecia and incubating these organs in petri dish humidity chambers for 2 weeks at 22 C. An average of 15% of the senescent organs which received the treatment were colonized by the pathogen (Table 4). Infection resulting from germinated ascospores did not occur on young organs receiving a similar treatment. Senescent and young leaves and flowers were inoculated with ascospore suspensions and then incubated in petri-dish humidity chambers to determine if ascospores would initiate infection (Table 5). From 70 to 83% of the ascospores placed on either young or senescent organs had germinated 2 days after inoculation. White mold developed on an average of 17 and 28% of the senescent leaves and flowers, respectively, but did not develop on any of the young organs.

TABLE 4. Development of white mold on senescent aerial bean (cultivar, Great Northern) organs which were exposed to mature apothecia of *Whetzelinia sclerotiorum* for 8 days, and incubated in petri dish humidity chambers

	Organs colonized (%)[a]					
	Leaves		Flowers		Cotyledons	
Trial	E[b]	N[b]	E	N	E	N
1	10	0	25	0	20	0
2	5	0	15	0	10	0
3	10	0	30	0	10	0

[a]Based on 20 plant organs; white mold did not develop on young organs.
[b]E = exposed to apothecia, N = not exposed to apothecia.

TABLE 5. Colonization of senescent and young detached bean (cultivar, Great Northern) leaves and flowers inoculated with ascospore suspensions (4,400 spores/ml of water) of *Whetzelinia sclerotiorum* and incubated at 22 C in petri-dish humidity chambers

	Organs colonized (%)[a]			
	Leaves[b]		Flowers[b]	
Trial	Senescent	Young	Senescent	Young
1	15	0	30	0
2	20	0	20	0
3	15	0	35	0

[a]White mold did not develop on noninoculated leaves and flowers.
[b]Twenty organs per treatment.

Under field conditions, white mold was never detected before bean flowers began to senesce, and was usually observed from 1 to 15 days after full bloom. The pathogen was isolated from senescent bean leaves and flowers, but not from any of the young bean organs, grasses, or other weeds collected in and around fields in the North Platte Valley. Many initial infections were observed on young organs (such as leaves, stems and flowers) in contact with colonized senescent flowers in the plant canopy or on the ground. Less than 10% of the colonized senescent flowers on the ground were infested by mycelium from germinated sclerotia. Since sclerotia were not found near the other colonized flowers, it is assumed that hyphae from germinated ascospores colonized more than 90% of these organs. Apothecia were found in numerous bean fields from July into September.

DISCUSSION.—The primary survival structures of *W. sclerotiorum* in the North Platte Valley of western Nebraska were found to be sclerotia. Sclerotia survived in the soil for at least 3 years and were the only structures of the fungus isolated from the soil. Failure to isolate any propagules other than sclerotia from the soil is consistent with the work of Lockwood (8) and van den Berg and

Lentz (17), who demonstrated the short-lived nature of the mycelium of *W. sclerotiorum* in soil. Many of the initial infections in dry edible bean fields could be explained only by airborne inoculum, and the absence of other infective propagules strongly suggested that ascospores were the predominant inoculum.

In some fields, *W. sclerotiorum* overwintered on the soil surface as mycelium in bean seeds. Overwintered seeds and plant debris from an infected crop of beans are plowed and disked into the soil each spring previous to planting. Also, most infected dry edible bean seeds do not germinate and those that do often fail to reach the soil surface (6). Thus, although the fungus can apparently overwinter in bean seed on the soil surface, it is doubtful that infected seeds play an important role in initiating the disease. It is probable, however, that in the soil seed infection results in colonization of the seed by *W. sclerotiorum* and sclerotial production (1) and thus, the inoculum potential of that field could increase.

The only significant carpogenic sclerotial germination occurred at 5- and to some extent the 10-cm depths in soil. Soil temperatures recorded near the bean field did not vary greatly between soil depths and cannot explain germination differences. Smith (13) recently reported that sclerotia buried in soils that are dried and remoistened leak nutrients and tend to germinate myceliogenically. Although soil moisture contents were not obtained, the tendency of Tripp very fine sandy loam soil in the North Platte Valley to dry repidly to a depth of 5 to 10 cm following irrigation or precipitation may partially explain germination at this depth. Williams and Western (19) have also attributed sclerotial survival patterns to soil moisture content. Smith (13) also correlated nutrient leakage and subsequent germination with microbial breakdown of sclerotia. In western Nebraska, however, high rates of sclerotial deterioration from one season to another were not always associated with stipe formation in soil. The laboratory survival trials also indicated a lack of correlation between carpogenic germination and sclerotial deterioration in soil.

Sclerotia of *W. sclerotiorum* do not exhibit dormancy relative to myceliogenic germination, but do for carpogenic germination (3). Stipe formation in culture was nearly maximal after 7 months of burial below the soil surface, indicating that constitutive dormancy (3) had been broken. Nevertheless, only sclerotia at the 5- and to some extent the 10-cm depths had germinated in the soil. Thus, sclerotia at lower depths were still under exogenous dormancy. In Nebraska, subsurface conditions from fall to spring activated sclerotia, but conditions on the soil surface did not. Further activation and subsequent germination occurred at depths of 5-10 cm. Although other factors could be involved, moisture fluctuations may be a primary germination stimulus.

Rotation of the dry bean crop with corn and sugar beets every third year, which is commonly practiced in the North Platte Valley, is not an effective control measure for white mold. This relates to the fact that the pathogen survives well in soil apart from its hosts. Sclerotia of *W. sclerotiorum* have the capability of forming secondary sclerotia, which could result in an increase in soil inoculum. In recent years, increased prevalence of the pathogen has added more sclerotia to the soil, thereby increasing the inoculum potential. Tillage practices employed with all crops would almost assure the presence of sclerotia on or near the soil surface each year.

Ascospores were found to be the primary source of initial inoculum in western Nebraska. The results of our study indicate that the pathogen does not survive as mycelium in soil or residue, and that survival of mycelium in seed is not important in disease initiation. Stevens and Hall (16) and Morgan (9) isolated sclerotia and mycelium of *W. sclerotiorum* from various weed hosts and suggested that infected weeds may serve as inoculum sources. There is no evidence at this time for considering weeds to be an important source of *W. sclerotiorum* in Nebraska. Mycelium from germinated sclerotia initiated less than 10% of the initial infections observed on beans in the field and greenhouse.

Many of the initial infections of beans in the field and greenhouse started in the plant canopy. Most of these infections were initiated by mycelium from colonized senescent flowers adhering to various plant organs. This infection pattern indicates air-borne inoculum, and correlates with the presence of nearby mature apothecia. Natti (10) suggested that wind-disseminated particles of plant debris containing mycelium may serve as the causal agent for initial infections in bean fields in New York, but did not account for the origin of the debris and mycelium. In Nebraska, mycelium of *W. sclerotiorum* did not overwinter in plant debris although it did survive in bean seed. Although based on indirect evidence, consideration of ascospores as the primary initial inoculum source is consistent with the available data.

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