Ecology and Epidemiology

Epidemiology of Sclerotinia Stem Rot of Soybean in Ontario

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ABSTRACT

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The epidemiology of Sclerotinia stem rot of soybean in Ontario was investigated in 1981 and 1982. Apothecia of *Sclerotinia sclerotiorum* appeared after the development of a crop canopy during mid to late July and were present for 5–6 wk thereafter. Soil matric potentials were generally ≥ -5 bars during the 2 wk preceding the occurrence of apothecia. Epidemics started in early to mid August and continued until near crop maturity. Disease first appeared after closure of the crop canopy, after crop flowering had started, after apothecia appeared within the crop, and after rain had initiated periods of plant surface wetness lasting for 40–112 hr. Mean daily air temperatures were in the range of 12–24 C. Disease development required 70–120 hr of continuous plant surface wetness at 20 C in controlled-environment inoculations with ascospore suspensions.

Additional keyword: *Glycine max*.

Sclerotinia stem rot of soybean (*Glycine max* (L.) Merr.), caused by *Sclerotinia sclerotiorum* (Lib.) de Bary, was first reported in Ontario in 1946 (16) and 1948 (13), but was not considered a threat to crop production. Severe localized outbreaks, however, have been reported from Arizona (14), Illinois (5,6), Minnesota (9), Virginia (22), and Wisconsin (10,11). Disease outbreaks have been associated with an expansion of soybean production into areas with a history of this pathogen in other crops, cool environmental conditions that favor disease, and changes in cultural practices such as row spacing and irrigation management (6,10).

The disease cycle of Sclerotinia stem rot of soybean has not been fully elucidated, but appears to be similar to white mold of white bean (*Phaseolus vulgaris* L.) (1,2). Disease symptoms appear after crop flowering and canopy enclosure. Lesions are commonly observed on the main stem 15–40 cm above the soil surface but are observed also on lateral branches and on the main stem at the soil surface (9,11). Stem and branch lesions commonly originate at leaf axils where soybean inflorescences are located. These observations suggest that air-borne ascospores are the primary inoculum for disease and that senescent flowers are the primary infection site. The pathogenicity of ascospores on flowering, but not prebloom, soybean plants has been demonstrated (6).

Little information is available on the disease cycle or epidemiology of Sclerotinia stem rot of soybean under field conditions. The purpose of the present study was to monitor selected biological and environmental conditions in field plots of soybean and to correlate these conditions with the temporal and quantitative occurrence of disease.

MATERIALS AND METHODS

A field plot (21×21.5 m) of soybean (cv. Evans) was established in 1981 and 1982 on sandy loam soil at Arkell, Ontario. The plot had a history of white mold of white bean, and a severe epidemic

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occurred in 1980. The plot was prepared for planting by spring ploughing and disking in 1981, and spring disking in 1982. The herbicide trifluralin (Elanco Prod. Div., Eli Lily and Co. Canada Ltd., Scarborough, Ontario) was applied as a preplant incorporated treatment at 1.5 L/ha each year. Spot applications of glyphosate (Monsanto Can. Inc., Streetsville, Mississauga, Ontario) were applied before planting in 1982 at 2.5 L/ha.

Seed was treated with B-3 seed treatment (diazinon, lindane, captan; Chipman Chem. Ltd., Stoney Creek, Ontario) and sown at a rate of 20 seeds per meter of row in 31 rows 72 cm apart. The plots were established on 2 June 1981 and 9 June 1982. In 1982, a 4-m unseeded gap was left in rows 12, 17, and 23 at the time the main plot was planted. These gaps were seeded on 16, 23, and 30 June, respectively, and were used as observation plots representing three additional planting dates.

Air temperature was monitored with a hygrothermograph (model 252; Lambrecht, Goettingen, West Germany) in a Stevenson shelter within the plot. Rainfall was measured with a tipping bucket rain gauge (model P-251; Weather Measure Corp., Sacramento, California). In both years, duration of plant surface wetness was monitored with a DeWit recorder (DeWit, Hengelo, The Netherlands) within the crop canopy at 20 cm above ground level. In addition, in 1982, plant surface wetness was monitored with electrical impedance leaf wetness sensors (18,27). Six sensors were wired in parallel and attached to leaves, petioles, and stems within the soybean crop canopy during August and September. Changes in electrical resistance were recorded on a Rustrak strip chart recorder (model 291; Gulton Industries Inc., Manchester, New Hampshire). The recording system was modified to accept alternating current (8). In 1982, soil moisture was determined daily by gravimetric determinations from 15 June to 4 September. Soil samples were taken from the surface 2–3 cm of soil at three locations within the field plot. Each sample was weighed before and after drying at 35 C for 2–3 days. Percentage soil moisture was converted to matric potential from a soil desorption curve determined with a pressure plate apparatus (23).

Crop stages were determined at weekly intervals by using soybean growth stage descriptions (7). The growth stages recorded were divided into vegetative, flowering, and ripening periods. When all plants had at least one open flower, the field was said to be in full bloom. When the foliage of adjacent rows touched, the canopy was said to be closed.

A plant was considered diseased if symptoms of Sclerotinia stem rot were detected on any part of the plant. Disease incidence was calculated as the number of diseased plants expressed as a percentage of the total number of plants in the sample. A disease severity index was calculated as the sum of (severity class × number of plants in class) × 100 ÷ (total number of plants) (11,25). The severity classes ranged from 0 to 4, in which 0 = no disease, 1 = small lesion(s) less than 5 cm in the longest dimension, 2 = expanding lesions on branch or stem, 3=up to one-half of branches or stem colonized, and 4=more than one-half of branches or stem colonized and/or plant dead.

In 1981, the field plot was divided into 310 subplots by establishing 10 subplots 2 m long in each row. Each subplot ranged to 36 cm on either side of the row. The sample area in each subplot was located on only one side of the row and was 2 m long and 36 cm wide. The number of apothecia in each sample area was counted in all subplots on 17 and 31 July, and in 160 subplots on 10 and 14 August. The mean number of apothecia per sample area was multiplied by two to correct for rating one-half of each subplot. Preliminary counts of apothecia had indicated that there were no consistent differences in the number of apothecia between the two sides of the subplots. The presence or absence of apothecia within the field plot was determined at frequent intervals. Disease incidence was determined in all subplots on 5, 10, 14, and 17 August and 10 September. Environmental variables and crop development were monitored from 2 June to 10 September.

In 1982, the field plot was again marked into 310 subplots. The presence of apothecia in the field was recorded throughout the season, and the number of apothecia in each subplot was counted on 9 and 24 August and 1 September. Disease incidence and severity were rated daily after disease was first observed by selecting 100 plants from the field plot. Detailed observations on the initiation and expansion of individual lesions were made in four observation plots representing four planting dates. Fifty consecutive plants (total of 200 plants) were marked with plastic tags and rated daily for the appearance of new lesions, lesion size, and secondary spread of disease. Environmental variables and crop development were monitored from 15 June to 14 September.

In 1982, an additional field plot of soybean was established at Woodstock, Ontario. This field plot consisted of one treatment within a cultivar trial containing seven soybean cultivars planted as a randomized complete block design with four replicates. Each plot was sown with 20 seeds per meter of row with four rows 36 cm apart. Each row was 7 m long at planting and later trimmed to 6 m. The herbicides metolachlor (Ciba Geigy Can. Ltd., Cambridge, Ontario) and metribuzin (DuPont Canada Inc., Mississauga, Ontario) were applied as a tank-mixed, preplant incorporated treatment at rates of 1.75 and 0.42 L/ha, respectively. The field was planted on 16 May. Disease incidence, the number of senescent flower blossoms per four plants, and the number of apothecia between two middle rows of each plot were determined at weekly intervals in each replicate of the cultivar Evans only. Plant surface wetness was recorded with a DeWit recorder within the plot canopy of one replicate only at 20 cm above soil level. Rainfall data were obtained from a private weather station approximately 1.5 km from the field plot.

The duration of plant surface wetness required to produce symptoms of Sclerotinia stem rot at 20 C was investigated in growth chamber studies. Plants were grown until flowering in a growth room maintained at 22 ± 2 C with a photoperiod of 14 hr. Fluorescent and incandescent lamps provided a quantum flux density at plant height of 150 $\mu E/m^2/sec$. Two flowering soybean plants, in each of 18 pots, were inoculated in each experiment. Plants were inoculated at sites where senescent flower petals had been placed or had lodged naturally. These sites were sprayed with a hand atomizer until runoff with a suspension of 3,500 viable ascospores per milliliter. Ascospores were obtained from field-collected apothecia and stored on Millipore filters (26) at 3–4 C in a desiccator (15). Viability was determined by placing filter sections on potato dextrose agar medium (BBL, Becton, Dickinson and Co., Cockeysville, Maryland) for 12 hr. Ascospores and germ tubes were stained with lactophenol cotton blue and counted at 400×. Ascospores were considered germinated if the germ tube was longer than the length of the ascospore. Three separate counts were made on each sample, and at least 100 spores were assessed per count.

Inoculated plants were placed in a mist chamber that maintained continuous plant surface wetness at 20 C. At 8–12 hr intervals, two pots of plants were removed and placed in a nonmisted growth chamber at the same temperature. Plants were usually dry 15–20 min after transferal between growth chambers. Observations on the number of lesions per pot were made 2–3 days after plants were removed from the mist chamber. The experiment was conducted three times.

RESULTS

Sclerotinia stem rot was initially observed in all field trials after crop flowering had started, after apothecia had appeared within the crop, and after extended periods of plant surface wetness had occurred (Figs. 1–3). Lesions of Sclerotinia stem rot were most frequently observed on developing and aborted pods attached to inflorescences at nodes 1–5 of the main stem and on lateral branches. Lesions expanded from the inflorescence to the main stem by direct mycelial colonization of tissues or by spread from colonized tissues that had fallen or lodged onto healthy tissues. Further colonization of host tissues resulted in extensive colonization of the main stem and lateral branches. Rapid colonization of lower branch tissues often resulted in spread of disease back into the main stem. Disease incidence continued to increase until crop maturity and reached 40% in 1981 (Fig. 1), 23% at Arkell in 1982 (Fig. 2), and 21% at Woodstock in 1982 (Fig. 3).

The initial appearance of apothecia or disease was associated with particular stages of crop development. The crop canopy became closed, and crop flowering was in at least the R1 stage of development before apothecia were observed, and at least the R3 stage of flowering before disease was observed, in 1981 and 1982 (Figs. 1 and 2). At Woodstock, the number of apothecia between the two middle rows of plants and the number of senescent blossoms per plant were at or near their maximum values when disease was first observed (Fig. 3).

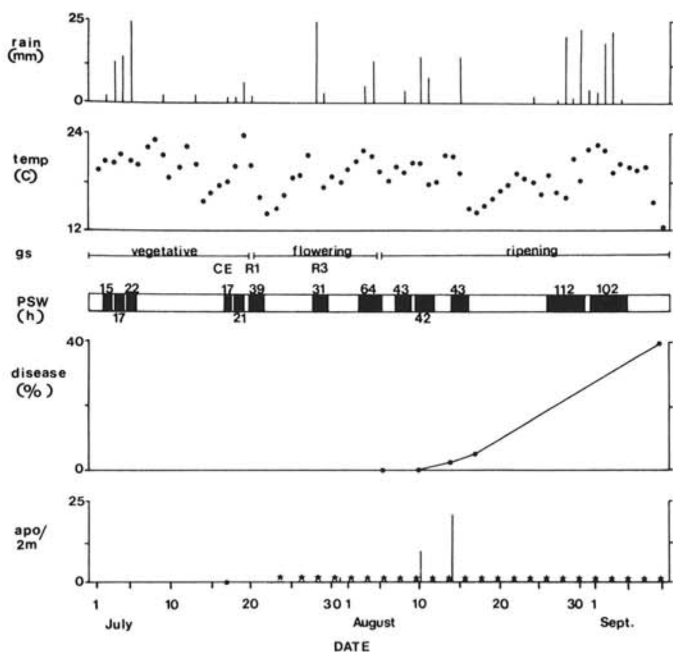

Fig 1. Incidence of Sclerotinia stem rot (disease) of soybean in relation to rainfall (rain), mean daily air temperature (temp), crop growth stage (gs), plant surface wetness (PSW), and mean number of apothecia per 2 m of crop row (apo/2 m) in a field plot at Arkell, Ontario in 1981 (C = canopy closure, R1 and R3 = crop growth stages, * = apothecia present within the field plot but not counted).

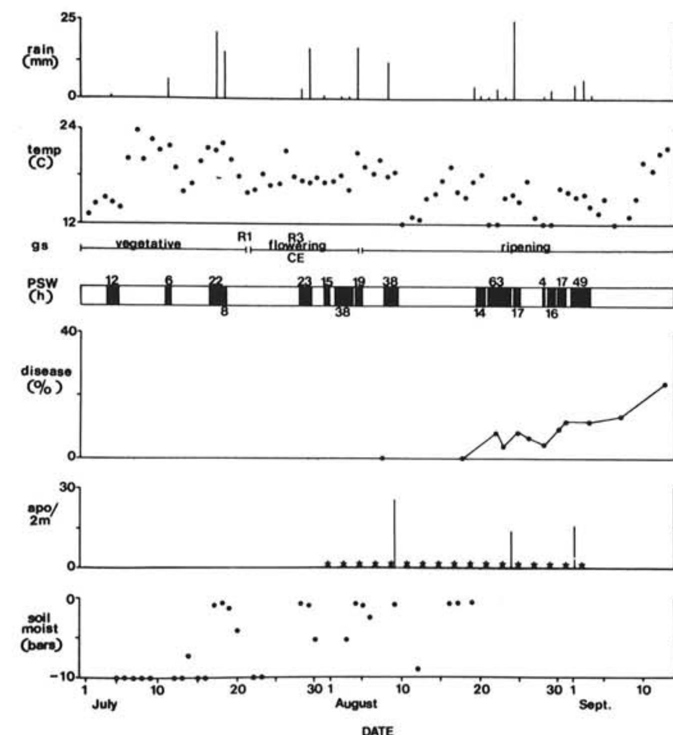

Fig. 2. Incidence of Sclerotinia stem rot (disease) of soybean in relation to rainfall (rain), mean daily air temperature (temp), crop growth stage (gs), plant surface wetness (PSW), mean number of apothecia per 2 m of crop row (apo/2 m), and matric potential (soil moist) in the surface 2–4 cm of soil in a field plot at Arkell, Ontario in 1982 (C = canopy closure, R1 and R3 = crop growth stages, * = apothecia present within the field plot but not counted).

Apothecia were present within all field plots before disease appeared and remained present until near crop maturity. Intermittent rainfall for 4 days (Fig. 1) and 2 days (Fig. 2) after canopy closure was associated with the initial appearance of apothecia 5 and 2 days later, respectively. Rainfall before canopy closure also may have influenced apothecial development. For example, soil matric potentials were ⩾ −5 bars in seven of nine consecutive measurements before the appearance of apothecia in 1982 (Fig. 2). The largest population of apothecia was observed on 9 August 1982 after 8–10 days of high matric potential (⩾ −5 bars). Apothecia were present within the field plots at Arkell for the remainder of the growing season in 1981 (Fig. 1) and until 2 September 1982 (Fig. 2). Apothecia were present at Woodstock from 22 July to 27 August 1982 (Fig. 3). The mean number of apothecia in all experiments ranged from 0–25 apothecia per 2 m of crop row (1.4 m^2).

The initial appearance of Sclerotinia stem rot lesions was associated with extended periods of plant surface wetness. In 1981, disease incidence increased to 2.7% following a 42-hr period of plant surface wetness from 10–12 August (Fig. 1). Most lesions at this time were small and restricted, but some lesions were more extensive and may have been initiated during earlier periods of plant surface wetness lasting 64 and 43 hr. In 1982, disease incidence increased to 8.0% during two interrupted periods of plant surface wetness lasting 14 and 63 hr (Fig. 2). Disease first occurred at Woodstock following a plant surface wetness period lasting 89 hr (Fig. 3). Subsequent increases of disease incidence in all three plots were associated with plant surface wetness periods lasting from 40–112 hr, as measured by the DeWit recorders (Figs. 1–3).

The development of lesions on 50 consecutive plants in the field plot was monitored after the epidemic started (Table 1). New lesions were observed on all eight dates that observations were made. Of 48 lesions observed, 28 appeared within 24 hr after extended periods of plant surface wetness from 19–23 August and 1–3 September, and an additional 10 lesions developed within the following 1–3 days. The remaining 10 lesions appeared separately in association with periods of plant surface wetness lasting for

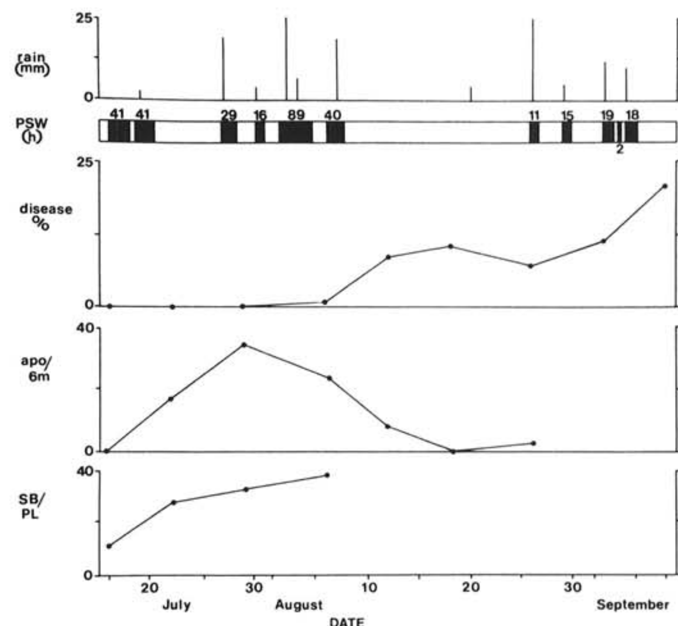

Fig. 3. Incidence of Sclerotinia stem rot (disease) of soybean in relation to rainfall (rain), plant surface wetness (PSW), mean number of apothecia per 6 m of crop row (apo/6 m), and mean number of senescent blossoms per plant (SB/PL) in a field plot at Woodstock, Ontario in 1982.

3.75–17.0 hr. Three lesions were caused by secondary spread of mycelium from diseased to healthy tissues.

Measurements of individual lesions indicated that environmental conditions associated with lesion initiation were also associated with increases of disease severity. Disease incidence and the disease severity index were correlated in the period 18 August to 13 September ($r = 0.99$; $P = 0.0001$). Lesions on stems and branches continued to develop in most conditions but expanded most rapidly during periods of extended plant surface wetness associated with dew and rainfall. Lesion expansion on pods was slow during evenings when only dew was present.

In controlled environment studies, the duration of plant surface wetness at 20 C required to initiate lesions of Sclerotinia stem rot averaged 95.5 hr and ranged from 70–120 hr.

DISCUSSION

The development of the crop, reproduction of the pathogen, and initiation of disease and environmental factors were all interrelated. The development of a closed crop canopy provided extended periods of high soil moisture required for carpogenic germination and permitted the appearance of apothecia within the field plot. Crop flowering began at approximately the same time as canopy closure. Subsequent development of disease followed periods of extended plant surface wetness and mean daily air temperatures of 12–24 C. The effect of closure of the crop canopy on providing cooler, wetter environmental conditions favoring carpogenic germination, spore survival, and disease development have been reported also for white mold of bean (3,4,24,29,30). Similarly, the appearance of apothecia in rapeseed fields coincided with the early bud development stage of the crop and was associated with microclimatic conditions affected by crop phenology (21).

TABLE 1. Time and duration of plant-surface wetness (PSW) in a field plot of soybean during August and September 1982 in relation to the appearance of lesions of Sclerotinia stem rot and change in disease severity index

Date	Time of initial wetness (hr)	Time of initial dryness (hr)	PSW duration (hr)	No. of new lesions per 50 plants[a]	Disease severity index[b]
August					
18-19	1845	0745	13.0	···	···
19	1015	1715	7.0	···	···
19-20	2145	1100	13.25	···	···
20-21	1830	1215	17.75	···	···
21-23	1930	1045	39.25	15	14
23	1315	1515	2.0	7	10
23-24	1630	0900	16.5	···	···
24-25	1500	0915	18.25	5	26
25-26	2115	0630	9.25	4	17
26-27	1830	0830	14.0	···	···
27	1200	1545	3.75	···	···
27-28	2130	0200	4.5	3 (1)[c]	12
28	1200	1630	4.5	···	···
28-29	1915	1030	15.25	···	···
29-30	1815	1115	17.0	7	31
30-31	1900	0745	12.75	···	32
September					
1	0315	0545	2.5	···	···
1-3	1900	1530	44.5	6 (1)	35
3-4	2100	1000	13.0	···	···
4-5	1815	0915	15.0	···	···
5-6	1845	0845	14.0	···	···
6-7	2145	0030	2.75	1 (1)	35
7-8	1900	0830	13.5	···	···
8-9	1945	0800	12.25	···	···
9-10	1800	0930	15.5	···	···

[a] Number of new lesions observed in 50 plants.

[b] Disease severity index = the sum of (severity class × number of plants in class) × 100/total number of plants. Severity classes are 0 = no disease; 1 = small lesion(s) less than 5 cm; 2 = expanding lesion on branch or stem; 3 = up to one-half of branches or stem colonized; and 4 = more than one-half of branches or stem colonized and/or plant death.

[c] Numbers in parentheses represent lesions initiated by secondary spread of disease (mycelial spread from diseased to healthy tissues).

Apothecia were observed after canopy closure and after soil matric potentials were increased to $\geqslant -5$ bars in association with periods of rainfall for most of a 16-day period. Controlled environment studies have indicated that preconditioned sclerotia require matric potentials at or above -7.5 bars for about 10 days or longer (12,19,28) to produce apothecia. However, drying of the soil occurred in our field study during the 16-day period, and new apothecia appeared 3 days after rain had rewetted the soil. Similarly, Morrall (20) reported that apothecia emerged as soon as 4 days after a period of intense rainfall. Sclerotia may be capable of continued carpogenic germination when exposed to brief periods of alternating high and low soil matric potentials.

Extended periods of plant surface wetness lasting 40–112 hr in field conditions and 70–120 hr in growth chamber conditions (20 C) were associated with disease occurrence. Abawi and Grogan (1) reported that 48–72 hr of continuous plant surface wetness were required for symptoms of disease to appear when ascospores were inoculated onto senescent blossoms lodged on snap bean plants in the growth room. High relative humidity (near 100%) was not sufficient for either lesion initiation or expansion on bean leaves. Similarly, Boland and Hall (3) reported that at least 54 hr of continuous plant surface wetness at 20 C was required for symptoms of white mold in bean to develop in controlled environment studies, but that symptoms in field conditions were observed after only 39 hr of plant surface wetness. Lamarque (17) reported that flowering heads of sunflower must remain continuously wet for at least 42 hr for infection to develop.

Once the epidemic had started, a number of new lesions of Sclerotinia stem rot appeared in association with shorter plant surface wetness periods of 3.75–17.0 hr. Abawi and Grogan (1) reported that three of 51 snap bean plants inoculated with an ascospore suspension became infected after 16 hr of plant surface wetness in field conditions. Boland nad Hall (3) found that lesion development in white beans was associated in some cases with 14–15 hr plant surface wetness. In Nebraska, white mold developed in irrigated fields of dry bean in association with daily plant surface wetness periods of 11–12 hr (2). The possibilities that *S. sclerotiorum* can infect following several consecutive short periods of plant surface wetness or that the inoculum concentration affects the duration of plant surface wetness required for infection should be explored.

Grau and Radke (10) reported that supplemental irrigation and amount of precipitation influenced severity of Sclerotinia stem rot but that disease development was predicted more accurately from air temperatures recorded before and after crop flowering. Minimum and maximum air temperatures recorded 2 wk before and 4 wk after flowering ranged from 12–18 C and 22–30 C, respectively, in years when disease occurred. The minimum and maximum temperatures when disease did not occur were 20 and 34 C, respectively (10). In our study, mean daily air temperatures ranged from 12–24 C during the 2 wk before and 4 wk after flowering, and the minimum/maximum air temperatures during this period were 5.5/30.5 in 1981 and 5.5/29.0 in 1982, respectively. These observations agree with those of Grau and Radke (10) and indicate that air temperatures were not a limiting factor during the 2 yr of this study.

Little information is available on the epidemiology of Sclerotinia stem rot of soybean, but the disease cycle appears to be similar to that of white mold of white bean. A comparable study of white mold (3) in a field plot adjacent to this study indicated a number of similarities in the two diseases. In both crops, the development of a crop canopy preceded the appearance of apothecia within the plot, and disease did not occur until crop flowering had started. The initiation of the epidemics in early to mid August and the occurrence of subsequent lesions were associated with colonized flower tissues and extended periods of plant surface wetness. The primary inoculum for both diseases appeared to be ascospores, and the primary infection sites appeared to be senescent tissues such as flower petals. Despite the

qualitative similarities in disease cycles, final disease incidences were considerably higher in white bean than in soybean.

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