Sclerotinia Blight of Soybean Caused by *Sclerotinia minor* and *Sclerotinia sclerotiorum*

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ABSTRACT

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In August 1978, Sclerotinia blight of soybean was found in southeastern Virginia where soybean and peanut are major field crops. *Sclerotinia minor* was identified as the cause of blight in all but one soybean field, where *S. sclerotiorum* was the cause of disease. Pathogenicity tests demonstrated that both species of the fungus were virulent on soybean and peanut. Because of the sporadic incidence of Sclerotinia blight of soybean, the disease is believed to have little or no effect on soybean yield. The continued intercropping of soybean and peanut may increase its occurrence on both hosts.

Additional key words: *Arachis hypogaea, Glycine max*

In 1946, Weiss (10) listed *Sclerotinia sclerotiorum* (Lib.) de Bary as causing a stem rot of soybean in Iowa, New York, Maryland, and Virginia. Subsequent reports of the disease have been infrequent. In most cases, serious localized outbreaks have been linked with prolonged cool, wet periods in areas where the fungus had become established on other hosts (1,2,4,5,9).

With the exception of the listing by Weiss (10), we know of no reports of *S. sclerotiorum* or *S. minor* Jagger (6) on soybean in Virginia. A severe disease of peanut caused by *S. minor* was first observed in Virginia and North Carolina in 1971 (8). By 1975, *S. minor* had become a major pathogen of peanut in all counties in Virginia where peanuts are grown.

This paper reports the first occurrence of a soybean disease caused by *S. minor*. Pathogenicity tests and axenic growth tests were performed to compare isolates of *S. minor* and *S. sclerotiorum* from naturally infected soybean. Sclerotinia blight of soybean is the proposed name for this disease to preclude confusion with southern stem rot caused by *Sclerotium rolfsii*, which also occurs on soybean and peanut.

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MATERIALS AND METHODS

More than 100 soybean fields in southeastern Virginia were surveyed for Sclerotinia blight in August and September of 1978, 1979, and 1980. Isolates of *Sclerotinia* spp. were obtained by washing sclerotia and tissues to remove soil and debris, surface sterilizing tissues for 1 min in 0.5% sodium hypochlorite, and plating materials on acidified potato-dextrose agar.

Isolates of *Sclerotinia* spp. from soybean were characterized in axenic culture by assessing the effect of temperature on growth and sclerotia production in incubators set to maintain temperatures of 5, 10, 15, 20, 25, and 30 C (± 1.5 C). These determinations utilized potato-dextrose agar (PDA) in 9-cm-diameter petri dishes. The thickness of the agar medium in each dish was standardized at 2 mm, and measurements of radial growth were made at 24-hr intervals after inoculation of plates. Sclerotia production was assessed by counting numbers of sclerotia formed after 12 days of incubation.

The pathogenicity of isolates on selected cultivars of soybean and peanut was determined in a controlled-environment chamber. Relative humidity near 100% was maintained by a water-mist system that sprayed plants for 15 min every hour during daytime hours (0600–1800) and 15 min every 2 hr during hours of darkness (1800–0600). Day temperatures in the chamber were maintained at 25 C (± 2 C) and night temperatures at 18 C (± 2 C).

Four-week-old soybean seedlings and 6-wk-old peanut seedlings in 15-cm-diameter clay pots were inoculated by placing a 3-mm PDA disk from 5- to 7-day-old cultures of the fungus against the main stem of plants at the soil surface. The agar disks were then covered with moist cheesecloth and held in place with toothpicks inserted in soil. Soybean cultivar inoculations were replicated six times with six plants per replicate, whereas peanut cultivar inoculations were replicated four times with three

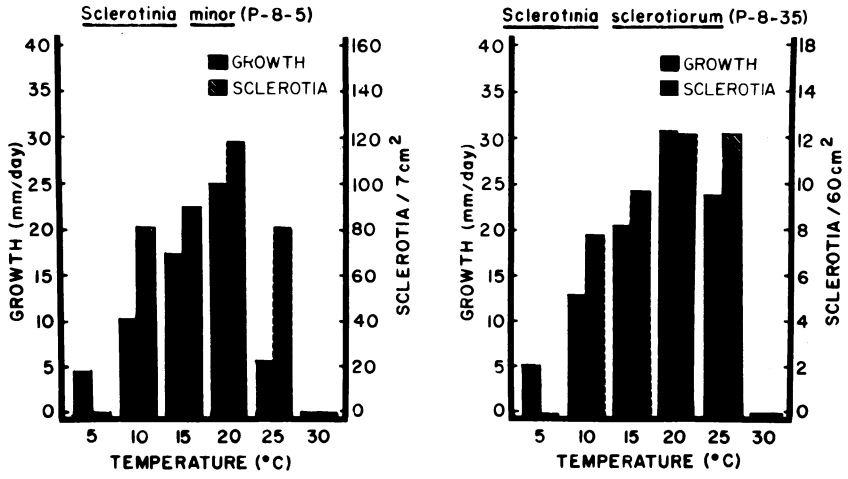

Fig. 1. Effect of temperature on mycelial growth and production of sclerotia by *Sclerotinia minor* (sclerotia per 7 cm^2) and *S. sclerotiorum* (sclerotia per 60 cm^2) in axenic culture. Both isolates were from naturally infected soybean plants.

Table 1. Reaction of three soybean cultivars to inoculation with isolates of *Sclerotinia minor* and *S. sclerotiorum* from naturally infected soybean

	Percentage diseased	
Cultivar	*S. minor* (P-8-5)	*S. sclerotiorum* (P-8-35)
Essex	78	67
York	86	57
Forrest	81	56
Mean[a]	81.7	60.0

[a] Means for inoculum were significantly different ($P = 0.01$), whereas means for varieties were not significantly different ($P = 0.05$) according to analysis of variance.

Table 2. Reaction of six peanut cultivars to inoculation with isolates of *Sclerotinia minor* and *S. sclerotiorum* from naturally infected soybean

	Disease index[a]	
Cultivar	*S. minor* (P-8-5)	*S. sclerotiorum* (P-8-35)
Florigiant	71	29
NC 6	63	25
NC 7	63	25
NC 3033	83	25
Argentine	63	42
VPG-1	88	42
Mean[b]	71.8	31.3

[a] 0 = no symptoms or signs of disease; 50 = symptoms and signs present, but plant not dead; 100 = symptoms and signs present and plant dead.

[b] Means for inoculum were significantly different ($P = 0.01$), whereas means for varieties were not significantly different ($P = 0.05$) according to analysis of variance.

plants per replicate. Observations for signs and symptoms of disease were made daily after inoculation; at the completion of each test, biopsy samples of symptomatic tissues were assayed for *Sclerotinia* spp. as described earlier with acidified PDA.

RESULTS

Field observations. Symptoms and signs of Sclerotinia blight of soybean were observed primarily at points where plant tissues (leaves, pods, stems) were in contact with soil. In all cases, the fungus appeared to initiate infection following growth from soil in contact with plant tissues. Frequently, such growth appeared to have been initiated first by colonization of a senescing leaf beneath a dense foliar canopy of growth. Diseased leaf petioles and soybean stems exhibited a reddish coloration at the advancing margin of lesions. Dead stems, pods, and leaf petioles were commonly light tan in color. No evidence of infection in taproots or lateral roots of plants was observed.

Signs of the disease were present as mycelium and sclerotia on the surface as well as inside infected tissues. With the exception of signs observed in one field, the sclerotia were small (1–2 × 1–5 mm) and irregular in shape with a black surface and cream-colored interior. These sclerotia were identical in appearance to sclerotia found associated with diseased peanuts exhibiting Sclerotinia blight caused by *S. minor*. In one soybean field, only large sclerotia (2–5 × 4–30 mm) characteristic of *S. sclerotiorum* were found associated with diseased soybean plants. In contrast to *S. minor*, which produced small sclerotia in the pith tissues of stems, *S. sclerotiorum* produced large sclerotia that completely occupied the normal pith region of stems. No apothecia of either *S. minor* or *S. sclerotiorum* were found in the vicinity of diseased plants in either soybean or peanut fields.

Extensive surveys from 1978 to 1980 for Sclerotinia blight of soybean in southeastern Virginia revealed its presence in three of the six major peanut-producing counties. In more than 100 fields surveyed, the disease occurred infrequently; only a few dead soybean plants with symptoms and signs of Sclerotinia blight were found. No suppression of crop yield and value as a result of this disease was apparent in any soybean field. Adjacent peanut fields in 1978 and 1979, however, often had up to 20% incidence of Sclerotinia blight caused by *S. minor*. Sclerotinia blight of peanut, caused by *S. sclerotiorum*, was not found in any of more than 100 peanut fields examined.

Effect of temperature on axenic growth of isolates. An isolate from soybean identified as *S. minor* (P-8-5) and one identified as *S. sclerotiorum* (P-8-35) were selected to assess the effect of temperature on axenic growth. The isolate of *S. minor* produced numerous small sclerotia (0.5–2 mm long) in culture, whereas the isolate of *S. sclerotiorum* produced a single concentric ring of large sclerotia (2–12 mm long) near the margin of petri dishes.

Mycelial growth and formation of sclerotia by both organisms increased as temperatures increased from 5 to 20 C (Fig. 1). At 25 C, mycelial growth by the isolate of *S. minor* was markedly limited to rates equivalent to growth at 5 C. Growth by the isolate of *S. sclerotiorum* at 25 C was less than the rate at 20 C but greater than its growth rate at 15 C. Neither isolate was able to sustain growth at 30 C.

Pathogenicity tests. In two preliminary tests with soybean and peanut seedlings, stems had to be wounded before inoculation for uniform infection and subsequent disease development. A needle probe was used to puncture the surface tissues of stems before placement of inoculum against a stem in all subsequent tests.

Essex, York, and Forrest cultivars were selected to assess the pathogenicity of *S. minor* and *S. sclerotiorum* on soybean. Collectively, these cultivars were planted to 86.1% of the soybean acreage in Virginia in 1978. *S. minor* was significantly ($P = 0.01$) more pathogenic than *S. sclerotiorum* on all soybean cultivars (Table 1). No significant differences ($P = 0.05$) in cultivar reaction to *S. minor* or *S. sclerotiorum* were apparent. In most instances, the appearance of symptoms and signs of disease was accompanied by a rapid wilt and subsequent death of soybean seedlings.

The reaction of selected peanut cultivars to *S. minor* and *S. sclerotiorum* was assessed because of the widespread occurrence and importance of Sclerotinia blight of peanut in Virginia caused by *S. minor*. As in soybean tests, *S. minor* was significantly ($P = 0.01$) more pathogenic on peanut than was *S. sclerotiorum* (Table 2). The peanut cultivars resistant to Cylindrocladium black rot (NC 3033, Argentine, VPG-1) appeared to be as susceptible as the commercial cultivars of peanut (Florigiant, NC 6, NC 7).

DISCUSSION

Sclerotinia blight of peanut, caused by *S. minor*, has been a major disease on many farms in Virginia for nearly a decade (8). Although *S. minor* has been reported pathogenic on soybean (3), field incidence of *S. minor* on soybean was not detected until 1978. *S. sclerotiorum* has never been associated with disease of peanut in Virginia, but it has been reported as a parasite of soybean (10). *S. sclerotiorum* causing disease of vegetables in southeastern Virginia was reported as early as 1917 (7).

The outbreaks of Sclerotinia blight of soybean caused by *S. minor* and in one case *S. sclerotiorum* in southeast Virginia in 1978 appear to be partly the result of increased intercropping of soybean and peanut. Pathogenicity tests indicated that *S. minor* was more pathogenic than *S. sclerotiorum* on both soybean and peanut. The greater pathogenicity of *S. minor*, coupled with the innate capacity to produce sclerotia more prolifically than *S. sclerotiorum*, may account for the more frequent occurrence of *S. minor* on soybean.

Our surveys for Sclerotinia blight of soybean and peanut indicated that infection by *S. minor* occurred at or near the soil surface in fields with dense canopies of foliage. We believe that sclerotia of *S. minor* were the primary propagule responsible for initiating infection and spread of the disease in soybean as well as peanut in Virginia, because apothecia were not found in spite of intensive scouting. Based on our observations of Sclerotinia blight incidence and severity in soybean fields in 1978, we believe that the disease resulted in no significant suppression of yield. In 1979, Sclerotinia blight was not found on soybean. Surveys in 1980 discovered only one field with a few Essex soybean plants showing symptoms and signs of Sclerotinia blight caused by *S. minor*.

The parasitic activity of *S. minor* and *S. sclerotiorum* on soybean and peanut may increase in southeast Virginia with continued intercropping of soybean and peanut. Although *S. sclerotiorum* appeared to be somewhat less aggressive than *S. minor* on these crops, the capacity of *S. sclerotiorum* to grow at slightly warmer temperatures may favor its increase on soybean and subsequent spread to peanut.

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