An Assessment of Environmental Conditions Preceding Outbreaks of Sclerotinia Blight of Peanut in Virginia[1]

P. M. Phipps[2]

ABSTRACT

Records of outbreaks of Sclerotinia blight of peanut at the Tidewater Center in Suffolk were summarized over a 16-yr period (1978-93). Initial outbreaks of the disease were determined through intensive scouting of fields at weekly intervals. First occurrences of Sclerotinia blight occurred between 10 July and 7 Sept., or 62 to 120 d after planting (DAP). The mean and median dates of initial outbreaks were 28 July (79 DAP) and 25 July (76 DAP), respectively. The initial onset of disease always occurred after vines were within 15 cm of touching between rows or after vines had lapped between rows. Under these conditions, a canopy of dense foliage shaded the soil surface and infection sites inside rows from direct sunlight. Rainfall summaries showed the heaviest accumulations were from 6 to 15 d before disease outbreaks. Maximum and minimum air temperatures over the 15-d period prior to disease onset averaged *ca.* 32 C and 20 C, respectively. Maximum and minimum soil temperatures at the 10-cm depth averaged *ca.* 30 and 25 C, respectively, during this same period. The results of this study provide evidence that vine growth and rainfall are primary determinants in triggering the onset of Sclerotinia blight of peanut in southeastern Virginia.

Key Words: *Sclerotinia minor*, plant growth, rainfall, temperature, epidemiology, disease forecasting.

Sclerotinia blight of peanut (*Arachis hypogaea* L.), caused by *Sclerotinia minor* Jagger, has become the most destructive disease of peanut in Virginia. The disease was first detected in the Virginia-North Carolina area in 1971 (12). Thereafter, the disease was found in the peanut-producing areas of Oklahoma (19) in 1972, and in Texas in 1981 (20). Trials in Virginia over a 4-yr period indicate that the disease claims an average of more than 32.9% of potential yield or 1598 kg/ha in naturally infested fields (18). These estimates were based on disease losses in plots treated and untreated with the experimental fungicide, fluazinam (3-chloro-N-[3-chloro-2, 6-dinitro-4-trifluoromethyl]-phenyl-5-trifluoromethyl-2-pyridinamine).

Over the last 24 yr, Sclerotinia blight has become a major concern of virtually every peanut grower in Virginia. Empirical notes along with several epidemiological studies have suggested that disease onset is triggered by environmental and host factors in the field. Both air and soil temperature along with available moisture have been implicated as primary factors in determining disease onset in the field (5). Vine growth or specifically the covering of middles between rows and the density of the foliar canopy are thought to be important host factors (3, 6). With regard to plant cultivars, it seems that the architectural characteristics of cultivars are responsible for much of the differences in susceptibility.

The purpose of this study was to identify the field conditions that act alone or in concert to trigger the onset of epidemics of Sclerotinia blight of peanut in Virginia. These definitions will provide a foundation for studies to develop algorithms for disease forecasting and an advisory program for application of fungicides.

Materials and Methods

Detailed records of disease onset in peanut fields at the Tidewater Center in Suffolk have been maintained since 1978. Beginning in June of each year, fields with histories of the disease were scouted every 10 to 14 d until the first week of July. Thereafter, scouting intervals were shortened to as little as 4 d until initial outbreaks were found. The areas scouted ranged from 0.8 to 1.6 ha. The scouting procedure consisted of walking across fields in a zigzag pattern. A T-shaped stick fabricated of either wood or PVC tubing was used to part vines and search for the initial symptoms and signs of disease beneath the foliar canopy. Different entry and exit points were used each time a field was scouted.

Peanut cultivars included Florigiant, NC 7 and NC 9 which are all susceptible to Sclerotinia blight. Rows were spaced 0.91 m apart and seed rates were *ca.* 123 kg/ha each year from 1978 to 1993. Planting dates were recorded and used to determine the time of disease onset in days after planting (DAP). Standard production practices as recommended by Virginia Cooperative Extension were followed each year. Observations of plant growth and canopy development were noted at disease onset. Thereafter, occurrences of defoliation by leaf spot diseases, corn earworms or other factors that might influence disease progress were also noted.

Weather data were collected adjacent to each field or within a 6.4-km distance of the field over a 13-yr period (1981-1993). Soil and air temperatures were obtained from records of the Virginia Tech/USDA Agro-Environmental Monitoring System (11, 17). Temperature sensors were located 10 cm below and 30 cm above fescue that was maintained by regular clipping. Daily maximum and minimum temperatures for soil and air were determined on the basis of readings at 10-min intervals each day. Rainfall was monitored continuously by the system. A NOAA weather station at the Tidewater Center served as a backup source of data.

Data were summarized using Microsoft® Excel on a 486 personal computer. Weather data were tabulated for the 30-d period preceding outbreaks of Sclerotinia blight from

[1]Contribution from Tidewater Agric. Res. & Ext. Center, Virginia Polytech. Inst. & State Univ., Suffolk, VA.

[2]Prof., Plant Pathology, Tidewater Agric. Res. & Ext. Center, 6321 Holland Rd., Suffolk, VA 23437.

1981 to 1993, except soil temperature data were not available in 1982. These summaries were then used to make inferences about the role(s) of environmental and host factors in triggering disease outbreaks.

Results and Discussion

Disease Onset. Planting dates over the 16-yr period (1978-1993) ranged from 1 May to 20 May, and averaged 10 May. The initial onset of disease ranged from 10 July to 7 Sept., or 62 to 120 DAP. The mean date of disease onset was 28 July or 79 DAP (Fig. 1). The median was 25 July or 76 DAP. The wide variation in time of disease onset makes the use of DAP of little value in scheduling the first application of fungicide for disease control.

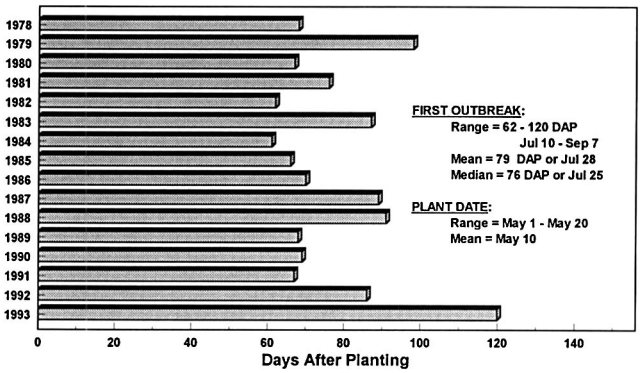

Fig. 1. A historical summary of the initial onset of Sclerotinia blight of peanut in Virginia (1978-1993).

These data provided justification for past recommendations to scout fields weekly and apply fungicide at the initial onset of disease. The data also demonstrated the need for development and implementation of a predictive advisory that would enable growers to apply a protective fungicide at the time when host and environmental factors are likely to trigger growth and infection processes of *S. minor* on peanut.

Vine Growth. Outbreaks of Sclerotinia blight occurred when vines in adjacent rows were <15 cm from touching or after the vines had overlapped between rows. At this time, plants had developed a canopy of foliage that shaded the soil surface in the pegging zone where stems were in contact with soil. Thereafter, disease progress was slowed or even stopped in some instances, if plants became defoliated by early leaf spot or corn earworms. Lateral limbs, leaflets, and pegs either in contact or close proximity to soil were initial sites of symptoms and signs of disease. In all cases, the infection sites were located beneath a dense foliar canopy that restricted air movement and shaded the infection site from direct sunlight.

Dow *et al.* (5) reported that the central main stem and lateral branches of peanut plants were equally susceptible to infection and colonization by *S. minor*. Their studies also demonstrated that relative humidities from 95 to 100% were required for germination of sclerotia on the soil surface. Thinning plants reduced canopy density, light interception by leaves, the frequency of initial infections, and the rate of tissue colonization. The importance of plant canopy development in disease onset was also suggested by a report that reduced seed rates and late plantings delayed the initial onset of disease in naturally infested fields (10). Brenneman *et al.* (2) reported that the age and developmental state of the lateral limbs of plants had a marked effect on colonization by *S. minor*. Succulent tissues near the growing tips of branches were the most rapidly colonized, while the more woody stems near the crown of plants were colonized at a slower rate. These studies also demonstrated that wounding tissue caused a dramatic increase in the frequency of infection. Additional evidence that plant growth and canopy density are important factors in the epidemiology of Sclerotinia blight was reported in studies of aerial infrared imagery of peanut fields (14). These images indicated that Sclerotinia blight was less severe in areas of sparse vegetative growth. Where plant growth was abundant, disease was most severe and readily associated with vine injury caused by tractor tires. In dry edible beans, dense vegetative growth has been shown to enhance the severity of disease caused by *S. sclerotiorum* (Lib.) de Bary (1). Small plants with open canopies were less susceptible than large plants with dense canopies. Plants with open canopies had a warmer and drier microclimate within the plant canopy as a result of more air circulation and light penetration, whereas plants with dense canopies had a cooler and wetter microclimate because of less air circulation and light penetration. The observations of Sclerotinia blight of peanut over a 16-yr period in the current study agree closely with those of others (3, 6, 10, 14) which collectively provide evidence that plant growth has a primary role in determining the time of disease onset.

Rainfall. Average daily rainfall over the 13-yr period from 1981 to 1993 showed a trend for heaviest accumulations from 3 to 17 d before disease onset (Fig. 2). Annual accumulations over 5-d intervals preceding disease onset showed the greatest levels occurred in the periods from 6 to 10 d and 11 to 15 d before disease onset (Table 1). However, no rainfall in the 6- to 15-d period before disease onset was recorded in 1983 or 1987. In 1983, disease onset was delayed to 8 Aug. or 87 DAP when vines were overlapping between rows. Rainfall was recorded only on day 3 (1.04 cm), day 16 (1.19 cm) and

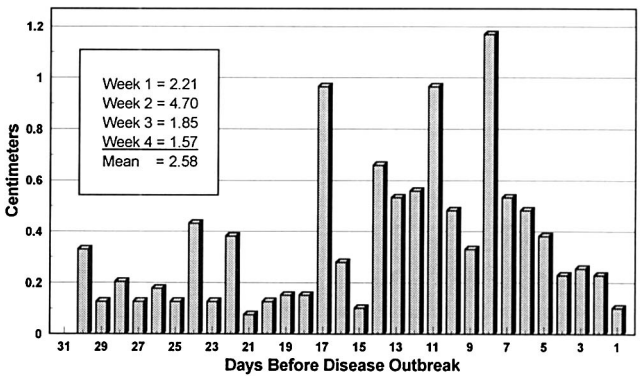

Fig. 2. The distribution of rainfall preceding outbreaks of Sclerotinia blight of peanut in Virginia (1981-1993).

Table 1. Rainfall accumulations preceding outbreaks of Sclerotinia blight (1981-1993).

Year	Accumulated rainfall on days					
	1-5	6-10	11-15	16-20	21-25	26-30
	cm					
1981	0.36	2.36	0.74	0.00	6.20	0.10
1982	0.23	1.91	3.76	0.94	0.84	0.08
1983	1.04	0.00	0.00	2.79	0.00	0.10
1984	2.24	4.45	4.17	4.98	0.18	2.18
1985	0.51	5.59	1.27	0.61	0.00	2.16
1986	0.38	0.66	8.20	0.99	0.00	1.50
1987	0.00	0.00	0.00	4.45	0.00	1.32
1988	1.30	4.24	6.55	0.00	0.58	0.00
1989	1.91	10.77	3.05	2.54	4.42	3.00
1990	0.00	1.78	3.45	0.56	1.19	0.00
1991	4.60	1.93	3.25	0.00	1.19	1.78
1992	1.04	2.03	2.21	4.06	0.00	0.71
1993	1.91	3.00	0.00	0.00	0.03	0.00
Avg	1.19	2.98	2.82	1.69	1.13	0.99

[a]Rainfall was measured by weather stations in the vicinity of fields with a history of sclerotina blight. Data are accumulations in the 5-d periods specified.

Table 2. Maximum and minimum temperatures of air and soil preceding outbreaks of Sclerotinia blight in Virginia (1981-1993).[a]

Days before disease onset	Obs.	Maximum			Minimum		
		Mean	S.D.	Median	Mean	S.D.	Median
	no.	C			C		
		Air temperature					
1-5	13	31.4	2.32	31.9	19.7	1.06	19.9
6-10	13	32.1	1.79	32.1	20.2	1.17	20.0
11-15	13	31.8	1.30	31.7	19.7	2.05	20.1
16-20	13	31.6	2.12	31.9	18.6	2.48	17.2
21-25	13	31.7	2.66	32.0	19.5	2.22	19.8
26-30	13	30.8	1.42	30.9	17.7	2.48	18.0
		Soil temperature					
1-5	12	29.6	1.39	29.8	25.1	0.81	25.3
6-10	12	29.9	1.61	29.8	25.1	0.76	25.2
11-15	12	29.3	1.65	29.1	24.8	0.98	25.1
16-20	12	29.2	2.30	28.6	24.3	1.41	24.5
21-25	12	28.4	1.38	28.2	23.8	1.47	23.8
26-30	12	28.0	1.62	27.7	22.6	1.92	23.0

[a]Air and soil temperature were measured 30 cm above and 10 cm below, respectively, below managed turf in the vicinity of peanut fields having a history of Sclerotinia blight. Data included the 13-yr period from 1981 to 1993, except soil temperatures, which were not available for 1982.

day 20 (1.6 cm) before disease onset. In 1987, heavy rain occurred only during the 16- to 20-d period (4.45 cm) and 26- to 30-d period (1.32 cm) before disease onset. The initial outbreak in 1987 was detected on 31 July or 89 DAP when vines had lapped in the middles between rows. Since a full foliar canopy was present in both years, perhaps heavy dews and/or extended periods of RH ≥ 95% had a role in triggering sclerotial germination and mycelial growth by *S. minor*.

Porter *et al.* (15) observed five times as many lateral branches of peanuts infected with *S. minor* in irrigated as compared to nonirrigated plots. Frequent rainfall and/or irrigation are thought to provide for a denser plant canopy, reduced temperatures in the plant canopy and soil, and an increase in exudates from senescing leaflets at the soil surface and beneath the plant canopy (1, 4, 7, 16). Hau *et al.* (7) showed sclerotial germination by *S. minor* was triggered by the release of volatile stimulates from remoistened peanut leaves. New flushes of succulent plant growth at branch tips after irrigation are also a likely source of increased vulnerability to infection by *S. minor* (2, 15). Observations in the current study and those of others (5, 6, 12) have indicated that mycelia from germinating sclerotia at the soil surface and near plant tissues (branches, pegs, leaves, etc.) were responsible for initiating infections in peanut fields.

Soil and Air Temperature. Maximum and minimum air temperatures averaged *ca.* 32 C and 20 C in the 15-d period prior to disease onset, respectively (Table 2). Maximum and minimum soil temperatures at the 10-cm depth averaged *ca.* 30 and 25 C, respectively, during this same period. As previously stated, these data were collected above and below managed turf. Weather monitors were not placed in peanut fields because of the spatial incompatibility with equipment used in performing standard crop management practices.

Imolehin *et al.* (8) reported that sclerotial germination and mycelial growth by *S. minor* on potato dextrose agar (PDA) occurred from 6 to 30 C. The optimum was between 12 and 24 C with minimal growth occurring at 6 and 30 C. Sclerotial germination and infection of lettuce tissue by hyphae of *S. minor* were observed at temperatures between 12 and 24 C. Similar studies with peanut isolates of *S. minor* have indicated that the cardinal temperatures for mycelial growth can vary according to the growth medium (20). The optimum for mycelial growth was 24 to 26 C on PDA, and 18 to 22 C on cornmeal agar and Czapek-Dox agar. The optimum for sclerotial germination varied from 18 to 26 C depending on the growth medium and isolate. In comparisons of fungicide-sensitive and insensitive isolates of *S. minor* from peanut fields in Virginia, maximum mycelial growth of all isolates on PDA occurred at 21 C (13). Growth was partially inhibited at 27 C and only traces of growth were measured at 30 C. Additional studies with peanut isolates of *S. minor* showed that optimum germination of sclerotia occurred at 20 to 25 C (5). Infection of peanut leaflets was better at 20 C than 15 or 25 C, and negligible germination and infection occurred at 30 C. Lee *et al.* (9) have speculated that *S. minor* is inactive in peanut fields after soil temperatures at the 5-cm depth exceed 28 C.

Based on ambient air temperatures in the 15 d preceding outbreaks of Sclerotinia blight from 1981 to 1993 in Virginia, it seems that air temperatures fluctuate between levels extremely favorable (20 C) and unfavor-

able (32 C) for growth of *S. minor*. Soil temperatures at a 10-cm depth beneath turf adjacent to peanut fields also fluctuated between levels somewhat favorable (25 C) and unfavorable (30 C) for growth of *S. minor*. Previous reports (1, 4, 16) have indicated that both plant growth, rainfall and irrigation can have a mitigating influence on temperatures beneath a dense foliar canopy in peanut fields. Sanders *et al.* (16) demonstrated that the temperature of the upper peanut canopy in Georgia was markedly affected by water stress. Late season canopy temperatures at 1 PM in plants without water stress averaged 28.5 C, while temperatures for plants under water stress averaged 35 C. Stem temperatures in water-stressed peanuts were 6 to 10 C higher than air temperatures in part due to exposure to direct solar radiation. However, stem temperatures were closely correlated with canopy temperatures in plants without water stress. Davidson *et al.* (4) reported that soil temperatures at the 5-cm depth under bare ground were similar to temperatures in the geocarposphere of peanuts until the canopy covered about 40% of the ground area. At this point, shading and transpiration by plants started cooling the geocarposphere below that of the bare ground temperature. When excessive vines and wet conditions lowered the maximum daily geocarposphere temperature below 27 C for periods of 3 to 7 d and below the dew point for >24 hr, growth of certain soilborne pathogens was noted. These pathogens included *Sclerotium rolfsii* Sacc. and *Rhizoctonia solani* Kuhn. High soil temperatures in the geocarposphere of peanuts were usually related to a small canopy or an extended interval between irrigation or rainfall.

Although both moisture and temperature are commonly mentioned as significant factors affecting development of diseases caused by *Sclerotinia* spp., results of the current study and others suggest that plant growth and rainfall are the primary forces responsible for triggering disease outbreaks in Virginia. The important role of rainfall is suggested by the elevated levels recorded during the period of 6 to 15 d prior to disease onset. Once the moisture requirements for sclerotial germination and mycelial growth of *S. minor* are met, temperature is likely the only remaining factor to be satisfied. In Virginia, temperature may be a limiting factor for disease when vines are >15 cm from touching between rows, or thereafter when plants are under moisture stress, and/or lack a dense foliar canopy for mitigation of temperatures at the infection court.

Summary and Conclusions

This study provided strong evidence that vine growth coupled with a dense foliar canopy and moist conditions were important components in the triggering mechanism for Sclerotinia blight of peanut. In addition to providing an abundance of sites for infection, plant growth was thought to be responsible for mitigation of otherwise unfavorable temperatures for *S. minor*. Free moisture for triggering sclerotial germination and subsequent mycelial infection of plants appeared to be provided by rainfall some 6 to 15 d before disease onset was visible. The results of this study have provided additional fundamental knowledge for developing algorithms to predict disease onset and the optimum timing of fungicide applications for disease management.

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