Effects of Crop Management on the Epidemiology of Southern Stem Rot of Peanut

B. B. Shew and M. K. Beute

Department of Plant Pathology, North Carolina State University, Raleigh 27650.
Journal Series Paper 8911 of the North Carolina Agricultural Research Service, Raleigh 27650.
The use of trade names does not imply either endorsement by the North Carolina Agricultural Research Service of the products named or criticism of similar ones not mentioned.
Accepted for publication 17 November 1983.

ABSTRACT

Shew, B. B., and Beute, M. K. 1984. Effects of crop management on the epidemiology of southern stem rot of peanut. Phytopathology 74:530-535.

The effects of moisture level, foliar mite and insect infestations, and leafspots on southern stem rot caused in peanut by *Sclerotium rolfsii* were examined during the summers of 1980, 1981, and 1982. Plants of peanut cultivar Florigiant were grown in field microplots (0.8 m in diameter) designed to exclude rainfall from August until digging (low moisture), or in plots that received natural rainfall (high moisture). Plots were sprayed or were not sprayed with an acaricide (dicofol in 1980 and propargite in 1981 and 1982), an insecticide (carbaryl), or a fungicide (chlorothalonil). Fungal inoculum was applied to all treatment combinations at initial densities of 10 or 100 sclerotia per plot. In all 3 yr, mean disease indices were greatest for high moisture + high inoculum plots, and least for low moisture + low inoculum plots. Within high moisture + high inoculum plots, treatments with dicofol in 1980 or with chlorothalonil in 1981 and 1982 increased incidence of stem rot. Highest disease incidence in all years was associated with treatments promoting development or maintenance of foliar canopy under the growing conditions of that year.

Additional key words: *Arachis hypogaea*, groundnut.

Southern stem rot caused by *Sclerotium rolfsii* Sacc. is a major disease of peanut (*Arachis hypogaea* L.) in North Carolina. Despite widespread occurrences, the epidemiology of southern stem rot on peanut is poorly understood and the sporadic outbreaks of disease are unpredictable (1).

Relationships between inoculum density and southern stem rot on peanut are not well defined. Difficulties in sampling caused by low and clustered sclerotial populations (16) limit usefulness of disease prediction based on sclerotial counts from soil samples, such as Leach (11) published for sugar beet. Furthermore, inoculations of plants with sclerotia of *S. rolfsii* in greenhouse or field tests often are unsuccessful, possibly because sclerotia produced in sterile culture for such inoculations are physiologically different from nonsterile sclerotia produced naturally (12). Recently, a method for production of nonsterile sclerotia in culture, which allows successful quantitative inoculation of peanut, has been developed (6).

Many researchers (1) have stated that moisture conditions are critical in the development of southern stem rot, but their conclusions are contradictory. Severe stem rot epidemics may occur in dry years (19), but epidemics are most often observed in wet years (1). Some researchers have suggested that alternating wet and dry periods enhance (6) or inhibit (17) disease development. Beute and Rodriguez-Kabana (6) proposed a possible mechanism for disease initiation during alternating dry and wet periods when they found that volatiles emanating from dried, remoistened peanut tissues stimulated germination of sclerotia of *S. rolfsii*.

Because remoistened peanut tissues stimulate sclerotial germination, and because the fungus uses these tissues (as well as other undecomposed organic debris) as a food base when attacking the plant (5,7), any stress that defoliates peanut plants could initiate or maintain stem rot epidemics. Among the biological stresses that defoliate peanuts are leafspot infections caused by *Cercospora aracadicola* Hori and *Cercosporidium personatum* (Berk and Curt) Deighton, feeding damage caused by chewing insect larvae, and infestations of two-spotted spider mites (*Tetranychus urticae* Koch). Ironically, chemical control of leafspots and insects on peanut favors increases in mite populations when certain fungicides (eg, chlorothalonil, benomyl, mancozeb, and copper + sulfur) or insecticides (eg, carbaryl and methomyl) are used (9). When fungicides and insecticides are used in the same field, mite populations may increase to levels that severely damage the crop. Environmental conditions during the growing season similarly influence leafspot, insect, and mite populations. Mite and insect problems occur in dry seasons, but leafspots prevail in moist seasons. Moisture conditions during the growing season may, therefore, influence stem rot epidemics directly by affecting sclerotial germination and growth, or indirectly by affecting populations of pests that defoliate plants.

The purpose of this research was to quantify the influence of inoculum density, plant or soil moisture conditions, leafspot control, insect control, and two-spotted spider mite control on the epidemiology of southern stem rot on peanuts.

MATERIALS AND METHODS

Establishment of microplots. One hundred twenty-eight microplots were established in March 1980 at the Upper Coastal Plain Research Station near Rocky Mount, NC. Fiberglass cylinders 80 cm in diameter and 60 cm high were buried 45–50 cm deep to form microplots (4). Soil type was a Shubuta gravelly sandy loam, pH 6.1. Soil fertility in microplots was adjusted prior to each growing season as recommended by the Agronomic Division, North Carolina Department of Agriculture, Soil Testing Laboratory. Twelve to 15 seeds of the cultivar Florigiant were inoculated with *Rhizobium* spp. and planted in each microplot on 19 May 1980, 26 May 1981, and 25 May 1982. Carbofuran (4.5 kg a.i./ha) and alachlor (3.3 kg a.i./ha) were applied at planting for nematode and insect control, and for weed control, respectively. After ~2 wk, plants were thinned to five per microplot. Gypsum (673 kg/ha) was applied at early-to-mid bloom.

Experimental design. The 2^5 factorial treatment design consisted of two levels (presence or absence) of three pesticides, initial inoculum levels of 10 or 100 sclerotia per microplot, and presence or absence of rain shields (covers) over the plots for a total of 32 treatment combinations. The four replicate treatments were arranged in a split-split plot design with presence or absence of covers as whole plots, and with initial inoculum level as the first subplot; pesticide treatments were randomized within covering by inoculum level combinations. Treatments were reestablished in the same microplots each year to examine cumulative effects of the

The publication costs of this article were defrayed in part by page charge payment. This article must therefore be hereby marked "*advertisement*" in accordance with 18 U.S.C. § 1734 solely to indicate this fact.

treatment combinations in continuous peanut culture.

Experimental pesticide treatments. Pesticides used to establish treatments and corresponding target organisms were chlorothalonil (1.8 L a.i./ha) for control of leafspots, dicofol (1.1 kg a.i./ha) in 1980 or propargite (1.7 kg a.i./ha) in 1981 and 1982 for control of two-spotted spider mites, and carbaryl (1.7 kg a.i./ha) for control of lepidopterous insect larvae. A hand-pumped garden sprayer was used to apply one pesticide at a time over the plant canopy within each microplot. Sprays were applied at approximately biweekly intervals. Number of sprays of pesticides and dates of first application for each year were as follows: acaricide applied five times beginning 5 August, fungicide applied six times beginning 2 July, insecticide applied five times beginning 16 July in 1980; acaricide applied two times beginning 31 August, fungicide applied six times beginning 9 July, insecticide applied four times beginning 30 July in 1981; acaricide applied once on 22 September, fungicide applied nine times beginning 22 June, insecticide applied twice beginning 25 August in 1982.

Production of inoculum. A culture of *S. rolfsii*, originally isolated from peanut, was grown on sterile oat grains for production of sclerotia. Colonized oat grains were incubated on moist, nonsterile soil, and sclerotia were collected, air-dried, and stored in the laboratory in capped glass vials. Viability of stored, nonsterile sclerotia was assayed prior to use by placing 25 or 50 sclerotia on the smoothed surface of 22 g of air-dried field soil, remoistened with 5.6 ml of 1% (v/v) aqueous methanol, in a 9-cm-diameter petri dish. Covered dishes were incubated in plastic bags for 48–72 hr, and germinated sclerotia were counted. Ten or 100 sclerotia of *S. rolfsii* were dispersed over the soil surface of microplots on 6 August 1980. Ten more sclerotia were added to all microplots on 22 July 1982 after no sclerotia were detected in soil assayed from low moisture + low inoculum plots in May 1982.

Establishment of moisture regimes. One-half of the microplots (high moisture treatment) received surface irrigation to supplement natural rainfall during the growing season. The remaining microplots (low-moisture treatments) were covered beginning in August to reduce moisture reaching the plots. Rain shields were constructed by attaching clear plastic film to a 107 × 107-cm wooden frame. The frame was supported 1 m above the ground by four posts placed at equal distances outside each microplot in such a way that the frame was held at a slight angle, which allowed run-off of rainfall from the cover. Plots were covered with frames on 6 August 1980, 30 July 1981, and 3 August 1982. Frames remained in place until peanuts were dug.

The amount of rainfall reaching noncovered plots from May through September was calculated from precipitation data from the Upper Coastal Plain Research Station weather station located ~400 m from the plots. Amount of rainfall reaching covered plots was calculated by assuming that no rain fell directly into these plots following covering.

Assessment of disease. Stem rot lesions per microplot were counted six times at approximately biweekly intervals after lesions were first seen. Stem rot lesions were characteristically brown-to-black regions which penetrated stems or crowns of peanut plants, causing a stringy dry rot. Signs (mycelium and sclerotia) were helpful in diagnosis, but were not present for every lesion counted. No more than one lesion per branch of a peanut plant was counted, and an infection present on a leaflet or peg, but not extending into a branch or the crown was not counted. Dead plants were estimated to have 12 lesions, the average number of branches per plant. Peanut plants were dug using a hand spade following the final disease assessment.

Percent defoliation (1981) or percent of the total canopy damaged by leafspots (1982) was visually estimated on stem rot assessment dates three through six. Percent foliage damaged by chewing insects and by mites was estimated on date six. Percent pod rot due to all causes, defined as [(number of pods or pegs with lesions)/(total pods and pegs produced)] × 100 was estimated after digging.

Assay of soil. Following digging, peanut vines were left to overwinter in microplots. The following spring vines were removed from plots to minimize accumulation of plant residues and soil was turned to a depth of 25–30 cm in preparation for planting. In 1982, soil samples were taken from microplots using a 2.5-cm-diameter soil probe inserted to a depth of 15 cm and cores were pooled to give a 1.5-kg sample per eight plots. Soil samples taken from microplots were assayed for viable sclerotia of *S. rolfsii* using a method modified from Rodriguez-Kabana and Beute (13). In the modified method, 1-kg (air-dried weight) soil samples were remoistened to ~−1/3 bar, stored 1–5 days, and processed using a semiautomatic elutriator (8). Organic debris and sclerotia collected on sieves with 420-μm opening during a 4 min elutriation and decanting cycle were rinsed with a 1% (v/v) aqueous methanol solution from sieves into 23 × 30-cm metal pans, which were lined with paper towels. Pans were enclosed in plastic bags, incubated 48 hr, and examined for germinating sclerotia.

Data analysis. Data were analyzed by analysis of variance (AOV) following appropriate transformations to normalize dependent variables and to stabilize variances. Significance of main and interaction effects of the experimental factors was evaluated by partitioning treatment sums of squares into appropriate components. Main and interaction effects were estimated from the AOV model, with presence or absence of a factor designated by a +1 or −1, respectively. Transformed data from subplots (moisture × inoculum levels) and disease data at different times were combined when variances across subplots or times were judged homogeneous by Bartlett's test (18). Otherwise, data were analyzed at the whole plot (moisture) or subplot (moisture × initial inoculum) level. Areas under disease progress curves (AUDPC) were calculated according to the formula:

$$\text{AUDPC} = \sum_{i=0}^{n} [(L_{ti+1} + L_{ti})\,(t_{i+1} + t_i)]/2$$

in which t_i = time in days, i = 0···n, and L_{ti} = number of lesions on day i. Leafspot epidemics (1982) were fitted by a polynomial regression model of percent leafspot (transformed by Sin^{-1} (percent leafspot/100)$^{1/2}$) versus time. Significance of linear and quadratic components and of lack-of-fit was evaluated by F-tests, and appropriateness of models was evaluated by inspection of plots of predicted values versus residuals (error). Similarly, regressions of lesion number (transformed) against time were used to compare epidemics of stem rot.

RESULTS

A severe drought occurred in North Carolina during 1980, with 31.6 cm total rainfall from 1 May to 30 September. Total water (rainfall and irrigation) reaching high moisture (noncovered) plots was 44.2 cm. Irrigation and rainfall directly reaching low moisture (covered) plots totaled 28.5 cm. Drought conditions favored severe mite damage, but little leafspot. In fungicide + insecticide treatments, from 40 to 95% defoliation due to mites was observed by 16 September, and five sprays were necessary to control mites in plots receiving acaricide (dicofol) treatments.

Total rainfall from 1 May to 30 September 1981 was 48.5 cm. Rainfall and irrigation totaled 50.4 cm in high moisture plots and water reaching low moisture plots directly totaled 28.4 cm. Although moisture levels in covered plots were very similar in 1980 and 1981, leafspots were relatively more severe than mite damage in all plots in 1981, probably because mite populations were lower and leafspot levels were higher in nearby fields compared to 1980 (Table 1). Pesticide treatments that included a fungicide suppressed leafspot damage recorded on day 74 (8 October, $P = 0.01$). Slight decreases in leafspot damage also were present in combinations that included insecticide ($P = 0.01$), but fungicide and insecticide together did not suppress leafspot as much as would be predicted from their main effects. Treatments that included a fungicide or an insecticide enhanced mite damage in high moisture plots and greatly enhanced damage in low moisture plots. When insecticide and fungicide were applied together in low moisture plots, more mite damage occurred than would be predicted from the main effects of the pesticides (Table 1). Because most defoliation was caused by leafspots, effects of pesticides on percent defoliation on days 30 through 74 generally corresponded to effects of pesticides

on percent leafspots observed on day 74.

From 1 May to 30 September 1982, 59.7 cm rain fell. Rainfall and irrigations totaled 61.2 cm in high moisture plots, and 39.1 cm rainfall reached low moisture plots directly. Leafspot epidemics were severe for treatment combinations that did not include fungicide. For example, the regression estimate of percent leafspot on the first day of leafspot assessment (31 August) in acaricide only and acaricide + insecticide plots was 31%. Fungicide only and fungicide + insecticide treatments slowed progress of leafspot epidemics. In low moisture plots, acaricide suppressed and insecticide or fungicide enhanced mite damage (average increase of 2 or 1%, respectively). Mean mite damage was 12%. In high moisture plots, treatments containing fungicide increased mite damage by an average of 1% over the high moisture mean of 6%.

Stem rot lesions were first observed in high moisture plots on 6 September 1980, 27 July 1981, and 4 August 1982 (day one). Little stem rot, as represented by AUDPC, was present in low moisture or high moisture plots infested with 10 sclerotia per plot, or in low moisture plots infested with 100 sclerotia per plot (Table 2). Pesticides did not affect stem rot in low moisture + low initial inoculum plots in any year ($P = 0.05$). In 1980, nonsprayed peanuts in high moisture plots infested with 10 sclerotia per plot had a much greater disease incidence than did peanuts sprayed with any pesticide combination (Table 3). Pesticides did not affect disease in high moisture + low initial inoculum or in low moisture + high initial inoculum plots in any other instance. Disease in these plots was similar in all 3 yr. On the average, greatest amounts of disease occurred in high moisture plots infested with 100 sclerotia per plot (Table 2), and pesticides influenced disease in these plots in 1980 and 1981 (Table 3).

Although common regression equations were not estimated due to unequal variances, results of regressions of ln(lesion number + 0.01) + 4.61 on ln time (time = 1 was dropped) in high moisture + low inoculum and low moisture + high inoculum plots were similar in 1981 and 1982. All values had 4.61 added to them to avoid negative numbers which resulted from taking logarithms of numbers <1. Equations fitted to disease progress data of 1981 were $Y = -5.29 + 2.11(\ln \text{time})$ for high moisture + low inoculum plots and $Y = -4.84 + 2.15(\ln \text{time})$ for low moisture + high inoculum plots. Treatments included in the linear regression for high moisture + low inoculum plots were acaricide only, fungicide + acaricide, and fungicide + insecticide. A separate regression, $Y = -35.09 + 19.83(\ln \text{time}) - 2.32(\ln \text{time})^2$ was fitted to disease progress data for the control treatment in high moisture + low inoculum plots. Pesticides did not affect regression in high moisture + low inoculum or low moisture + high inoculum plots in 1982.

The relationship between ln time (time = 1 was dropped) and ln(lesion number + 0.01) + 4.61 was highly significant in high moisture + high inoculum density treatments in 1980 and 1982 (Table 4). In 1981, regressions of $(\text{lesion number})^{1/2}$ on $(\text{time})^{1/2}$ were fitted to disease progress data for high moisture, high inoculum plots (Table 4).

In high moisture + high inoculum plots in 1980, disease

TABLE 1. The effect of three pesticide treatments on mite damage and leafspot incidence on peanut in microplots in 1981

Pesticide[a]	Mite damage		Leafspot
	Low moisture	High moisture	
Acaricide (A)	2[b]	0	63
Insecticide (I)	1	1	53
Fungicide (F)	6	3	7
F + A	9	4	6
I + A	1	0	50
I + F	46	8	7
F + I + A	37	8	5
None	1	0	68

[a] Acaricide = propargite, insecticide = carbaryl, fungicide = chlorothalonil.
[b] Percent of the canopy affected by mite damage and leafspot on day 74. Values presented were obtained from reverse arcsin transformation of means.

TABLE 2. Influence of moisture level and of initial inoculum density of *Sclerotium rolfsii* on mean area under the disease progress curve (AUDPC) of southern stem rot on peanut

Moisture level	Sclerotia per plot[a]	AUDPC + s.e.[b]			Lesion number[c]		
		1980	1981	1982	1980	1981	1982
Low	10	51 ± 45	30 ± 27	9 ± 5	1	1	1
Low	100	90 ± 54	168 ± 77	119 ± 47	2	5	5
High	10	113 ± 37	172 ± 58	92 ± 15	2	5	6
High	100	774 ± 102	647 ± 58	208 ± 41	12	17	5

[a] Average number of lesions per microplot on final assessment date.
[b] AUDPC over six assessment dates (s.e. = standard error).
[c] Microplots were infested with the indicated number of sclerotia in August 1980.

TABLE 3. Influence of three pesticides and initial inoculum density of *Sclerotium rolfsii* on area under the disease progress curve (AUDPC) of southern stem rot on peanut grown in high moisture microplots in 1980 and 1981

Pesticide[a]	1980		1981
	10 sclerotia per plot	100 sclerotia per plot	100 sclerotia per plot
Acaricide (A)	109 ± 78[b]	1,522 ± 379	355 ± 250
Insecticide (I)	4 ± 4	517 ± 304	254 ± 105
Fungicide (F)	45 ± 22	688 ± 380	1,161 ± 101
F + A	94 ± 47	823 ± 373	796 ± 158
I + A	24 ± 14	1,485 ± 256	758 ± 236
I + F	7 ± 7	179 ± 62	555 ± 78
F + I + A	0 ± 0	616 ± 298	639 ± 332
None	618 ± 263	364 ± 114	355 ± 62

[a] Acaricide = dicofol in 1980 and propargite in 1981, insecticide = carbaryl, and fungicide = chlorothalonil.
[b] AUDPC calculated over six assessment dates. Values are AUDPC ± standard error.

TABLE 4. Influence of acaricide, insecticide, and fungicide on progress of stem rot epidemics on peanut grown in microplots. Regression equation was lesion number = $b_0 + b_1$ (time) + b_2 (time)^2

Treatment	b_0	b_1	b_2[a]
1980			
Acaricide (A)	−0.990	4.323	−0.528
Insecticide (I)	−0.656	1.671	–
Fungicide (F)	−1.170	1.671	–
Control	−0.732	1.671	–
I + A	1.280	1.671	–
F + A	4.655	0.578	–
F + I	1.401	0.578	–
F + I + A	3.019	0.578	–
1981			
Acaricide	−0.707	0.514	–
Insecticide	−1.833	0.514	–
I + A	−0.201	0.514	–
Fungicide	−9.784	3.641	−0.220
F + A, F + I + A	−2.019	0.753	–
F + I	−3.583	0.930	–
Control	−7.137	2.393	−0.142
1982			
Fungicide	−5.535	2.162	–
Control	−4.814	2.162	–
F + I + A	−2.738	2.162	–
I, I + A, F + I	−6.769	2.946	–
F + A	−8.923	3.737	–

[a] Lesion number was transformed by ln(lesion number + 0.01) + 4.61 (to avoid negative values) in 1980 and 1982, and by $(\text{lesion number})^{1/2}$ in 1981 to stabilize variances. Time was transformed to ln time in 1980 and 1982, and to $(\text{time})^{1/2}$ in 1981 to linearize data. b_2 was estimated only when there was a significant ($P < 0.05$) quadratic effect.

progressed most slowly in plots receiving fungicide + insecticide sprays (Fig. 1). Disease progressed at a similar rate on plants in plots treated with fungicide + insecticide + acaricide, or fungicide + acaricide, but more lesions were present in these plots early in the epidemic. Stem rot progressed more rapidly in plots treated with fungicide only, no pesticide, insecticide only, and insecticide + acaricide. Among these treatments, lesion number on day 11 (16 September) was least in fungicide only treatments and greatest in insecticide + acaricide treatments.

In 1981, disease progressed most slowly in plots treated with insecticide only, acaricide only, or insecticide + acaricide (Fig. 2). Among these treatments, least disease was initially present in insecticide only plots, and most disease in insecticide + acaricide plots. Disease progressed more rapidly in plots treated with fungicide + acaricide and fungicide + insecticide + acaricide. Lesion number on day 17 (12 August) was least on plants in plots treated with fungicide + insecticide, but disease progressed more rapidly in these plots than in any other treatment. Although positions of regression lines for control and fungicide treatments were much different, the curve shapes were very similar, with a peak in the rate of disease increase late in the epidemic.

In high moisture + high inoculum plots in 1982, disease progressed most slowly in control, fungicide + insecticide + acaricide, and fungicide only treatments, with least disease on day 17 in nontreated plots and most disease in plots treated with fungicide (Fig. 3, Table 4). Stem rot increased most rapidly in plots treated with fungicide + acaricide. No regression could be fitted to disease progress data for acaricide only data.

Pod rot incidence was affected by moisture and inoculum level in 1980 and 1982 ($P = 0.05$). Plants in low moisture plots had more pod rot than plants in high moisture plots, and plants in plots initially infested with 100 sclerotia had more pod rot than plants in plots initially infested with 10 sclerotia. In 1981, pot rot in microplots was suppressed by treatments that included insecticide ($P = 0.05$). Average pod rot was 9% in 1980, 10% in 1981, and 4% in 1982.

An average of 3.3 sclerotia per kilogram of air-dried soil was recovered from high moisture + high inoculum plots in March 1982. An average of 0.5 sclerotia per kilogram of soil was recovered from high moisture + low inoculum plots, and 0.8 sclerotia per kilogram of soil were recovered from low moisture + high initial inoculum plots. No sclerotia were detected in low moisture + low inoculum plots. Corresponding numbers of sclerotia per plot (assuming the 80-cm-diameter × 15-cm-deep volume of soil sampled had a bulk density of 1.35 g/cm^3) were 337 for high moisture + high inoculum plots, 51 for high moisture + low inoculum plots, and 82 for low moisture + low inoculum plots.

DISCUSSION

Moisture levels and the number of sclerotia added to plots in 1980 were very important in determining total amount of stem rot present over the 3 yr of the experiment. A high level of initial inoculum and high moisture conditions were necessary for maximum disease development. Very little stem rot occurred in low moisture + low inoculum plots, but inoculum apparently survived at least 1 yr under these conditions, because in 1981, disease was found in plots where no disease was found in 1980. The effect of low levels of inoculum was partially offset in high moisture plots, and high inoculum levels partially compensated for inhibition of disease development in low moisture plots. Compensation among factors favorable to disease has been reported in other pathosystems (14).

Low moisture and high moisture plots were irrigated differentially, and covers acted as a barrier to rain falling directly

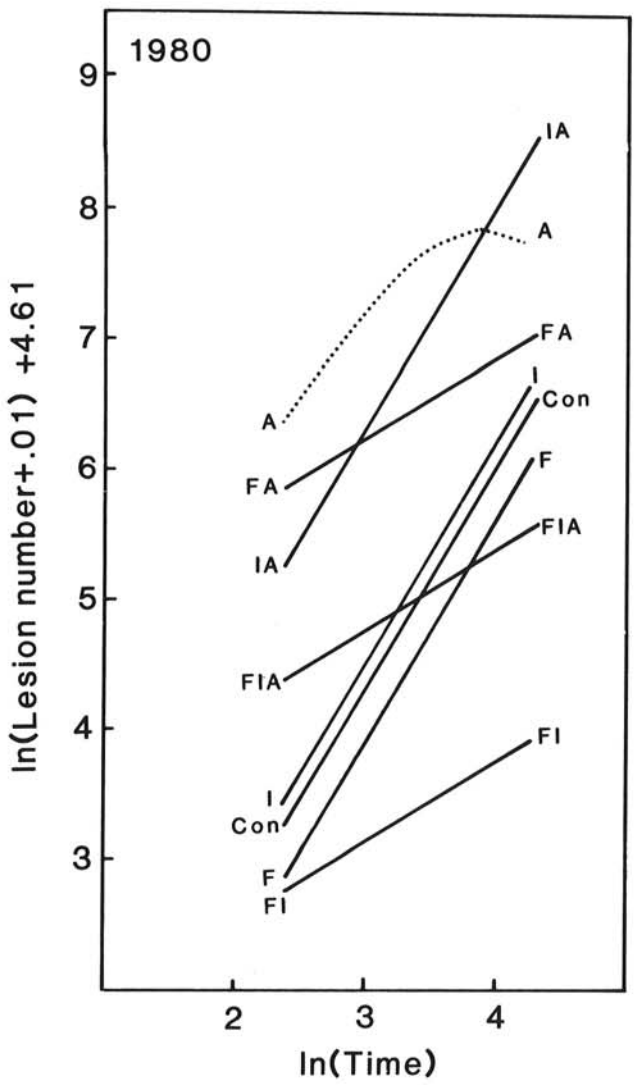

Fig. 1. Progress of southern stem rot on peanut caused by *Sclerotium rolfsii* in 1980. Plots are of regressions fitted to ln(lesion number + 0.01) + 4.61(4.61 is −ln 0.01) versus ln time in high moisture + high inoculum plots. Time in days from first disease assessment on 6 September. Pesticide treatments are indicated by A = acaricide, F = fungicide, I = insecticide, and CON = no pesticide.

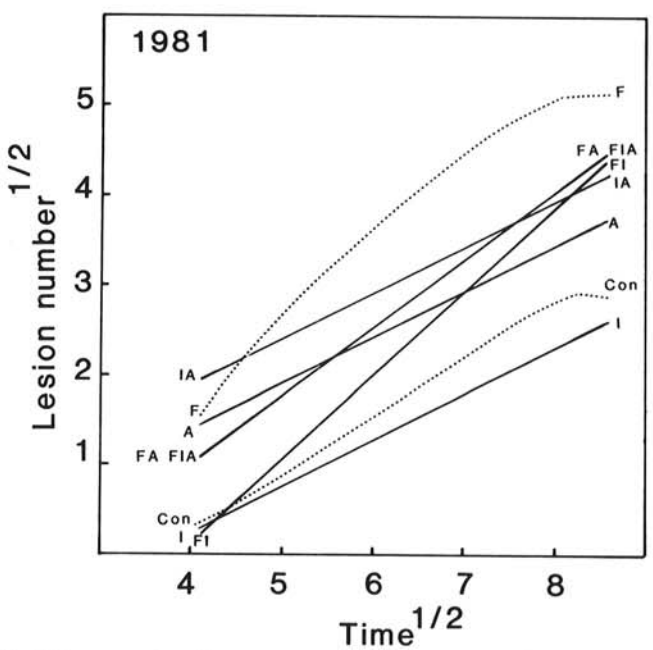

Fig. 2. Progress of southern stem rot caused in peanut by *Sclerotium rolfsii* in 1981. Plots are of regressions fitted to (lesion number)$^{1/2}$ versus (time)$^{1/2}$ in high moisture + high inoculum plots. Time expressed in days from first disease assessment on 27 July. Pesticide treatments are indicated by A = acaricide, F = fungicide, I = insecticide, and CON = no pesticide.

into plots from August until digging. Inconsistent differences in measured plant water potentials (*unpublished*), and the lack of association between leaf shedding and covering alone (which would indicate drought stress in covered plots) suggests that some water from rainfalls was available through capillary action to peanuts growing in covered plots. Additional water probably reached covered plots when high winds accompanied rainfall. Apparently, large differences in moisture levels in the upper 7.5 cm of soil (for example, −1 bar versus −10 bars average soil moisture potential measured on five dates in September 1982 in noncovered and covered plots, respectively; *unpublished*) and in the plant canopy resulted in large differences in the amount of stem rot that developed in covered and noncovered plots.

Greatest amounts of pod rot were found in low moisture + high inoculum plots in 1980 and 1982. In 1981, insecticide inhibited pod rot, perhaps by limiting pod injuries by insects. These results are consistent with frequent associations made between pod rot and dry growing seasons (J. E. Bailey, Department of Plant Pathology, North Carolina State University, Raleigh; *personal communication*).

Cool temperatures limit development of stem rot epidemics (1). Cool temperatures (mean daily maximum = 28 C and mean daily minimum = 16 C) during August and September 1982 probably depressed maximum disease levels, narrowing the difference between epidemics in high moisture + high inoculum and intermediate plots. Furthermore, a smaller relative difference in moisture reaching covered and noncovered plots during 1982 may have contributed to the greater similarity in disease levels between subplots.

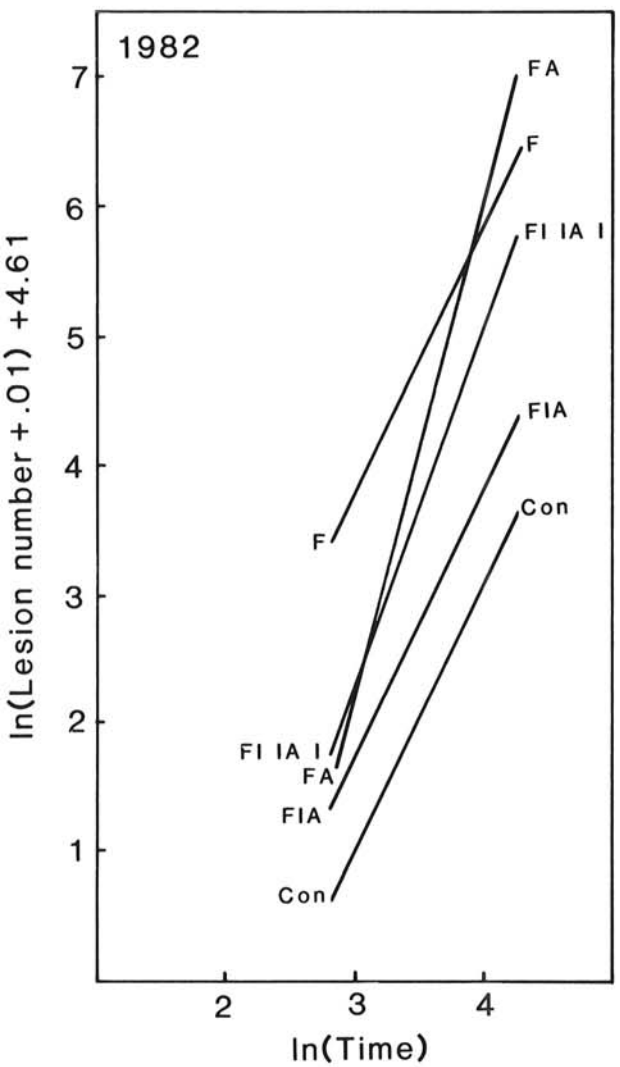

Fig. 3. Progress of southern stem rot on peanut caused by *Sclerotium rolfsii* in 1982. Plots are of regression of ln(lesions + 0.01) + 4.61 versus ln time for high moisture + high inoculum plots. Time expressed in days from first disease assessment on 4 August. Pesticide treatments are indicated by A = acaricide, F = fungicide, I = insecticide, and CON = no pesticide.

Because a food base is usually necessary for *S. rolfsii* to infect a host (7) and because remoistened dried peanut tissues stimulate sclerotial germination (6), researchers expect stem rot to be most severe when biological or physical stresses cause defoliation (10). In Alabama, however, more stem rot occurred in a dry season when leafspots (which defoliate peanut) were controlled (3). In our experiments, development of stem rot in high moisture + high inoculum plots was enhanced by treatments that inhibited defoliation. For example, percent control of leafspots was positively correlated with number of stem rot lesions in 1981 ($r = 0.37$, $P < 0.05$). A dense foliar canopy was found to enhance white mold caused by *S. sclerotiorum* on bean (15). Apparently, a moist microenvironment was necessary for the fungus to colonize senescent blossoms prior to infection of the host. Similarly, high humidity within intact peanut canopies may have favored colonization of food bases and infection following germination of sclerotia of *S. rolfsii*.

In 1980, disease incidence in high moisture + low inoculum plots receiving no pesticides was greater than in pesticide-treated plots. Under the growing conditions in 1980, plants in control plots retained most of their leaves. In 1981, however, disease in the high moisture + low inoculum treatment was again greatest in plots that received no pesticide, even though peanuts in these plots were severely defoliated by leafspot. Better survival of *S. rolfsii* in unsprayed plots, regardless of defoliation severity, could explain enhanced stem rot severity in control treatments having low inoculum densities because *S. rolfsii* is known to be slightly sensitive to chlorothalonil (3) and insecticides (2). The relatively weak effects of these chemicals on *S. rolfsii* would be more difficult to detect in high inoculum plots.

Stem rot incidence increases under continuous peanut culture (10). We found that, in addition to pesticide effects on the canopy, disease severity early in the epidemic in 1981 and 1982 was affected by severity of disease in the previous year. High incidence of disease in 1 yr was followed by higher initial disease incidence in the following year. In 1981, for example, the initial lesion numbers were lowest in insecticide only plots (insecticide plots had little disease in 1980) and highest in insecticide + acaricide plots, which had the greatest amount of stem rot in 1980. In either year, however, canopy microenvironment could alter the influence of earlier epidemics. In 1981, for example, plants treated with fungicide + insecticide had the least disease early in the epidemic, reflecting little disease in 1980, but the fastest rate of disease increase, reflecting conditions very favorable for stem rot in fungicide + insecticide plots in 1981.

Our results indicate that incidence of stem rot is greatest when inoculum is abundant, moisture is adequate, and defoliating pests are controlled. Volatiles originating from defoliated leaves, and from other sources as well, apparently are present at levels sufficient to stimulate sclerotial germination within intact peanut canopies. Management and environmental factors probably influence stem rot epidemics more strongly through their effects on host infection and colonization processes than on sclerotial germination. Therefore, maintenance of the canopy and a moist microclimate enhances the development of stem rot epidemics when neither moisture nor inoculum is limiting.

LITERATURE CITED

1. Aycock, R. 1966. Stem rot and other disease caused by *Sclerotium rolfsii*. N. C. Agric. Exp. Stn. Tech. Bull. 174. 202 pp.
2. Backman, P. A., and Hammond, J. M. 1981. Suppression of peanut stem rot with the insecticide chlorpyrifos. Peanut Sci. 8:129-130.
3. Backman, P. A., Rodriguez-Kabana, R., and Williams, J. C. 1975. The effect of peanut leafspot fungicides on the nontarget pathogen, *Sclerotium rolfsii*. Phytopathology 65:773-776.
4. Barker, K. R., Daughtry, B. I., and Corbett, D. W. 1979. Equipment and techniques for establishing field microplots for the study of soilborne pathogens. J. Nematol. 11:106-107.

5. Beute, M. K., and Rodriguez-Kabana, R. 1979. Effect of volatile compounds from remoistened plant tissues on growth and germination of sclerotia of *Sclerotium rolfsii*. Phytopathology 69:801-805.
6. Beute, M. K., and Rodriguez-Kabana, R. 1979. Effect of wetting and the presence of peanut tissues on germination of sclerotia of *Sclerotium rolfsii* produced in soil. Phytopathology 69:869-872.
7. Boyle, L. W. 1956. Fundamental concepts in the development of control measures for southern blight and root rot on peanuts. Plant Dis. Rep. 40:661-665.
8. Byrd, D. W., Jr., Barker, K. R., Ferris, H., Nusbaum, C. J., Griffin, W. E., Small, R. H., and Stone, C. A. 1976. Two semi-automatic elutriators for extracting nematodes and certain fungi from soil. J. Nematol. 8:206-212.
9. Campbell, W. V. 1978. Effect of pesticide interactions on the two-spotted spider mite on peanuts. Peanut Sci. 5:83-86.
10. Garren, K. H. 1961. Control of *Sclerotium rolfsii* through cultural practices. Phytopathology 51:124-128.
11. Leach, L. D., and Davey, A. E. 1938. Determining the sclerotial population of *Sclerotium rolfsii* by soil analysis and predicting losses of sugarbeets on the basis of these analyses. J. Agric. Res. 56:619-631.
12. Linderman, R. G., and Gilbert, R. G. 1973. Behavior of sclerotia of *Sclerotium rolfsii* produced in soil or culture regarding germination, stimulation by volatiles, fungistasis, and sodium hypochlorite treatment. Phytopathology 63:500-504.
13. Rodriguez-Kabana, R., Beute, M. K., and Backman, P. A. 1980. A method for estimating number of viable sclerotia of *Sclerotium rolfsii*. Phytopathology 70:917-919.
14. Rotem, J. 1978. Climatic and weather influences on epidemics. Pages 317-337 in: Plant Disease: An Advanced Treatise, Vol. 2. J. G. Horsfall and E. B. Cowling, eds. Academic Press, New York. 436 pp.
15. Schwartz, H. F., Steadman, J. R., and Coyne, D. D. 1978. Influence of *Phaseolus vulgaris* blossoming characteristics and canopy structure upon reaction to *Sclerotinia sclerotiorum*. Phytopathology 68:465-470.
16. Shew, B. B. 1983. The epidemiology of southern stem rot caused by *Sclerotium rolfsii* on peanut. Ph.D. thesis, North Carolina State University, Raleigh. 95 pp.
17. Smith, A. M. 1972. Drying and wetting of sclerotia promotes biological control of *Sclerotium rolfsii* Sacc. Soil Biol. Biochem. 4:119-123.
18. Snedecor, G. W., and Cochran, W. G. 1967. Statistical Methods, 6th ed. The Iowa State University Press, Ames.
19. Watkins, G. M. 1961. Physiology of *Sclerotium rolfsii* with emphasis on parasitism. Phytopathology 51:110-113.