Peanut Science (1979) 6, 77-79

Sclerotinia Blight of Peanuts in Oklahoma and Occurrence of the Sexual Stage of the Pathogen[1]

D. F. Wadsworth[2]

ABSTRACT

Sclerotinia blight of peanuts was discovered in Oklahoma in 1972. The disease is caused by *Sclerotinia sclerotiorum* (Lib.) de Bary varieties "Minor" and "Major" Purdy (*Whetzelinia sclerotiorum* (Lib.) Korf & Dumont). *S. sclerotiorum* "Minor" is more prevalent. Sclerotinia blight has been found in seven of the 23 major peanut-producing counties in Oklahoma since its discovery and it is regarded as a major peanut disease. Both fungal varieties overwintered in the field as sclerotia and produced apothecia and ascopores from late April to early June. Peanut plants exposed to natural ascospore discharges in the field became infected with both varieties of the fungus.

Key Words: *Arachis hypogaea*, *Sclerotinia sclerotiorum* "Minor" *Sclerotinia sclerotiorum* "Major", apothecial stage, *Whetzelinia sclerotiorum*.

A stem blight of a Spanish type peanut (*Arachis hypogaea* L.) was observed in irrigated fields in the Canadian River Valley near Prague, Oklahoma in 1972 (9). Diseased plants had small black sclerotia on and/or within dead shredded stems, but no other stage of the pathogen was found. Porter and Beute (4, 5) described a similar disease of peanuts found in Virginia and North Carolina in 1971 and 1972. They identified the pathogen as *Sclerotinia sclerotiorum* (Lib.) de Bary (*S. minor* Jagger), small sclerotial type, and called the disease Sclerotinia blight. Their identification was based on the sclerotial stage of the pathogen. The disease found in Oklahoma was identified as Sclerotinia blight also, and was based on the small sclerotial stage of the pathogen and the description from Porter and Beute. Other symptoms of the disease in Oklahoma include flagging, wilting, and necrosis of one or more stems. Necrotic areas are common on stems near or in contact with soil, but may occur several cm above the soil surface. Infected areas are usually covered with white, fluffy mycelia during periods of high humidity. All plant parts below ground are subject to attack by the fungus. Sclerotia are produced abundantly on pods and roots. Symptoms and signs of the disease and pathogen in Oklahoma closely resemble those previously reported (5).

During 1974, one field of peanuts with Sclerotinia blight was found with larger sclerotia than those found earlier. The pathogen was tentatively identified as *S. sclerotiorum* variety "Major". However, symptoms of the disease associated with larger sclerotia were indistinguishable from those with small sclerotia.

This paper reports on the occurrence of Sclerotinia blight of peanuts in Oklahoma, two sclerotial form-varieties of the pathogen, overwintering studies, the occurrence of the sexual stages of both form-varieties under field conditions, and confirms the original identification of the pathogen and the disease.

Materials and Methods

Cultural studies

Three isolates of the small and two isolates of the large sclerotial form-varieties were grown on potato-dextrose-agar (Difco) in darkness at 22°C. Size measurements were recorded for 120 sclerotia of each isolate.

Overwintering studies.

Two lots of 1000g of oat kernels each were moistened with 1000ml distilled water and sterilized in autoclavable plastic bags (Clavies). Each lot was inoculated with one variety of *S. sclerotiorum*. After 16 days at 22°C, sclerotia were formed abundantly by each fungal variety.

In February 1977, sclerotia of the small sclerotial form, produced on the grain medium, were buried at Stillwater in soil at depths ranging from approximately 19-50 mm in an area about 0.6 m square. Burial sites were in a peanut field which had been severely affected with Sclerotinia blight in 1976 from artificial infestation of soil with the small sclerotial form. In addition, several plants killed by Sclerotinia blight in this field in 1976, and having sclerotia on lower parts of stems, were protected by wire cages and left undisturbed to overwinter. Beginning in April 1977, four groups of potted peanut plants were placed close to apothecia produced at the base of dead plants protected by cages. Each group consisted of twelve, 15 cm clay pots containing 4-5 week-old flowering plants of the Spanish peanut cultivar 'Comet'.

The first group was exposed to ascospore discharge from April 23 to May 3. Three additional groups were exposed at 10-day intervals of May 3 to 13, May 13 to May 23, and May 23 to June 2, respectively. After the exposure period, each group of potted peanut plants was removed from the field and placed in a moist chamber for three days and then into a greenhouse maintained at 16-24°C at night and 25-30°C daytime for disease development.

In February 1976, sclerotia of the large sclerotial form, produced on the grain medium, were buried at Stillwater as previously described except that burial was in the fence-row area partially shaded and free from disturbance. By May 18, 1976, abundant apothecia were produced on sclerotia. Three days later, as previously described, a group of potted peanut plants was placed in the field in close proximity to these apothecia for seven days. Plants were then placed in a moist chamber and later into a greenhouse. Plants were observed daily for disease development. Three additional groups of plants were handled similarly.

[1]Journal Article No. 3516 of the Oklahoma Agricultural Experiment Station, Oklahoma State University, Stillwater, OK 74074.

[2]Professor, Department of Plant Pathology, Oklahoma State University, Stillwater, OK 74074

Results

Mycelia grew readily from sclerotia of both varieties of the fungus on PDA and formed new sclerotia within five days. Sclerotia of the small sclerotial form measured 0.5-2.0 x 0.5-4.0 mm (Fig. 1), however, sclerotia produced on the host under field conditions ranged up to 5.0 mm in length. Sclerotia of the large form, measured 1.6-6.0 X 2.0-9.0 mm. Sclerotia produced in the field were much larger and ranged from 2.0-4.0 X 4.0-75.0 mm (Fig. 1). Based on the consistent size differences of the two forms, the small sclerotial form was identified as *S. sclerotiorum* variety "Minor" and the large sclerotial form as *S. sclerotiorum* variety "Major" which follows the concept of Purdy (6). Further reference will be variety "Minor" or variety "Major".

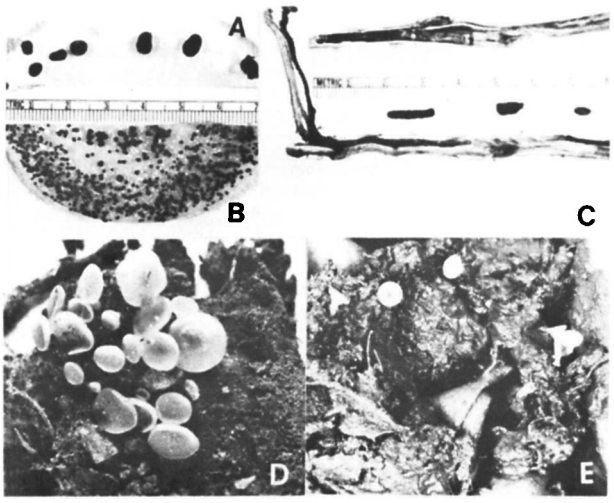

Fig. 1. (A to E). Sclerotia and apothecia of *Sclerotinia sclerotiorum* varieties "Major" and "Minor" from isolates infecting peanuts. A) Sclerotia of variety "Major" produced on PDA; B) Sclerotia of variety "Minor" produced on PDA; C) Sclerotia of variety "Major" produced inside peanut stems; D) Apothecia of variety "Major"; and E) Apothecia of variety "Minor". Apothecia are about twice natural size.

Sclerotia of the variety "Minor" from cultures which were buried in soil at the shallowest depth of approximately 19 mm produced only two apothecia. However, numerous apothecia were found on April 18, 1977 from naturally-occurring sclerotia associated with Sclerotinia-blighted plants left to overwinter in place in the field. Apothecia were found frequently in shallow holes in the soil made by rodents at the base of diseased plants. Apothecia of variety "Minor" were about 2 mm in diamter with stipes 8-10 mm long (Fig. 1). Measurements of ten asci averaged 8.6 X 138.6 μm and ascospores averged 5.0 X 12.1 μm. These measurements were in agreement with findings by Purdy (6).

Several apothecia of variety "Minor" were removed from the field and taken into the laboratory where the stipes were pressed into water-agar petri plates. These plates were inverted and suspended over PDA plates within a small plastic moist chamber. Many cultures developed from ascospores and produced sclerotia typical of the variety "Minor".

Only one potted peanut plant exposed to natural ascospore discharge from April 23 to May 3 became infected. Isolations from this plant produced typical small sclerotia of variety "Minor". Temperatures averaged slightly above normal during the 6-week exposure to ascospores of variety "Minor". Windy conditions were frequent, dew rarely occurred, and rainfall provided relatively short wetting periods.

Apothecia from buried sclerotia of variety "Major" were observed in the overwintering site on April 23, 1976 and were abundant by May 18. Apothecia continued to be produced at the overwintering site until mid-June. Apothecia of variety "Major" ranged from 4-6 mm in diameter on stipes up to 40 mm long depending on depth of sclerotia in the soil (Fig. 1). In a few cases, a single sclerotium produced as many as 18 apothecia. Measurements of ten asci averaged 7.3 X 143.5 μm and ascospores 5.0 X 12.2 μm. These measurements were within the range reported by Purdy (6). Infection sites developed on lower leaves and blossoms of about one half of the peanut plants exposed to ascospores from these apothecia between May 21 and June 3. Isolations from infected plant tissue produced cultures with sclerotia typical of the variety "Major". No symptoms occurred on plants of the last two groups exposed after June 3. Exposure of the first two groups of plants to ascospores from May 21 to June 3 occurred during cool, rainy weather but exposure of the last two groups from June 3 to June 17 occurred during warm, dry weather. Some sclerotia of the variety "Major" buried in 1976 produced apothecia in 1977.

Discussion

The original identification of Sclerotinia blight of peanuts in the United States by Porter and Beute (4) was based on the sclerotial (asexual) stage of the pathogen. In studies reported here, the sexual (apothecial) stage was produced in the field on sclerotia of two form-varieties of the pathogen. Measurements of apothecia, asci and ascospores fit ranges reported by Purdy (6) for *Sclerotinia sclerotiorum* varieties "Minor" and "Major". In Oklahoma the variety "Minor" occurs more frequently than "Major".

Observations of the disease showed infections developed primarily near the crown of plants from hyphae of germinating sclerotia. However, infection sites occurred occasionally on upper branches at 15-23 cm above the soil surface that may have resulted from ascospore infection and not from sclerotial hyphae.

Occasionally, fields of peanuts without histories

of Sclerotinia blight became severely infected with the variety "Minor" which might have resulted from infected seed, and/or from airborne ascospores. Remnants of original seed sources were not available to test for seed infection, but apothecia of *S. sclerotiorum* might have been produced in nearby fields, and windblown ascospores might have caused the infections. Natti (3) found a similar relationship in studies to determine sources of inoculum for white mold caused by *S. sclerotiorum* in eight bean fields. He failed to find apothecia within the fields, but did find apothecia in sod at the edge of one field. Seven of the eight fields studied developed severe out-breaks of the disease indicating an abundance of inoculum even though apothecia were not found within the fields. In other studies, Abawi and Grogan (1) found apothecia of the white mold fungus in bean fields and in fruit orchards with sod culture. Steadman et al. (7) reported ascospores of *S. sclerotiorum* landing on withered flowers of beans, germinate and after colonization, infected living plant parts. Young flowering peanut plants affected with Sclerotinia blight have not been observed in Oklahoma, but apothecia and ascospores of both varieties "Minor" and "Major" were produced from late April to early June. Peanut fields are usually planted in Oklahoma during early May. Flowering of Spanish peanuts begins about four weeks after planting and ascospores discharged late in May would over-lap the first flowers of early planted fields. However, infection is not limited to withering flowers. Hungerford and Pitts (2) reported stems of young bean seedlings were infected by direct penetration from germinated ascospores of *S. sclerotiorum* at the soil-line and near cotyledons and ascospore infection of 27 day-old bean plants was reported in which most infections were associated with injured areas rather than colonized blossoms (1). Thus, airborne ascospores could play an important role in initiating infection of seedlings and account for rapid spread and sudden occurrence of this disease in peanuts.

In one trial in the spring of 1977, only a few infection sites were found on peanut plants exposed to ascospores of the variety "Minor" in the field. During this time temperatures were slightly above normal and wetting periods were of short duration. In contrast, infections readily occurred with the variety "Major" in 1976 when cooler, wetter conditions prevailed during the first two exposures to ascospore discharge. Apothecia from overwintering sclerotia of the variety "Minor", produced viable ascospores in the laboratory and showed viability was not a limiting factor for infection in 1977. Therefore, spring weather in 1977 may have been a limiting factor in disease development and since apothecia were frequently found in shallow holes in the soil made by rodents, escape from infection may have occurred also.

Although many fields in which Sclerotinia blight occurs are irrigated, irrigation is not practiced early in the growing season and would not ordinarily affect primary infections. Since its discovery in Oklahoma, Sclerotinia blight of peanut has continued to increase in prevalence and economic importance. The disease was estimated to have caused about 1.5 percent loss in Oklahoma in 1977 (8). It is now known to occur in seven of 23 peanut-producing counties (Bryan, Caddo, Hughes, Atoka, Lincoln, Grady and Pottawatomie).

Literature Cited

1. Abawi, G. S. and R. G. Grogan. 1975. Source of primary inoculum and effects of temperature and moisture on infection of beans by Whetzelinia sclerotiorum. Phytopathology 65: 300-309.

2. Hungerford, C. W. and R. Pitts. 1953. The Sclerotinia disease of beans in Idaho. Phytopathology 43:519-521.

3. Natti, J. J. 1971. Epidemiology and control of bean white mold. Phytopathology 61:669-674.

4. Porter, D. M. and M. K. Beute. 1973. Peanut blight caused by a Sclerotinia species. Proc. Am. Peanut Res. and Ed. Assoc. 5(1):199 (Abstr.)

5. Porter, D. M. and M. K. Beute. 1974. Sclerotinia blight of peanuts. Phytopathology 64:263-264.

6. Purdy, L. H. 1955. A broader concept of the species Sclerotinia sclerotiorum based on variability. Phytopathology 45:421-427.

7. Steadman, J. R., E. D. Kerr and D. S. Wysong. 1974. White mold of dry beans. NebGuide G74-196. Coop. Ext. Serv. Inst. of Agric. and Nat. Res., Univ. of Nebr.

8. Sturgeon, R. V., Jr. and K. Jackson. 1977. Sclerotinia blight of peanuts. Current Rep. CR-7606.1-4. Okla. St. Univ. Coop. Ext. Serv.

9. Wadsworth, D. F. 1973. Research on the nature and control of peanut diseases in Oklahoma. Okla. Agric. Exp. Stn. Rep. P-683. 17 pp.

Accepted June 27, 1979