CHAPTER 1. INTRODUCTION AND OBJECTIVES

Sclerotinia blight was first recognized as a disease of peanut in Argentina in 1922 and since has been observed in most peanut producing countries of the world (35, 49). In the United States, Sclerotinia blight was first observed in Virginia in 1971 and has since spread to the peanut growing regions of North Carolina, Oklahoma, and Texas (48, 70, 76). Yield losses to the disease in Virginia were estimated to be 7.5% and 15% in 1977 and 1979, respectively (47, 69). By 1986, Sclerotinia blight was considered the most important disease of peanut in Virginia and Oklahoma (66). Replicated trials in naturally infested fields over a 4-yr period from 1988 to 1991 in Virginia demonstrated yield losses in excess of 32.9% in plots not receiving fungicide treatments (61).

Disease Cycle and Symptomology. The causal agent of Sclerotinia blight is the fungus, *Sclerotinia minor* (Jagger) Kohn (32). Sclerotia of this fungus serve as primary inoculum which may overwinter in the top 20 cm of soil (49, 54). Sclerotia survive in soil for several years and inoculum densities as low as one sclerotium per 100 g of soil can cause severe disease. Host tissue in close proximity to the soil surface is infected following myceliogenic germination of sclerotia (2, 48). The first visible symptoms of Sclerotinia blight are interveinal chlorosis of leaves followed by wilting of infected branches (49, Phipps *personal communication*). Stem lesions at first appear water soaked and later appear light tan with a distinct demarcation zone between healthy and infected tissue (48). White, cottony mycelium can be observed on infected tissue during cool, moist periods (49). Older lesions later appear shredded as a result of rapid decay which is likely due to the combined effects of oxalic acid and enzymes produced by the pathogen (48, 59). Black, irregular-shaped (.5-3mm dia.) sclerotia are produced on the surface and within diseased tissue, and serve as the source of inoculum for future plantings of peanut.

Environmental factors. Temperature has long been known to be an important determinant in the growth and development of *S. minor*. In 1939, Keay reported that the optimum temperature for myceliogenic germination and growth of *S. minor* was between 20-

25 C (31). Since then, several studies have confirmed that temperatures in the aforementioned range are optimum for growth, sclerotial germination and development of diseases caused by *Sclerotinia* spp. (1, 19, 30, 73, 76). Lee et al. (33) speculated that *S. minor* is inactive when soil temperatures at the 5-cm depth exceed 28 C.

Moisture is also a contributing factor to the development of diseases caused by *Sclerotinia* spp. Grogan and Abawi (1,22) found that mycelial growth and lesion expansion of *Sclerotinia sclerotiorum* (Lib.) DeBary on agar amended with soluble salts and on moistened bean stems increased as water potential and moisture levels increased, respectively. Sclerotia germinated at soil moisture tensions ranging from -1/3 to -15 bars, but the highest percentage germination occurred at -1/3 bar (30). Porter et al. reported significantly higher disease incidence in irrigated compared to non-irrigated peanuts (55). These results are similar to those observed in irrigation studies concerning white mold of edible dry beans caused by *S. sclerotiorum* (8, 73). Rainfall has also been associated with Sclerotinia blight outbreaks. Phipps reported that rainfall accumulations were heaviest 6 to 15 days before disease outbreaks (42). Moisture in the form of relative humidity (RH) has also been implicated in the growth of *S. minor* as well as in the development of Sclerotinia blight. Dow et al. (19) showed that 80% sclerotial germination occurred when sclerotia were incubated at 100% RH for periods of 12 hr or longer. Colonization of lateral branches and mainstems of peanut was facilitated as exposure to 100% RH was extended (19).

Vine Growth and Plant Foliar Canopy. Vine growth coupled with plant canopy density have an effect on soil temperature, soil moisture, duration and amount of leaf wetness, canopy RH, and canopy temperature (38). Therefore, host growth and architecture can play a major role in the microclimate around infection sites and disease development. According to a study by Blad et al. (8), dense canopies in beans had a cooler and more humid microclimate than open canopies, and resulted in a higher incidence of white mold caused by *S. sclerotiorum*. Another study showed that bean cultivars with dense foliar canopies had significantly higher disease severities than cultivars producing more open canopies (58). These same dense canopy characteristics in peanut seem to have a similar effect on

Sclerotinia blight of peanut, caused by *S. minor* (4, 20). Both Davidson et al. (16) and Sanders et al. (57) reported that plant canopy development in peanut has a direct effect on temperatures at or below the soil surface. Dow et al. and Bailey demonstrated that thinning plants reduced disease incidence and severity (4, 20). A 16-yr study confirmed these findings by showing that the initial onset of disease always occurred after peanut vines were within 15 cm from touching between rows or after vines overlapped between rows (42).

Other Factors. Additional factors which may influence the development and severity of Sclerotinia blight of peanut are often related to crop management practices. Frequent applications of chlorothalonil for control of leaf spot diseases have been shown to significantly increase the severity of Sclerotinia blight. Possible explanations for this increase in disease may include altered metabolic systems in the pathogen as well as suppression of microflora which are antagonistic to *S. minor* (24, 45, 46). High soil pH (6.0-7.0) and desiccated plant tissue on the soil surface have also been implicated as stimulating factors for the increased severity of Sclerotinia blight (23). Mechanical injury of vines by tractor tires represents yet another factor reported to predispose peanuts to infection by *S. minor* (12, 52). When Phipps (41) compared two planting dates (23-25 April and 14-15 May) and two seeding rates (78 and 157 kg/ha) he reported that disease incidence appeared earlier in the early planting date and was higher where the higher seeding rate was used. However, no differences in yield were detected between planting dates and seeding rates.

Disease Control. Resistant varieties are an ideal management tool for reducing losses to diseases. In peanut, however, true resistance to Sclerotinia blight has not been demonstrated (3, 14, 15). Rather than physiological or genetic forms of resistance, partial resistance of peanut cultivars is generally attributed to architectural characteristics of the host, such as an open foliar canopy and upright growth of lateral limbs (3, 14, 15). This partial resistance may be due to the spatial relationship of sclerotia of *S. minor* and host tissue as suggested in previous studies (29).

Canopy density and architecture are thought to be significant factors in development of diseases caused by *Sclerotinia* spp. (20, 42, 65). Dense canopies block fungicide sprays

from reaching lower leaves and stems near the soil surface (26, 34). Suppressing vine growth may improve fungicide penetration through the foliar canopy, and result in improved control of soilborne diseases (26, 34). Hawthorne noticed that incidence of lettuce drop caused by *S. minor* was less on plants with a more erect growth habit which allows for greater air movement and higher temperatures near the soil surface (25). This relationship has also been recognized in other studies of resistance (3, 14, 15). Wider row spacing has been shown to reduce white mold incidence in Great Northern beans (65). Thinning of the peanut canopy has been shown to significantly decrease the development of Sclerotinia blight of peanut (4,20). Mechanical pruning reduced the area under the disease progress curve (AUDPC) of Sclerotinia blight in peanuts, but reduced yield has also been associated with this type of canopy modification (4, 20).

Changes in plant canopy can be brought about by foliar-feeding insects and foliar diseases. The early instars of corn earworm (*Helicoverpa zea* Boddie) can cause significant defoliation of peanut (28). Defoliation by this insect usually occurs well after pod set and generally has no significant effect on yield (63). However, peanuts rarely are subjected to the full effect of this defoliation due to insecticide sprays (Herbert, *personal communication*). Leaf spot caused by *Cercospora arachidicola* Hori and *Cercosporidium personatum* (Berk. and Curt.) Deighton can also cause severe defoliation which may lead to a 50% loss in yield in years of heavy disease pressure; however yield loss is not likely if 40% or less defoliation occurs late in the season (59, Phipps, *personal communication*).

Plant growth regulators have been shown to modify the canopy architecture without reducing peanut yield (6, 37). In Bauman and Nordans study (6) the use of daminozide resulted in a more compact plant by shortening the internodes of lateral limbs and mainstems. This product is no longer used due to consumer concerns over chemical residues in peanut kernels and products (56). Prohexadione calcium, a new plant growth regulator for peanut, alters plant growth in a manner similar to daminozide and is pending for registration in the future (37). Chlorimuron, a systemic herbicide registered for use on peanut, has been evaluated for growth suppression (36, 67, 68). This chemical is effective in controlling

excessive vine growth, but may have detrimental effects on yield and quality (36). Paraquat is a contact herbicide used for early season weed control in peanuts (71, 72, 74, 75). However, this method of canopy alteration may cause yield losses if applied subsequent to 60 days after emergence of peanut (71) and has not been investigated for disease control. Modifications in the plant canopy by insects, diseases, and chemicals have not been tested for effects on Sclerotinia blight of peanut.

Currently, peanut growers depend on fungicide applications for suppression of Sclerotinia blight of peanut. In 1975, fungicides such as dicloran, PCNB, and benomyl provided partial control of Sclerotinia blight when multiple applications were made at high rates (7, 40). Procymidone, a dicarboximide fungicide, was reported to give effective control in field trials in 1980 (47). Other dicarboximides, such as iprodione and vinclozolin, were later shown to provide some control of the disease (18,40). Dicloran was used pursuant to section 18 of FIFRA for control of Sclerotinia blight prior to the registration of iprodione in 1985. Brenneman et al. (10) determined that the dicarboximides, iprodione and vinclozolin, were more effective in suppressing Sclerotinia blight and were able to deliver more residual control than dicloran. Lab studies utilizing detached peanut stems produced similar results (12). However, the dicarboximides were found to be more susceptible to leaching than dicloran (21).

Currently, iprodione is the only fungicide registered for control of Sclerotinia blight of peanut in the U. S. Iprodione provided 31% suppression of disease incidence and increased yield by 718 kg/ha over a 4-yr period in replicated field trials from 1989 to 1991 (61). This low level of control was previously thought to be the result of resistance to the dicarboximides by *S. minor*. Isolates with reduced sensitivity to the dicarboximides were observed *in vitro*, but field studies failed to show any differences in control between dicarboximide-treated microplots inoculated with dicarboximide-sensitive and -insensitive isolates of *S. minor* (9, 11, 50, 51, 62). Hubbard et al. (27) in 1997 suggested that rapid breakdown of iprodione by soil microflora was the probable reason for the lack of control associated with lettuce drop caused by *S. minor*. The new fungicide, fluazinam, was shown

to provide good to excellent suppression of Sclerotinia blight in 1987. Reports in 1991 and 1992 documented that fluazinam limited disease incidence and increased yield of peanut greater than the dicarboximides, iprodione or vinclozolin (60, 61). In these trials, fluazinam at 0.56 kg a.i./ha resulted in 69% suppression of disease incidence and increased yield by 1598 kg/ha.

The timing of fungicide application appears to be critical for control of diseases caused by *Sclerotinia* spp. (64). When applied just prior to infection of lettuce by *S. minor*, sprays of vinclozolin and iprodione were just as effective as calender sprays and more effective than post-infection or "demand" treatments (39, 77). Fluazinam, when applied just prior to inoculation of detached peanut limbs, reduced the AUDPC greater than post-inoculation applications (13). The "demand" program relies on intensive weekly scouting for disease. The initial fungicide application is made upon detection of disease and subsequent sprays are made at 3 or 4-wk intervals. A negative aspect of the "demand" program is that fungicide applications are made often only after disease has already caused significant damage. Furthermore, subsequent applications are made on a calender schedule instead of when weather conditions favor disease development. The above observations underscore the need for a predictive advisory program to improve the efficiency of fungicide use and eliminate needless sprays.

With the knowledge that fungicides for control of Sclerotinia blight are more effective when applied as preventative treatments (13, 64, 77), recent studies have focused on developing algorithms to predict disease onset and increase the efficiency of fungicide sprays. Researchers in these studies assigned indices to environmental and host factors, which are known to favor disease development, and utilized these indices as input for algorithms to predict disease onset and determine when fungicide sprays are needed (5, 33, 43, 44). These algorithms have shown potential for reducing the number of fungicide applications below that of "demand" treatments while maintaining similar yields (5, 43, 44). However, algorithms have not been tested across many locations or a wide range of environments.

Objectives. The primary goal of this research was to develop and refine algorithms which may provide an early warning for the onset of Sclerotinia blight of peanut through the use of crop growth parameters and detailed weather data which is currently available in Virginia (17). The warnings triggered by the algorithms will be used to time fungicide applications. A secondary goal was to determine methods by which plant growth and canopy characteristics could be altered to favor improved disease management through manipulation of planting date, growth regulators, herbicides, and management of foliar insects and diseases.

The objectives were: 1) to develop and validate an algorithm for predicting the onset of Sclerotinia blight and determine the duration of plant protection following a fungicide application (Chapter 2); 2) to determine the effect of planting date on disease management and fungicide performance (Chapter 3); and 3) to evaluate the utility of plant growth and canopy modifiers as tools for suppressing disease (Chapter 4).

LITERATURE CITED

1. Abawi, G. S., and Grogan, R. G. 1975. Source of primary inoculum and effect of temperature and moisture on infection of beans by *Whetzelina sclerotiorum*. Phytopathology 65:300-309.
2. Adams, P. B., and Tate, C. J. 1976. Mycelial germination of sclerotia of *Sclerotinia sclerotiorum* on soil. Plant Dis. Rptr. 60:515-518.
3. Akem, C. N., Melouk, H. A., and Smith, O. D. 1992. Field evaluation of peanut genotypes for resistance to Sclerotinia blight. Crop Prot. 4:345-348.
4. Bailey, J. E., and Brune, P. D. 1997. Effect of crop pruning on Sclerotinia blight of peanut. Plant Dis. 81:990-995.
5. Bailey, J. E., Phipps, P. M., Lee, T. A., and Damicone, J. 1993. Utilization of environmental thresholds to minimize fungicide applications for control of Sclerotinia blight of peanut. (Abstr.) Proc. Am. Peanut Res. Educ. Soc. 25:46.
6. Bauman, R. W., and Nordan, A. J. 1971. Effect of plant growth regulators on

vegetative and reproductive characteristics of six peanut genotypes. J. Am. Peanut Res. Educ. Assoc. 3:75-80.

7. Beute, M. K., Porter, D. M., and Hadley, B. A. 1975. Sclerotinia blight of peanut in North Carolina and Virginia and its chemical control. Plant Dis. Rptr. 59:697-701.
8. Blad, B. L., Steadman, J. R., and Weiss, A. 1978. Canopy structure and irrigation influence white mold disease and microclimate of dry edible beans. Phytopathology 68:1431-1439.
9. Brenneman, T. B., Phipps, P. M., and Stipes, R. J. 1987. Control of Sclerotinia blight of peanut: Sensitivity and resistance of *Sclerotinia minor* to vinclozolin, iprodione, dicloran, and PCNB. Plant Dis. 71:87-90.
10. Brenneman, T. B., Phipps, P. M., and Stipes, R. J. 1987. Performance characteristics of dicloran, iprodione, and vinclozolin for control of Sclerotinia blight of peanut. Plant Dis. 71:546-548.
11. Brenneman, T. B., Phipps, P. M., and Stipes, R. J. 1987. Sclerotinia blight of peanut: Relationship between in vitro resistance and field efficacy of dicarboximide fungicides. Phytopathology 77:1028-1032.
12. Brenneman, T. B., Phipps, P. M. and Stipes, R. J. 1988. A rapid method for evaluating genotype resistance, fungicide activity, and isolate pathogenicity of *Sclerotinia minor* in peanut. Peanut Sci. 15:104-107.
13. Butzler, T. M., Bailey, J. E., and Beute, M. K. 1993. Effects of pre-inoculation and post-inoculation application of fluazinam on *Sclerotinia minor* using three peanut genotypes. (Abstr.) Proc. Am. Peanut Res. Educ. Soc. 25:19.
14. Chappell, G. F. II, Shew, B. B., Ferguson, J. M., and Beute, M. K. 1995. Mechanisms of resistance to *Sclerotinia minor* in selected peanut genotypes. Crop Sci. 35:692-696.
15. Coffelt, T. A., and Porter, D. M. 1982. Screening peanuts for resistance to Sclerotinia blight. Plant Dis. 66:385-387.
16. Davidson, J. I., Blankenship, P. D., Henning, R. J., Guerte, W. R., Smith, R. D., and Cole, R. J. 1991. Geocarposphere temperature as it relates to Florunner peanut

production. Peanut Sci. 18:79-85.

17. Deck, S. H., and Phipps, P. M. 1995. Critical crop and weather information go online in Virginia. Resource 3:8-11.
18. Dougherty, D. E., Sajorak, D. J., and Locher, F. 1983. Fungicides for reducing losses caused by Sclerotinia blight on peanut. Plant Dis. 67:312-314.
19. Dow, R. L., and Porter, D. M., and Powell, N. L. 1988. Effect of environmental factors on *Sclerotinia minor* and Sclerotinia blight of peanut. Phytopathology 78:672-676.
20. Dow, R. L., Powell, N. L., and Porter, D. M. 1988. Effect of modification of the plant canopy environment on Sclerotinia blight of peanut. Peanut Sci. 15:1-5.
21. Elmer, W. H., and Stipes, R. J. 1985. Movement and disappearance of dicloran, iprodione, and vinclozolin in peanut and nonpeanut soils. Plant Dis. 69:292-294.
22. Grogan, R. G., and Abawi, G. S. 1975. Influence of water potential on growth and survival of *Whetzelina sclerotiorum*. Phytopathology 65:122-128.
23. Hau, F. C., and Beute, M. K. 1982. Effect of soil pH and volatile plant stimulants from remoistened peanut leaves on germination of *Sclerotinia minor*. Plant Dis. 66:223-224.
24. Hau, F. C., and Beute, M. K. 1983. Effects of chlorothalonil on the virulence and physiology of a nontargeted pathogen, *Sclerotinia minor*. Phytopathology 73:475-479.
25. Hawthorne, B. J. 1974. *Sclerotinia minor* on lettuce: Effect of plant growth on susceptibility to infection. New Zealand J. Agr. Res. 7:387-392.
26. Henning, R. J., Allison, A. H., and Tripp, L. D. 1982. Cultural practices. Pages 123-138 in: Peanut Science and Technology. H. E. Pattee and C. T. Young, eds. Am. Peanut Res. Educ. Soc., Yoakum, TX.
27. Hubbard, J. C., Subbarao, K. V., and Koike, S. T. 1997. Development and significance of dicarboximide resistance in *Sclerotinia minor* isolates from commercial lettuce fields in California. Plant Dis. 81:148-153.
28. Huffman, F. R., and Smith, J. W. Jr. 1979. Bollworm: Peanut foliage consumption

and larval development. Environ. Entomol. 8:465-467.

29. Imolehin, E. D., and Grogan, R. G. 1980. Factors affecting survival of sclerotia, and effects of inoculum density, relative position, and distance of sclerotia from host on infection of lettuce by *Sclerotinia minor*. Phytopathology 70:1162-1167.
30. Imolehin, E. D., Grogan, R. G., and Duniway, J. M. 1980. Effect of temperature and moisture tension on growth, sclerotial production, germination, and infection by *Sclerotinia minor*. Phytopathology 70:1153-1157.
31. Keay, M. A. 1939. A study of certain species of the genus *Sclerotinia*. Ann. Appl. Biol. 26:227-246.
32. Kohn, L. M. 1979. A monographic revision of the genus *Sclerotinia*. Mycotaxon 9:365-444.
33. Lee, T. A., Woodard, K. E., and Simpson, C. E. 1990. Prediction of Sclerotinia blight of peanut based on soil temperatures at 5 cm. (Abstr.) Proc. Am. Peanut Res. Educ. Soc. 22:45.
34. Maloy, O. C. 1993. Plant Disease Control: Principles and Practices. John Wiley and Sons, New York. 345 pp.
35. Marchionatta, J. B. 1922. Peanut wilt in Argentina. Rev. Fac. Agron. La Plata 3: 385-387.
36. Mitchem, W. E., York, A. C., and Batts, R. B. 1995. Evaluation of chlorimuron as a growth regulator for peanut. Peanut Sci. 22:62-66.
37. Mitchem, W. E., York, A. C., and Batts, R. B. 1995. Peanut response to prohexadione calcium, a new plant growth regulator. Peanut Sci. 23:1-9.
38. Oke, T. R. 1978. Boundary layer climates. John Wiley and Sons, New York. 372 pp.
39. Patterson, C. L., and Grogan, R. G. 1985. Differences in epidemiology and control of lettuce drop caused by *Sclerotinia minor* and *S. sclerotiorum*. Plant Dis. 69:766-770.
40. Phipps, P. M. 1983. Control of Sclerotinia blight of peanut with fungicides. (Abstr.) Proc. Am. Peanut Res. Educ. Soc. 13:801.

41. Phipps, P. M. 1987. An evaluation of cultural and chemical practices for management of Sclerotinia blight of peanut in Virginia. Biological and Cultural Tests for Control of Plant Disease 2:52.

42. Phipps, P. M. 1995. An assessment of environmental conditions preceding outbreaks of Sclerotinia blight of peanut in Virginia. Peanut Sci. 22:90-93.

43. Phipps, P. M. 1995. An algorithm for predicting outbreaks of Sclerotinia blight of peanut and improving the efficiency of fungicide sprays. (Abstr.) Proc. Am. Peanut Res. Educ. Soc. 27:43.

44. Phipps, P. M., Deck, S. H., and Walker, D. R. 1997. Weather-based crop and disease advisories for peanuts in Virginia. Plant Dis. 81:236-244.

45. Porter, D. M. 1977. Effect of chlorothalonil and benomyl on the severity of Sclerotinia blight of peanuts. Plant. Dis. Rptr. 61:995-998.

46. Porter, D. M. 1980. Increased severity of Sclerotinia blight in peanuts treated with captafol and chlorothalonil. Plant Dis. 64:394-395.

47. Porter, D. M. 1980. Control of Sclerotinia blight with procymidone. Plant Dis. 64: 865-867.

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

49. Porter, D. M., and Melouk, H. A. 1997. Sclerotinia blight. Pages 34-36 in: Compendium of peanut diseases (2nd ed.). N. Kokalis-Burelle, D. M. Porter, R. Rodriquez-Kabana, D. H. Smith, and P. Subrahmanyam, eds. APS Press. St Paul, MN.

50. Porter, D. M., and Phipps, P. M. 1985. Tolerance of *Sclerotinia minor* to procymidone and cross tolerance to other dicarboximide fungicides and dicloran. Peanut Sci. 12:41-45.

51. Porter, D. M., and Phipps, P. M. 1985. Effects of three fungicides on mycelial growth, sclerotial production, and development of fungicide-tolerant isolates of *Sclerotinia minor*. Plant Dis. 69:143-146.

52. Porter, D. M., and Powell, N. L. 1978. Sclerotinia blight development in peanut vines injured by tractor tires. Peanut Sci. 5:87-90.

53. Porter, D. M., Powell, N. L., and Cobb, P. R. 1977. Severity of Sclerotinia blight of peanut as detected by infrared aerial photography. Peanut Sci. 4:75-77.

54. Porter, D. M., and Steele, J. L. 1983. Quantitative assay by elutriation of peanut field soil for sclerotia of *Sclerotinia minor*. Phytopathology 73:636-640.

55. Porter, D. M., Wright, F. S., and Powell, N. L. 1987. Effects of sprinkler irrigation on peanut diseases in Virginia. Plant Dis. 71:512-515.

56. Reynolds, S. 1986. Blackmailed by bad science, the facts about Kylar. Peanut Farmer. June, Pages 6-7.

57. Sanders, T. H., Blankenship, P. D., Cole, R. J., and Hill, R. A. 1985. Temperature relationships of peanut leaf canopy, stem, and fruit in soil of varying temperature and moisture. Peanut Sci. 12:86-89.

58. Schwartz, H. F., Steadman, J. R., and Coyne, D. P. 1978. Influence of *Phaseolus vulgaris* blossoming characteristics and canopy structure upon reaction to *Sclerotinia sclerotiorum*. Phytopathology 68:465-470.

59. Smith, D. H., and Littrell, R. H. 1980. Management of peanut foliar diseases with fungicides. Plant Dis. 64:356-361.

60. Smith, F. D., Phipps, P. M., and Stipes, R. J. 1991. Agar plate, soil plate, and field evaluation of fluazinam and other fungicides for control of *Sclerotinia minor* on peanut. Plant Dis. 75:1138-1143.

61. Smith, F. D., Phipps, P. M., and Stipes, R. J. 1992. Fluazinam: A new fungicide for control of Sclerotinia blight and other soilborne pathogens of peanut. Peanut Sci. 19:115-120.

62. Smith, F. D., Phipps, P. M., and Stipes, R. J., and Brenneman, T. B. 1995. Significance of insensitivity of *Sclerotinia minor* to iprodione in control of Sclerotinia blight of peanut. Plant Dis. 79:517-523.

63. Smith, J. W., Jr., and Barfield, C. S. 1982. Management of preharvest insects. Pages

250-325 in: Peanut Science and Technology. H. E. Pattee and C. T. Young, eds. Am. Peanut Res. Educ. Soc, Yoakum, TX.

64. Steadman, J. R. 1979. Control of plant diseases caused by *Sclerotinia* species. Phytopathology 69:1070-1071.

65. Steadman, J. R., Coyne, D. P., and Cook, G. E. 1973. Reduction of severity of white mold disease on great northern beans by wider row spacing and determinate plant growth habit. Plant Dis. Rptr. 57:1070-1071.

66. Sturgeon, R. V. 1986. Peanut disease loss estimates for major peanut production states in the United States for 1984 and 1985. Proc. Am. Peanut Res. Educ. Soc. 18:24-26.

67. Swann, C. W. 1992. Response of Virginia-type peanut cultivars to chlorimuron. (Abstr.) Proc. Am. Peanut Res. Educ. Soc. 23:31.

68. Swann, C. W. 1995. Response of NC-9 to chlorimuron. (Abstr.) Proc Am. Peanut Res. Educ. Soc. 27:49.

69. Thomas, D. F., Powell, N. L., Porter, D. M., and Phipps, P. M. 1980. Use of aerial infrared photography to determine estimates of peanut crop losses due to Sclerotinia blight of peanut. (Abstr.) Proc. Am. Peanut Res. Educ. Soc. 13:89.

70. Wadsworth, D. F. 1979. Sclerotinia blight of peanuts in Oklahoma and occurrence of the sexual stage of the pathogen. Peanut Sci. 6:77-79.

71. Whetje, G., McQuire, J. A., Walker, R. W., and Patterson, M. G. 1986. Texas panicum control in peanuts with paraquat. Weed Sci. 34:308-311.

72. Wehtje, G., Wilcut, J. W., McGuire, J. A., and Hicks, T. V. 1991. Foliar penetration and phytotoxicity of paraquat as influenced by peanut cultivar. Peanut Sci. 18:67-71.

73. Weiss, A. E., Kerr, E. D., and Steadman, J. R. 1980. Temperature and moisture influence on development of white mold disease (*Sclerotinia sclerotiorum*) on great northern beans. Plant Dis. 64:757-759.

74. Wilcut, J. W., and Swann, C. W. 1991. Timing of paraquat applications for weed control in Virginia-type peanuts (*Arachis hypogaea*). Weed Sci. 38:558-562.

75. Wilcut, J. W., Whetje, G., Cole, T. A., and Hicks, T. V. 1989. Postemergence weed control systems without dinoseb for peanuts. Weed Sci. 37:385-391.

76. Woodard, K. E., and Simpson, C. E. 1993. Characterization of growth and sclerotial production of *Sclerotinia minor* isolated from peanut in Texas. Plant Dis. 77:576-579.

77. Wymore, L. A., and Lorbeer, J. W. 1986. Timing fungicidal control of *Sclerotinia minor* causing drop of lettuce on organic soils of New York. Plant Dis. 70:1134-1138.