EFFECT OF POSTEMERGENCE HERBICIDES ON (CERCOSPORA ARACHIDICOLA), THE CAUSAL AGENT OF EARLY LEAFSPOT DISEASE IN PEANUT (ARACHIS HYPOGAEA)

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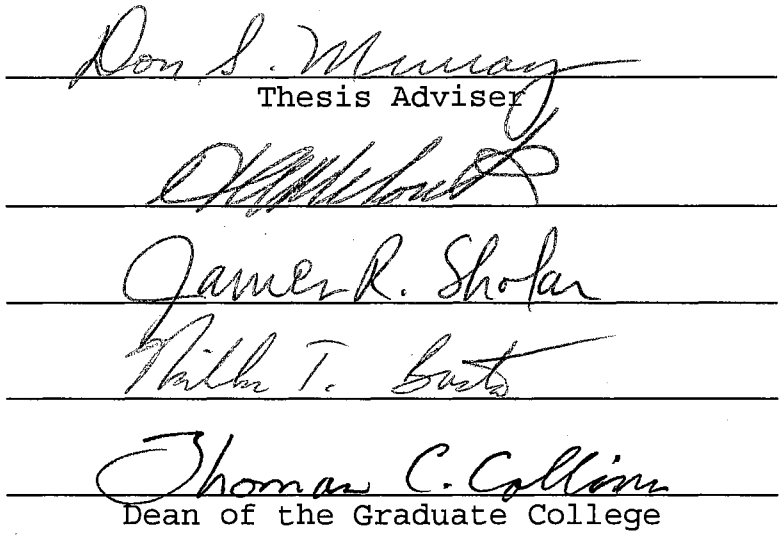

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ALTERATIONS OF LEAF EPICUTICULAR WAX OF PEANUT (ARACHIS HYPOGAEA) BY APPLICATIONS OF HERBICIDE AND ADJUVANT

Abstract. Leaf surface morphology of untreated peanut leaves and peanut leaves treated with herbicide and adjuvants were examined using scanning electron microscopy. Electron micrographs revealed that the adaxial peanut leaf surface was covered with crystalline wax platelets above an amorphous layer of wax. Electron micrographs revealed that peanut leaves treated with acifluorfen plus nonionic surfactant, bentazon and lactofen with crop oil concentrate, and 2,4-DB, altered the leaf surface morphology when compared to peanut leaves that were untreated. Alterations in the leaf epicuticular wax structures occurred and appeared amorphous-like rather than normal plate-like structures. Nonionic surfactant and crop oil concentrate applied alone to peanut leaves altered the epicuticular wax structures similarly to that of herbicides and adjuvants. **Nomenclature:** Acifluorfen, 5-[2-chloro-4-(trifluoromethyl)phenoxy]-2-nitrobenzoic acid; bentazon, 3-(1-methylethyl)-(1H)-2,1,3-benzothiadiazin-4(3H)-one 2,2-dioxide; lactofen, (±)-2-ethoxy-1-methyl-2-oxoethyl 5-[2-chloro-4-(trifluoromethyl)phenoxy]-2-nitrobenzoate; 2,4-DB,

4-(2,4-dichlorophenoxy)butanoic acid; Peanut, Arachis hypogaea L.

Additional index words: Epicuticular wax, scanning electron microscopy, adjuvant, leaf surface morphology.

INTRODUCTION

Postemergence (POST[1]) herbicide applications are important for weed control and for economical peanut production. Surfactants or spray adjuvants are used with many POST herbicide spray solutions to enhance activity on weeds (11, 24). The role of adjuvants is to aid in the surface spreading and penetration properties of the herbicide through the leaf cuticle of the target species (9, 18, 24). However, some spray from topical applications is intercepted by the peanut plant and may result in temporary injury to peanut leaves. Contact herbicides that cause this crop injury have usually been reported to have minimal negative effects on peanut pod yield (13, 27).

Plant cuticles consist of waxes, pectin, cutin, and cellulose material (5, 11, 24). The composition of these cuticular components varies with plant species. The cuticle provides a barrier between the environment and the plant's internal cells and the cuticle is the first plant

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[1]Abbreviations: POST, postemergence; SEM, scanning electron microscopy; WAP, weeks after planting; COC, crop oil concentrate; NIS, nonionic surfactant.

structure to be attacked by insects or plant pathogens (16). The cuticle surface wax or epicuticular wax is an important barrier to ion and water movement across the cuticle (1). This wax is made up of crystalline deposits which overlay the cuticle as plate, ribbon, tube, or rod-like structures (2). The amount of epicuticular wax varies with plant species and environment. Plant leaves with thicker deposits of wax tend to be more hydrophobic, thus decreasing water droplet and herbicide spray retention and possibly infection by pathogens (1, 9, 16).

Numerous researchers have reported on the effects of herbicide spray formulations and surfactants on leaf surface characteristics (3, 7, 8, 10, 12, 15, 20, 22, 26). Whitehouse et al. (26) suggested that certain herbicides may partition into the epicuticular wax more readily than others causing an alteration in the wax barrier which reduces foliar entry of other herbicides. Several reports (8, 15, 20, 26) have identified leaf surface alterations by herbicides through the use of scanning electron microscopy (SEM[1]). In one report (20), SEM micrographs showed that the surfactant Tween® 20[2] dissolved some leaf surface wax of Eucalyptus polyanthemos Schau. and altered the physical form of the remaining surface wax to globular appearing formations.

The effect of POST herbicides on peanut leaf surfaces has not been well studied, nor have the leaf surface

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[2]ICI Americas Inc., New Murphy Rd., Wilmington, DE 19897.

morphology and epicuticular wax formations been adequately illustrated. It is therefore difficult to assess any direct effects herbicides may have on the epicuticular wax functions (i.e. barrier to insects and pathogens). The objective of this study was to examine and illustrate the response of several POST applied herbicides and adjuvants on the adaxial peanut leaf surface topography, specifically the epicuticular wax, with the use of SEM.

MATERIALS AND METHODS

Plant material used. Peanut seed of the cultivar 'Okrun' were planted in individual 12 cm containers in the greenhouse that consisted of a medium of soil, sand, and finely shredded peat (1:1:2, v/v/v). Greenhouse air temperature during the day was 28 ± 3 C, the night temperature was 22 ± 3 C, and relative humidity was 65 ± 20%. Leaf samples collected from the different peanut plants used in this experiment were from the same vegetative growth stage (i.e. node number). The epicuticular wax structures in some plant species change or become more developed as the plant grows, therefore we wanted to insure a uniformity of epicuticular wax structures in the leaves we examined.

Herbicides. Four weeks after planting (WAP[1]), 12 to 14 cm peanut plants were treated with postemergence herbicides

and adjuvants using a laboratory table sprayer equipped with an 8002 even flat fan nozzle delivering 140 L/ha. Herbicide treatments were: acifluorfen at 0.56 kg ai/ha, bentazon at 0.84 kg/ha, lactofen at 0.21 kg/ha, and 2,4-DB at 0.45 kg/ha. The adjuvants used were: crop oil concentrate (COC[1])[3] applied at a rate equivalent to 2.3 L/ha and nonionic surfactant (NIS[1])[4] applied at 0.25% v/v. Acifluorfen and lactofen treatments were in combination with NIS and bentazon was in combination with COC. The 2,4-DB treatment contained no additional adjuvant. Each adjuvant was applied alone as a treatment for comparison with the other herbicide treatments and to illustrate any leaf surface activity.

Scanning electron microscopy. Peanut leaves were placed in 2% gluteraldehyde in 0.1 M sodium carodylate buffer at pH 7.2 for 3 wk. The samples were then given three 20-minute buffer washes (0.1 M sodium carodylate buffer pH 7.2) and dehydrated in a graded ethanol series of 50, 70, 90, 95, and 100%. The tissue remained in the alcohol for 20 minutes each, ending with three changes of 100% for 20 minutes each. The samples were critical point dried in a

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[3]Cornbelt® Crop Oil Concentrate. Cornbelt Chemical Company, P.O. Box 410, McCook, NE 69001.

[4]Triton AG-98®. Rohm and Hass Co., Independence Mall W., Philadelphia, PA 19105.

liquid CO_2 critical point dryer[5]. Specimens were mounted on aluminum stubs with double sticky tape and were coated with 200 Å of gold and palladium using a sputter coater[6]. All SEM examinations were performed with a JEOL-JSM 35U scanning electron microscope[7] and photographed at accelerating potentials of 25kV.

This experiment was conducted two times with each herbicide treatment replicated four times. Photographs presented in this report were selected for their clarity and are representative of numerous SEM micrographs taken from each treatment and experiment.

RESULTS AND DISCUSSION

Scanning electron microscopy. The SEM micrographs show that the adaxial peanut leaf surface is covered with well developed crystalline wax platelets above an amorphous layer of wax (Figure 1), and resembles that of micrographs of pea (Pisum sativum L.) in previous reports (4, 19, 21, 22). The crystalline wax formations are less abundant on the periclinal walls of the guard cells. This similarity

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[5]Tousimis PVT-3 CPD, Tousimis Research Corp., Rockville, MD 20852.

[6]Hummer II. Techniques, 5510 Vine Street, Alexandria, VA 22310.

[7]JSM 35U. JEOL(U.S.A.), 11 Dearborn Road, Peabody, MA 10960.

has been noted by other researchers with different plant species (9, 21). The adaxial surface is stomatous and free of trichomes.

Alterations of peanut leaf epicuticular wax were very evident with applications of acifluorfen plus NIS (Figure 2). Areas of herbicide deposition appeared very dark and smooth in texture. The epicuticular wax structures were altered, resulting in an amorphous appearance. Nalewaja et al. (17) reported dark areas below glyphosate [*N*-(phosphonomethyl)glycine] crystal deposits seen on micrographs of common sunflower (Helianthus annuus L.) leaves. These areas may represent cuticle injury and phytotoxicity from the herbicide. It is not known whether the original amount of wax was still present in those areas of herbicide deposition or if it was reduced. We do not rule out the possible presence of some epicuticular wax on the leaf surface but it may be in the form of a continuous sheet, with no crystalline, plate-like structures. Acifluorfen-sodium applied to soybean [Glycine max (L.) Merr.] produced similar results in a previous report (9).

SEM micrographs of NIS applied without a herbicide (Figure 3) illustrate similar results as those with the combination of acifluorfen plus NIS. This suggests that NIS is a major component in the alteration of peanut epicuticular wax. Takeno and Foy (23) reported that a lipophilic polysorbate surfactant had altered the ultrastucture of epicuticular wax on cotton (Gossypium

hirsutum L.) leaves, but they noticed no erosion of the surface wax. In a more recent report, Falk et al. (6) reported that certain surfactants induced phytotoxicity to several plant species but they did not observe morphological changes in surface wax. In other reports (14, 15) that support our findings, applications of surfactants have altered the leaf wax morphology in Brassica species.

Peanut leaves treated with lactofen plus NIS had epicuticular wax alterations along with significant cell damage (Figure 4). The loss of cell membrane integrity is the characteristic mode of action of lactofen, a herbicide classified in the diphenyl-ether herbicide family (25). Acifluorfen, another diphenyl-ether, did not damage cell membranes to the extent that lactofen did therefore peanut leaf necrosis was visually greater with lactofen treatments (author's observations). Acifluorfen treated peanut leaves had minimal leaf tissue necrosis and were lightly bronzed in appearance.

Applications of COC alone to peanut leaves altered the epicuticular wax of peanut into an amorphous-layered structure (Figure 5). The crystalline structures appear to have been altered in the center of the spray deposition areas and the effect gradually lessens toward the outer edges. When bentazon was added to COC, micrographs of the combination showed little differences in epicuticular wax alteration compared to COC applied alone (Figure 6). The

only differences observed were more particulate deposits on the leaf surface. This may be due to the nature of the commercial formulation of bentazon in solution.

Peanut leaves that received 2,4-DB applications had epicuticular wax alterations similar to the bentazon and COC treatment (Figure 7). There were no adjuvants added to the 2,4-DB so the epicuticular wax alterations were related solely to herbicide application. There was no leaf tissue necrosis symptoms induced with this treatment; however, plant hormone regulating characteristics were noticed. With this treatment it is not understood if this injury may be the reason for the leaf wax alterations. One possibility is be that the nature of the acid formulation of 2,4-DB could have contributed to the wax alterations.

These observations indicate and illustrate that different herbicides and adjuvants under controlled conditions, cause morphological modifications to peanut leaf epicuticular wax. The use of SEM has been very useful in demonstrating the leaf epicuticular wax alterations. This information may be helpful in explaining some of the phytotoxic activity that occurs with the use of these herbicides and possibly the effects this activity may have on other organisms (i.e. pathogens, insects) that share the same environment. The information obtained from this study will be valuable in future research that involve herbicides and peanut leaf cuticles.

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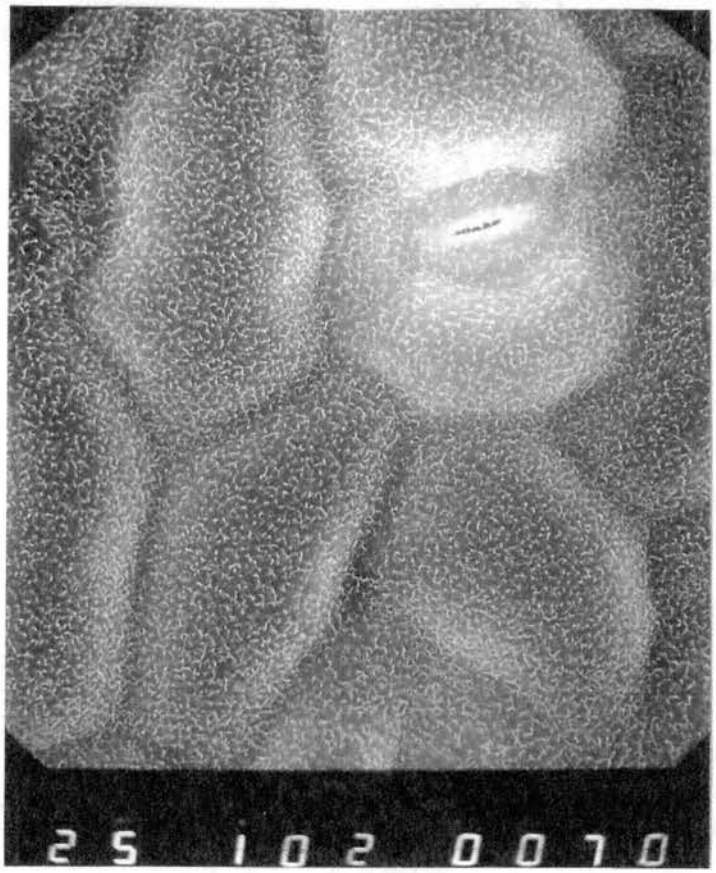

Figure 1. Scanning electron micrograph of the adaxial leaf surface of an untreated peanut at 1000x magnification.

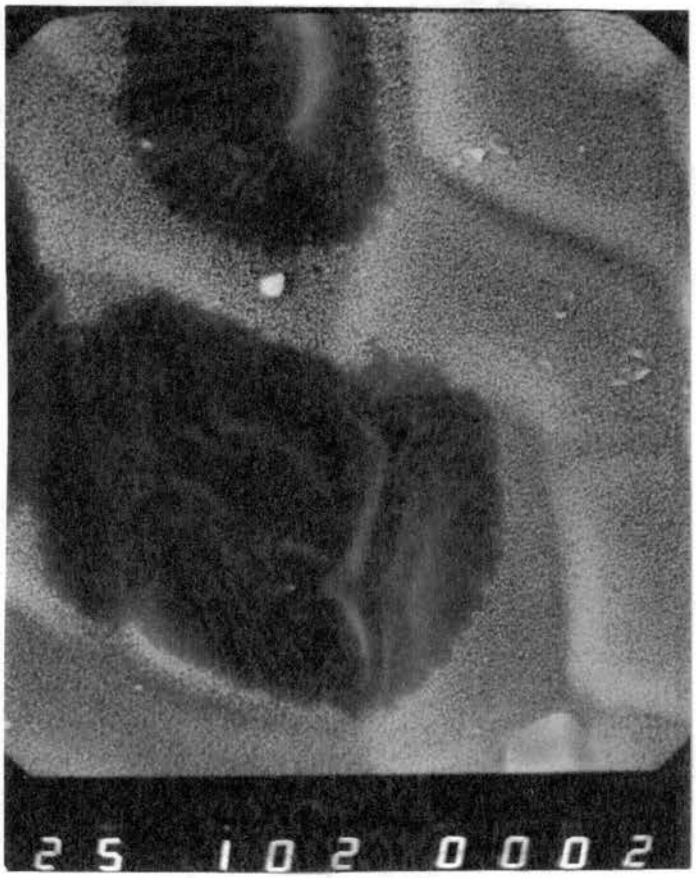

Figure 2. Acifluorfen (0.56 kg/ha in 140 L/ha water carrier) and nonionic surfactant (0.25% v/v) applied to peanut. The dark areas depict crystalline wax degradation by herbicide and surfactant droplet. Magnification 1000x.

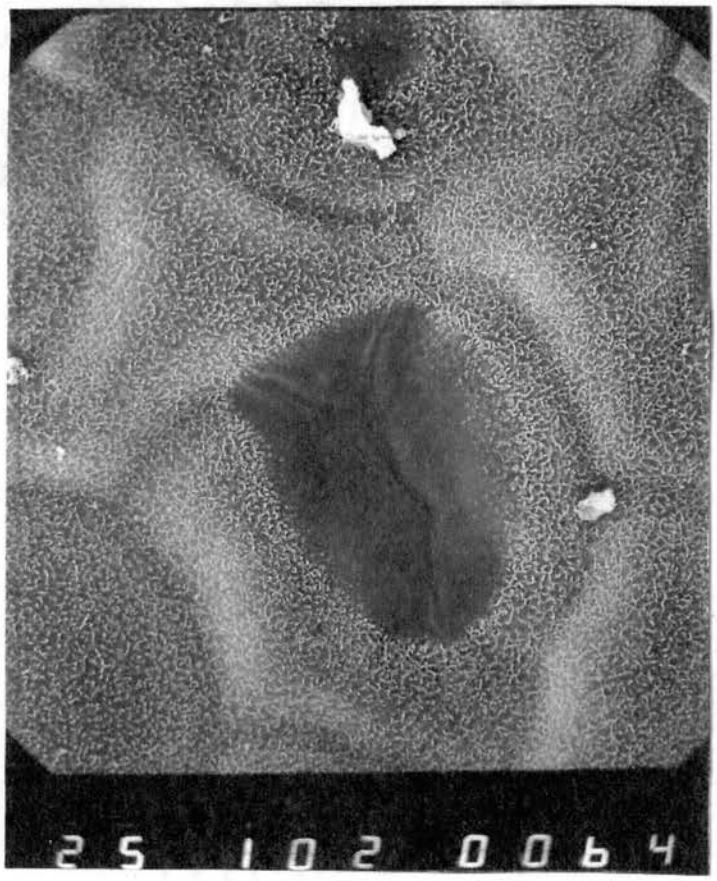

Figure 3. Nonionic surfactant (0.25% v/v in 140 L/ha water carrier) applied to peanut. Magnification 1000x.

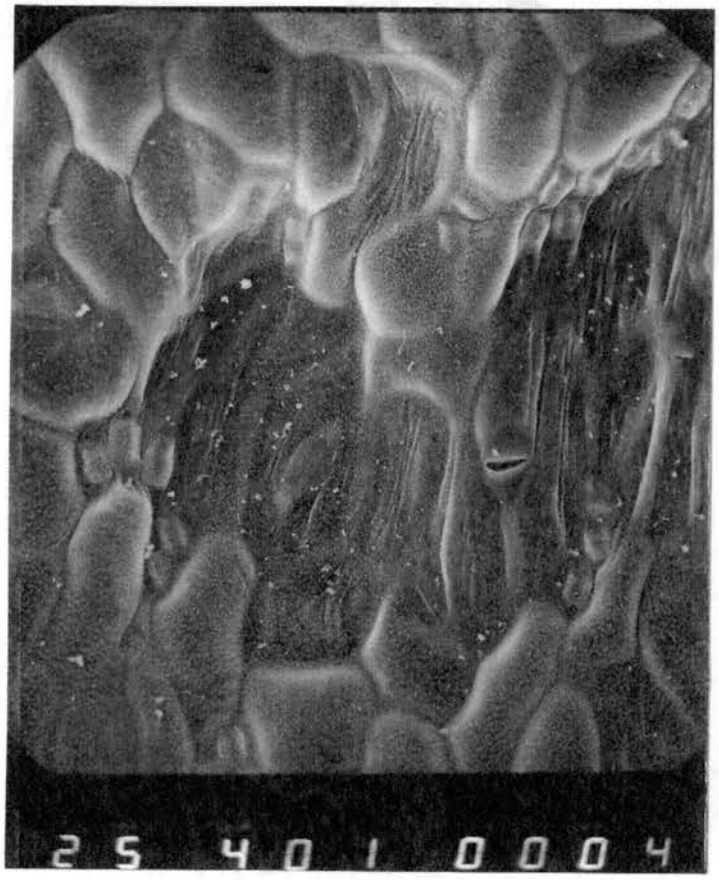

Figure 4. Lactofen (0.21 kg/ha in 140 L/ha water carrier) and nonionic surfactant (0.25% v/v) applied to peanut. Note the ruptured cell membranes. Magnification 400x.

Figure 5. Crop oil concentrate (2.3 L/ha in 140 L/ha water carrier) applied to peanut. Magnification 1000x.

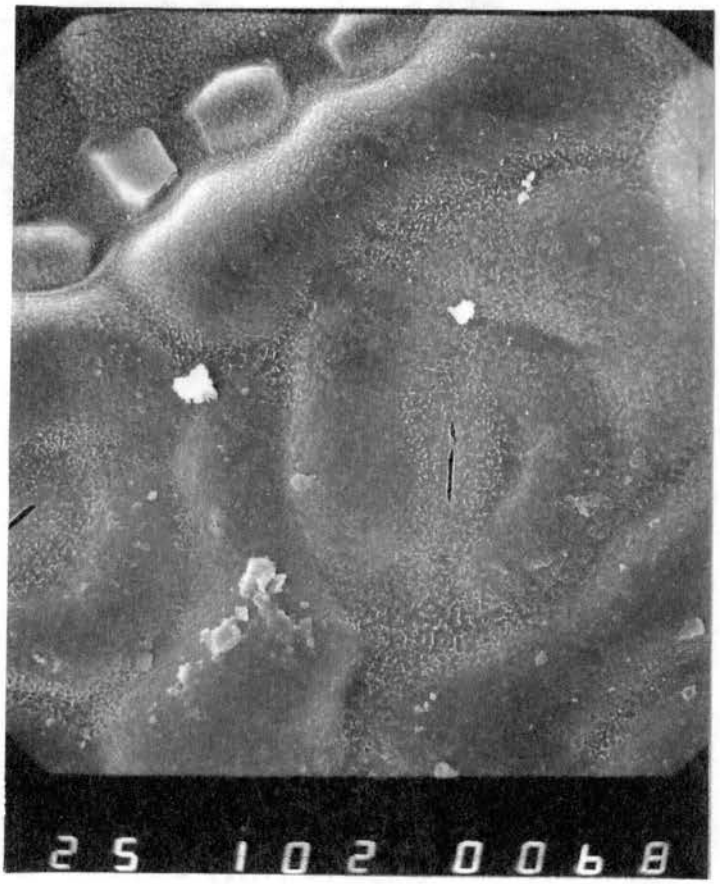

Figure 6. Bentazon (0.84 kg/ha in 140 L/ha water carrier) and crop oil concentrate (2.3 L/ha) applied to peanut. Magnification 1000x.

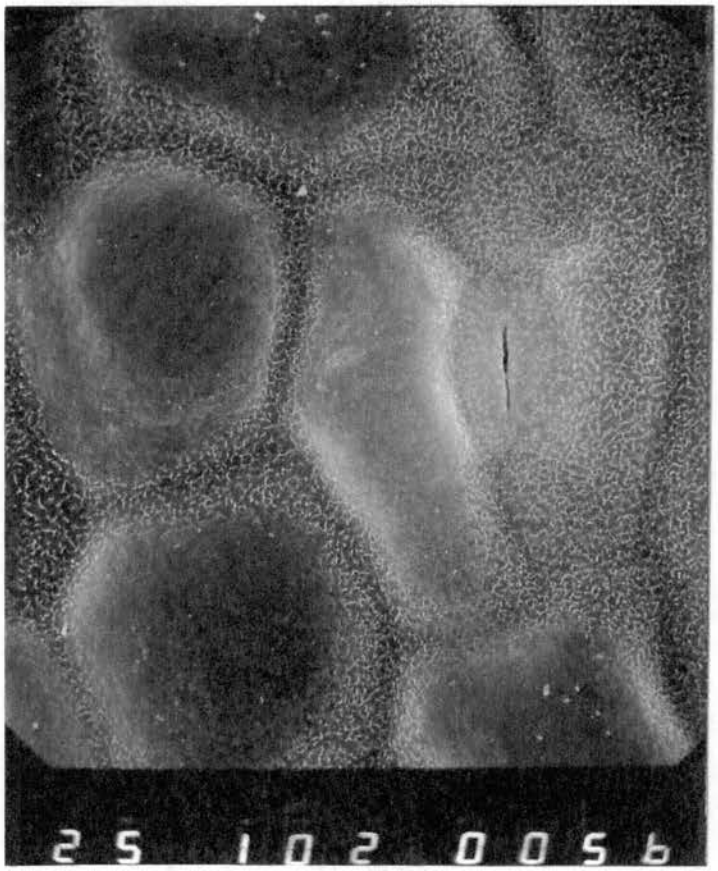

Figure 7. 2,4-DB (0.45 kg/ha in 140 L/ha water carrier) applied to peanut. Magnification 1000x.

CHAPTER II

EFFECT OF POSTEMERGENCE HERBICIDES ON (*CERCOSPORA ARACHIDICOLA*)

EFFECT OF POSTEMERGENCE HERBICIDES ON

(CERCOSPORA ARACHIDICOLA)

Abstract. Early leafspot is a common disease in peanut that is caused by the fungus Cercospora arachidicola Hori. Experiments were conducted in 1991 and 1992 to evaluate the effect of postemergence herbicides on the conidial germination of Cercospora arachidicola Hori. and on the incidence of early leafspot disease in peanut (Arachis hypogaea L.). Conidial germination was enhanced (>100%) at concentrations of 1, 100, 1000, 5000, and 10000 mg/L of bentazon and imazethapyr when compared to the untreated control. Lactofen inhibited conidial germination (decreased 23% compared to control) at concentrations as low as 1 mg/L and completely inhibited germination at concentrations ≥5000 mg/L. Solutions of 2,4-DB had a stimulating effect on conidial germination (>100%) at concentrations ≤1000 mg/L. Concentrations of 10000 mg/L acifluorfen and 2,4-DB completely inhibited conidial germination. The herbicides investigated in these experiments did not increase early leafspot incidence on peanut plants nor did they increase the number of early leafspot lesions per leaflet when compared to diseased peanut which received no herbicide. In fact, some

herbicide treatments reduced early leafspot disease in peanut. Lactofen reduced leafspot incidence 12% and decreased sporulation of lesions 22% from the untreated check. All herbicides decreased sporulation of early leafspot lesions except for bentazon and imazethapyr. Peanut plants treated with 2,4-DB alone did show a trend for decreased early leafspot severity. **Nomenclature:** Acifluorfen, [5-[2-chloro-4-(trifluoromethyl)phenoxy]-2-nitrobenzoic acid]; bentazon, [3-(1-methylethyl)-(1H)-2,13-benzothiadiazin-4(3H)-one 2,2-dioxide]; imazethapyr, [2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1H-imidazol-2-yl]-5-ethyl-3-pyridinecarboxylic acid]; lactofen, [(±)-2-ethoxy-1-methyl-2-oxoethyl-5-[2-chloro-4-(trifluoromethyl)phenoxy]-2-nitrobenzoate]; 2,4-DB, [4-(2,4-dichlorophenoxy)butanoic acid]; peanut, Arachis hypogaea L.; early leafspot, Cercospora arachidicola Hori. **Additional index words:** Herbicide-plant disease interaction, fungus, leafspot incidence, leafspot severity, sporulation.

INTRODUCTION

Weed and disease management are essential for profitable peanut production (7, 22). Weed species composition and accompanying weed management systems vary across peanut growing regions of the U.S. However, a common disease problem throughout the U.S. peanut producing areas is early

leafspot, caused by the fungus Cercospora arachidicola Hori. Early leafspot is considered a polycyclic disease which occurs early or within 3 to 5 wk after peanut planting and persists throughout the growing season (17). Early leafspot can be very destructive and is considered one of the most serious diseases of peanut on a world-wide basis (17). Cultural practices that reduce the initial inoculum can be used to partially manage early leafspot (22, 23); however, properly timed fungicide applications (26) are normally used.

Postemergence (POST)[1] herbicide applications are made to peanut during the early part of the growing season for weed control and may precede or coincide with early leafspot disease. Oklahoma peanut growers rely on POST herbicides to control weeds that germinate after planting and escape earlier control attempts. POST herbicides are often included in peanut weed control programs and are applied following routine soil herbicide applications. The biological activity of herbicides is not restricted to the weed flora but may affect other organisms including plant pathogens. In vitro laboratory studies provide reliable information concerning the fungitoxicity of herbicides against specific pathogens (24).

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[1]Abbreviations: POST, postemergence; COC, crop oil concentrate; NIS, nonionic surfactant; LSI, leafspot incidence; LSS, leafspot severity; SF, degree of sporulation; PAI, potential inoculum availability index.

Certain herbicides have been found to increase plant disease while others have decreased disease incidence (2, 4, 5, 8, 13, 21, 30). Shennan and Fletcher (25) reported that colony growth of selected species of fungi, yeasts, bacteria, actinomycetes, and green algae in vitro, were not inhibited in the presence of 2,4-D [(2,4-dichlorophenoxy)acetic acid] and 2,4-DB at concentrations comparable to field rates of application; however, at concentrations ≥500 mg/L, 2,4-DB was highly toxic and inhibited colony growth, whereas 2,4-D had no effect and disease growth and development was normal.

Screening tests (2) revealed that 25 commercial crop production herbicides stimulated the growth of Rhizoctonia solani in vitro at concentrations up to 1000 mg/L. In these tests, 12 of the 25 herbicides had little effect on the fungus at a concentration of 10000 mg/L while two inhibited fungal growth completely.

The in vitro effect of 2,4-D upon tobacco mosaic virus (TMV) was determined at varied concentrations of 2,4-D (8). Cucumber (Cucumis sativus L.) plants inoculated with TMV-2,4-D mixtures resulted in 38%, 18%, 73%, 66%, and 78% reduction in lesion numbers with concentrations of 1, 5, 25, 125, and 625 mg/L, respectively, indicating an in vitro 2,4-D inhibition of TMV. In another study (18), the fungal growth of C. arachidicola was completely inhibited by 73.5 mg/L aqueous preparation of the acaricide, cyhexatin (tricyclohexyl hydroxystannane).

Several POST herbicides currently used in peanuts cause temporary leaf burn or leaf bronzing (6, 9, 28, 31). Affected peanut leaves have areas of necrotic tissue accompanied by some chlorosis. Injury from these contact herbicides has been reported in other broadleaf crops and injury was temporary due to new growth and there was no effect on yield (11, 14, 16).

The interactions between herbicides and certain plant diseases have been reviewed (3, 15, 29) but, the effects of POST herbicide injury to peanut on the incidence of early leafspot is not well documented. Herbicides have the ability to interact with certain stages of development of any disease organism and may cause an increase, a decrease, or no change in disease severity or disease incidence. This may be the result of morphological and physiological alterations in leaf surface wax characteristics, nutrient composition of the host plants, and a retardation or stimulation of plant growth which alters the coincidence of plant pathogen presence and susceptible growth stages of the host (3).

Katan and Eshel (15) discussed possible mechanisms involved in the increase of disease incidence due to herbicide application as the: a) direct stimulatory effect on the pathogen, b) increased virulence of the pathogen, c) increased susceptibility of the host, and d) suppression of microorganisms antagonistic to the pathogen. They also stated that a decrease in disease incidence due to

herbicides might be a result of the reversal of any one of the previous four mechanisms of disease increase (15).

Oklahoma peanut growers and researchers have expressed concern about the possibility of increased early leafspot disease incidence resulting from POST herbicide use. Knowledge of herbicide activity in peanut and the effects of this activity on early leafspot disease can help peanut growers make decisions that will optimize weed and disease management.

We hypothesized that POST herbicides degrade the peanut leaf surface thus predisposing it to early leafspot. There is limited information on the effect of POST herbicides on C. arachidicola and the disease early leafspot. Therefore, to test our hypothesis, experiments were conducted to investigate the effect of commercially formulated herbicides on the conidial germination of C. arachidicola and on disease parameters of early leafspot disease in peanut.

MATERIALS AND METHODS

Effect on conidial germination. Laboratory experiments were conducted to determine the conidial germination of C. arachidicola in different concentrations of five foliar-applied herbicides commonly used in peanut production and one herbicide being developed for such use. Herbicide treatments included: acifluorfen, bentazon, acifluorfen

plus bentazon (a prepackage commercial mixture)[2], lactofen, imazethapyr, and 2,4-DB.

Conidia of C. arachidicola were obtained from infected peanut plants grown in a greenhouse. Leaflets from plants with mature lesions were placed in petri dishes lined with moist filter paper and incubated at 100% humidity for 2 to 3 days. The leaflets were then placed in minimal amounts of distilled water and agitated to displace conidia from the mature lesions. The concentration of conidia (40000/ml) in suspension was determined with a hemacytometer (10).

The germination of conidia was tested in distilled water and with each herbicide treatment at concentrations of 1, 100, 1000, 5000, and 10000 mg/L using the depression slide technique (24). Each herbicide-conidia solution was pipetted into two wells per slide and replicated four times. Slides were placed into petri dishes that were lined with moist filter paper and covered. This provided a humid environment inside to prevent water from evaporation and drying of wells. Dishes containing the slides were placed into an incubation chamber at a constant temperature of 27 C to promote germination (1). Slides were removed 72 h after the initiation of the experiment and observed under a microscope. Conidia were counted in each depression well

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[2]Storm® (a mixture of 159 g/L acifluorfen and 320 g/L bentazon). BASF Corp., 100 Cherry Hill Rd., Parsippany, NJ 07054.

at four different microscope grids and percent germination was calculated for each treatment.

Data analysis. Experiments had a randomized complete block design and were conducted twice. Data were subjected to analysis of variance and mean separation was done with a protected Least Significant Difference (LSD) Test at the 0.05 probability level. The analysis of variance for each experiment indicated no time by treatment interaction, therefore data presented are the pooled means of each herbicide treatment expressed as the percentage of conidia germination in comparison with germination of viable conidia in distilled water (control). Similar presentations of this nature have been made previously (18, 25).

Effect on disease parameters. In the greenhouse, peanut cultivars that are highly susceptible to early leafspot disease do not perform well when subjected to disease experiments. Early leafspot disease is accelerated in the warm, moist environment of the greenhouse and highly susceptible peanut cultivars rapidly defoliate due to the disease, thus hindering data collection. The runner-type peanut cultivar 'Okrun', is less susceptible to early leafspot disease than other peanut cultivars grown in Oklahoma and therefore was used in these experiments. Seed were germinated on moist paper towels at 29 C for 48 h prior to planting in 12 cm diameter pots containing a mixture of soil, sand, and finely shredded peat (1:1:2,

v/v/v). Pots were placed in a greenhouse maintained at 28 C during the day and 22 C at night. A randomized complete block design with four replications was used with each experiment.

Four wk after planting, individual peanut plant main stems and horizontal branches were marked with string and a water-resistant permanent marker[3] to indicate the growing points. The number of peanut leaflets on each plant was counted and recorded. Peanut plants were then treated with POST herbicides using a laboratory table sprayer equipped with an 8002 even flat fan[4] nozzle delivering 140 L/ha. Herbicide treatments included: acifluorfen at 0.56 kg ai/ha, bentazon at 0.84 kg/ha, acifluorfen plus bentazon at 0.28 and 0.56 kg/ha, imazethapyr at 0.071 kg/ha, lactofen at 0.21 kg/ha, 2,4-DB at 0.45 kg/ha, acifluorfen plus 2,4-DB at 0.28 and 0.15 kg/ha, and bentazon plus 2,4-DB at 0.43 and 0.15 kg/ha. Crop oil concentrate[5] (COC)[1] was added to the bentazon treatment at 1.25% (v/v) of the spray volume. Other treatments, except 2,4-DB alone, were applied with a nonionic surfactant[6] (NIS)[1] at 0.25% (v/v).

One wk after herbicide treatment, peanut plants were

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[3]Sharpie. Sanford Corporation, Belwood, IL 60104.

[4]Spraying Systems Co. Wheaton, IL 60187.

[5]Cornbelt Crop Oil Concentrate, Cornbelt Chemical Co., P.O. Box 410 McCook, NE 69001.

[6]Triton AG-98®. Rohm and Haas Co., Independence Mall W., Philadelphia, PA 19105.

inoculated with C. arachidicola conidia previously obtained from infected peanuts grown in the greenhouse. Conidia were suspended (2 x 10^4 conidia/ml) in 100 ml of distilled water and Amway[7] all purpose surfactant (2 drops/100 ml of water). The abaxial and adaxial surfaces of the peanut leaflets were misted with the conidial suspension using an atomizer[8] following the procedures of previous investigations (10, 20). Plants were placed into an environment controlled chamber and maintained in the dark for 96 h at 21 C and 100% relative humidity, then maintained with a 12 h light cycle for 72 h at 27 C and 60% RH. Plants were removed from chambers and returned to the greenhouse for 1 wk.

Leaflets that were treated with herbicide were sampled 2 wk after inoculation and placed into petri-dishes (100% relative humidity) for 4 d to promote sporulation of early leafspot lesions. The total number of lesions and sporulated lesions per leaflet were counted and recorded. The disease parameters for each treatment were:

a) Leafspot incidence = no. of treated leaflets with lesions/total no. of treated leaflets;

b) Leafspot severity = total no. of lesions/total no. of treated leaflets with lesions;

c) Degree of sporulation = no. of sporulated lesions/total

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[7]Amway Corp., Ada, MI 49301.

[8]DeVilbiss No. 152 atomizer. The DeVilbiss Company, Somerset, PA 15501.

no. of lesions;

d) Potential inoculum availability index = LSS * SF.

The PAI indicates how each herbicide treatment affects available inoculum for secondary infection cycles. A similar index was used by Melouk et al. (19) to determine genotype reactions to leafspot.

Data analysis. All data from three separate experiments were analyzed following standard procedures for analysis of variance and means were separated using a protected LSD Test at a 0.10 probability level. The analysis of variance indicated no experiment by treatment interaction, therefore data from all three experiments were pooled in analysis.

RESULTS AND DISCUSSION

Effect on conidial germination. Treatments of acifluorfen plus bentazon, bentazon, and imazethapyr did not inhibit the germination of conidia at any concentration with the exception of acifluorfen plus bentazon at 100 mg/L where slight inhibition did occur (Table 1). There was a stimulation in conidial germination at all concentrations of bentazon and imazethapyr solutions. Concentrations of ≤1000 and ≤5000 mg/L of 2,4-DB and acifluorfen, respectively, also stimulated conidial germination. However, concentrations >1000 and >5000 mg/L were dramatically inhibitory. Shennan and Fletcher (25) reported similar results with various species of fungi and

MCPA [(4-chloro-2-methylphenoxy)acetic acid] at different concentrations. They suggested that certain species of fungi utilized the herbicide as an additional carbon source. Altman (2) reported that numerous herbicides stimulated the growth of Rhizoctonia solani in vitro and that the fungus possibly utilized the herbicides as a source of energy.

When comparing 10000 mg/L solutions of acifluorfen plus bentazon and acifluorfen alone, there was a significant difference in the percent germination (Table 1). It appears that the addition of bentazon to the solution safened or reduced the inhibitory effect that acifluorfen exhibited on conidia when used alone. However, due to the ratio of acifluorfen in the acifluorfen plus bentazon treatment, the actual concentration of acifluorfen is lower as compared to acifluorfen alone at 10000 mg/L.

Lactofen inhibited conidial germination at concentrations as low as 1 mg/L with only 77% of the viable conidia germinating and completely inhibited germination at concentrations ≥5000 mg/L. Visual observations with a microscope of conidia in 10000 mg/L solutions of lactofen, 2,4-DB, and acifluorfen revealed that plasmolysis of individual conidial cells had occurred (Baysinger personal observation). Similarly, plasmolysis of conidial cells of Drechslera sorokiniana (Sacc.) Subram. & Jain occurred after exposure to 1000 mg/L 2,4-D, 2,4,5-T [(2,4,5-trichlorophenoxy)acetic acid], dicamba (3,6-dichloro-2-

methoxybenzoic acid), and mecoprop [2-(4-chloro-2-methylphenoxy)propionic acid] (13). The possible use of lactofen, 2,4-DB, and acifluorfen in actual field conditions may reduce the initial inoculum of C. arachidicola and possibly reduce the number of fungicide applications needed to manage the disease.

Recommended peanut field use rates of acifluorfen, acifluorfen plus bentazon, bentazon, and 2,4-DB range from 5000 to 10000 mg/L, while lactofen[9] and imazethapyr use rates range from 1000 to 5000 mg/L (based on 187 L/ha spray volume). The range of herbicide concentrations used in this study were equal to or less than that recommended for peanut production (12). According to the results of this study, acifluorfen plus bentazon, bentazon, and imazethapyr would have no effect on conidia present at the time of herbicide application. Therefore, routine fungicide programs would have to be utilized for early leafspot management in peanut. However, these results do indicate the possibility that lactofen, 2,4-DB, and acifluorfen could minimize the germination of conidia present on peanut at time of application.

Effect on disease incidence. Peanut plants with no herbicide (untreated) produced equivalent or higher disease

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[3]Lactofen is not currently registered for use in peanut with the Environmental Protection Agency. Use rates of lactofen were derived from previous experimental trials not mentioned in this report.

parameter values (increased disease incidence) than did herbicide treated peanut plants (Table 2). The LSI in peanut treated with acifluorfen, bentazon, imazethapyr, 2,4-DB, acifluorfen plus 2,4-DB, and bentazon plus 2,4-DB were not significantly different from the untreated peanut check. There was a decrease in LSI of peanut treated with acifluorfen plus bentazon (9%) or lactofen (12%) compared to the untreated check. However, the LSI of peanut treated with lactofen was not significantly different from the other herbicide treatments with the exception of 2,4-DB which was only 3% less than the untreated check.

Peanut injury (leaf burn) did occur after acifluorfen, acifluorfen plus bentazon, acifluorfen plus 2,4-DB, and lactofen treatments. Peanut leaf tissue necrosis was more severe with acifluorfen alone, acifluorfen plus bentazon, and lactofen treatments. The damaged and reduced area of healthy peanut leaf tissue may partially explain the decrease in LSI (disease incidence). Conidia of C. arachidicola appear to germinate on and infect healthy peanut leaf tissue as indicated with the untreated peanut in this experiment. Due to this reduction in healthy leaf tissue, conidia may not have survived on necrotic leaf tissue, thus reducing the infection process of the disease cycle.

The LSS of any herbicide treated peanut was not significantly different from the untreated check (Table 2). Although peanut treated with bentazon had the highest

number of leafspot lesions (LSS = 6.6), this alone does not imply that bentazon promotes increased early leafspot disease in peanut. The SF and PAI values need to be considered and for bentazon treated peanut, those two values were numerically lower than those of untreated peanut. The PAI value in this experiment describes the overall effect bentazon will have on inoculum availability for secondary disease cycles of early leafspot.

Peanut treated with 2,4-DB showed a trend for decreased leafspot lesions (LSS = 4.6). An explanation for the decrease in LSS is not immediately apparent. The auxin activity in the peanut plant due to 2,4-DB presence may be producing an inhibitory affect on the pathogen. There are no reports of 2,4-DB toxicity to C. arachidicola; however, Shennan and Fletcher (25) reported that 2,4-DB was highly toxic to selected species of fungi, yeasts, and bacteria in vitro at concentrations greater than 500 mg/L.

Leafspot lesions present on peanut leaflets treated with acifluorfen, acifluorfen plus 2,4-DB, or lactofen had the lowest SF values (<53%) when compared to other herbicide treated leaflets. The reduction in sporulation is epidemiologically important because it may reduce the apparent infection rate or the rate of disease increase (27). Leafspot lesions on untreated peanut leaflets produced the highest SF value (69%) and had a high PAI (4.1) value.

This study did not deal with different inoculum

densities, nor did it deal with variable rates of the herbicides used. Also, this study investigated only one disease cycle (monocyclic) of early leafspot in which the amount of sporulation was measured to predict the possible effect of herbicides on the secondary inoculum potential. The results of this research were negative in support of our hypothesis which was the assumption that POST herbicides cause an increase in early leafspot disease. It can be concluded that the POST herbicides investigated in this experiment did not increase disease.

In actual field situations, inoculum densities, environmental conditions, and several disease cycles may have an effect on the amount of disease. However, if the results of these experiments can be repeated in the field, peanut growers will be able to select a herbicide that will reduce the amount of disease and available inoculum or at least have some assurance that the use of a POST herbicide will not cause them greater problems from foliar diseases. Pesticide use and cost per acre may be decreased if this integrated pest management approach is utilized.

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Table 1. Effect of herbicides at different concentrations on the germination of C. arachidicola conidia in vitro after 72 h[a].

Herbicide	mg ai/L				
	1	100	1000	5000	10000
	% of Untreated				
Acif.+Bent.[b]	102	88	129	119	100
Bentazon	106	100	133	113	119
Imazethapyr	117	117	119	115	108
Lactofen	77	58	13	0	0
2,4-DB	146	158	163	8	0
Acifluorfen	96	104	171	144	0
Untreated[c]	100	100	100	100	100
LSD (0.05)	8				

[a]Means presented for each concentration are a percentage of the control.

[b]Acif. = acifluorfen, Bent. = bentazon.

[c]Viable conidia in distilled water.

Table 2. The effect of postemergence herbicides on the incidence, severity, degree of sporulation, and potential available inoculum of Cercospora arachidicola Hori. in peanut.

Herbicide[a]	Additive[b]	Rate (kg ai/ha)	LSI[c]	LSS[d]	SF[e]	PAI[f]
Acifluorfen	NIS	0.56	0.59 abc[f]	5.6 ab	0.52 cd	2.9 ab
Bentazon	COC	0.84	0.62 abc	6.6 a	0.61 ab	4.0 ab
Acif.+ Bent.	NIS	0.28 + 0.56	0.57 bc	5.2 ab	0.59 bc	3.1 ab
Imazethapyr	NIS	0.071	0.59 abc	6.2 ab	0.63 ab	3.9 ab
Lactofen	NIS	0.21	0.54 c	5.2 ab	0.47 d	2.4 b
2,4-DB	None	0.45	0.63 ab	4.6 b	0.59 bc	2.7 ab
Acif.+2,4-DB	NIS	0.28 + 0.15	0.58 abc	5.9 ab	0.52 cd	3.1 ab
Bent.+2,4-DB	NIS	0.43 + 0.15	0.60 abc	5.4 ab	0.59 bc	3.2 ab
Untreated	None	None	0.66 a	6.2 ab	0.69 a	4.3 a

[a]Acif. = acifluorfen, Bent. = bentazon.

[b]NIS=nonionic surfactant (0.25% v/v), COC=crop oil concentrate (1.25% v/v).

[c]LSI=Leafspot incidence (no. treated leaflets with lesions/total no. treated leaflets).

[d]LSS=Leafspot severity (total no. lesions/total no. treated leaflets with lesions).

[e]SF=Degree of sporulation (no. sporulated lesions/total no. lesions).

[f]PAI=Potential available inoculum (LSS * SF).

[g]Letters within columns indicate significant differences (P≤0.10) using protected LSD comparisons.

VITA

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Thesis: EFFECT OF POSTEMERGENCE HERBICIDES ON (CERCOSPORA ARACHIDICOLA), THE CAUSAL AGENT OF EARLY LEAFSPOT DISEASE IN PEANUT (ARACHIS HYPOGAEA)

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