Chloroacetamide Herbicides and Chlorimuron Do Not Predispose Peanut (*Arachis hypogaea*) to Stem Rot (*Sclerotium rolfsii*)[1]

W. Carroll Johnson, III*, Timothy B. Brenneman, and Benjamin G. Mullinix, Jr.[2]

ABSTRACT

Field studies were conducted from 1991 through 1993 at Plains, GA to determine the effect of chloroacetamide herbicides and chlorimuron on incidence of stem rot in peanut. The experimental site had a cropping history of more than 25 yr in continuous plantings of peanut and extremely high incidence of stem rot. Incidence of stem rot and peanut yield were not affected ($P \leq 0.05$) by chloroacetamide herbicides in 1991, 1992, and 1993. Chlorimuron applied 60 d after emergence had no effect on stem rot incidence or peanut yield. Paired plots treated with thifluzamide had very low stem rot incidence and 27% greater peanut yields. Based on these studies, neither chloroacetamide herbicides nor chlorimuron predisposed peanut to stem rot.

Key Words. Alachlor, herbicide-disease interaction, metolachlor, thifluzamide.

It has been proposed since the 1950's that the use of herbicides affect the severity of diseases of peanut (*Arachis hypogaea* L.). Chappell and Miller (6) demonstrated *in vitro* that dinoseb [2-(1-methylpropyl)-4,6-dinitrophenol] and PCP (pentachlorophenol) reduced growth of five pathogenic fungi and one plant parasitic nematode. When expanded into field studies, dinoseb reduced stem rot (*Sclerotium rolfsii* Sacc.) and sting nematode (*Belonolaimus gracilis* Steiner), while dinoseb and PCP reduced Cercospora leaf spot. However, herbicide injury has been speculated to increase diseases of peanut, particularly seedling disease. Boyle *et al.* (5) reported that the initial widespread use of sesone [2-(2,4-dichlorophenoxy)ethyl sodium sulfate] in Georgia was correlated with severe reductions in peanut stand from seedling diseases. Although results from greenhouse tests were inconclusive, the authors suggested that complex interactions existed among peanut seed quality, herbicide injury, and incidence of seedling disease.

Most of the reports of herbicides affecting incidence of plant diseases have been with preplant herbicides and seedling diseases. Dinitroaniline herbicides inhibited ethylene production by host plants and increased resistance to Fusarium root rot (8). However, field studies in Michigan illustrated that the use of EPTC (*S*-ethyl dipropyl carbamothioate) increased Fusarium-root rot in navy bean (*Phaseolus vulgaris* L.) (24). Increased incidence of Fusarium root rot was due in part to greater chlamydospore production in the presence of EPTC (9).

Dinitroaniline herbicides affected incidence of diseases caused by *Rhizoctonia* in vegetable crops and cotton (*Gossypium hirsutum* L). Applications of trifluralin [2,6-dinitro-*N*,*N*-dipropyl-4-(trifluoromethyl) benzenamine], nitralin [4-(methylsulfonyl)-2,6-dinitro-*N*,*N*-dipropylaniline], butralin [4-(1,1-dimethylethyl)-*N*-(1-methylpropyl)-2,6-dinitrobenzenamine], and dinitramine (N,N^3-diethyl 2,4-dinitro-6-trifluoromethyl-1,3-benzenediamine) increased host resistance to *Rhizoctonia* in eggplant (*Solanum melongena* L.), tomato (*Lycopersicon esculentum* Mill.), and pepper (*Capsicum* spp.) (11). In contrast, benefin [*N*-butyl-*N*-ethyl-2,6-dinitro-4 (trifluoromethyl) benzenamine] and isopropalin [4-(1-methylethyl)-2,6-dinitro-*N*,*N*-dipropylbenzenamine] had no effect.

Trifluralin inhibited *R. solani* on cotton in greenhouse studies, but in field studies trifluralin injured cotton and increased losses from seedling disease (17). Chandler *et al.* (7) conducted similar studies and reported that the field interaction between *Rhizoctonia*-induced disease of cotton and trifluralin occurred only when trifluralin was applied at greater than registered rates. Such interactions were due to herbicides injuring the host (17) or altering the soil microflora allowing the pathogen to increase (15). Moustafa-Mahmoud and Sumner (16) found that the interaction between herbicides and *Rhizoctonia* induced seedling disease was due to inhibition of fungicide efficacy.

Stem rot caused by *S. rolfsii* can be affected by herbicides. Atrazine [6-chloro-*N*-ethyl-*N'*-(1-methylethyl)-1,3,5-triazine-2,4-diamine], EPTC, and paraquat (1,1'-dimethyl-4,4'-bipyridinium ion) inhibited the growth of *S. rolfsii in vitro* (19,21,22,23), but only at concentrations that exceeded registered rates. When EPTC was applied to ladino clover in greenhouse studies, the incidence of diseases from *S. rolfsii* was increased (23). In contrast, dinitramine at high rates reduced stem rot of peanut under field conditions (10). Similarly, dinoseb combined with oxadiazon [3-[2,4-dichloro-5-(1-methylethoxy)phenyl]-5-(1,1-dimethylethyl)-1,3,4-oxadiazol-2-(3*H*)-one] reduced stem rot in peanut (4). This was due to a combination of the toxic effect of dinoseb to *S. rolfsii* and alteration of peanut growth habit by oxadiazon making fungal infection less likely.

Stem rot of peanut is capable of causing the greatest yield losses of all diseases of peanut in the United States (3). In 1992, losses of peanut due to stem rot in Georgia were estimated at $53,000,000 (P. F. Bertrand, pers. commun. 1993). Registered fungicides are marginally effective in controlling stem rot, with less than 40% control being the norm (G. B. Padgett, pers. commun. 1994). Rotation with a monocotyledonous crop is recommended in an integrated system to manage stem rot in peanut. However, peanut acreage in the southeastern U. S. has increased and now approximately 60% of the acreage is not rotated to a monocotyledonous crop. Corresponding to the increase in pea-

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[2]Res. Agron. USDA-ARS, Assoc. Prof., and Agric. Res. Statistician, Coastal Plain Exp. Stn., Tifton, GA 31793-0748.

*Corresponding author.

nut acreage has been a regional increase in stem rot and lower peanut yields.

Given the documented cases of herbicides affecting the incidence of plant diseases and greater losses due to stem rot, concerns have been expressed that herbicides may predispose peanut to stem rot. Chloroacetamide herbicides and chlorimuron [2-[[[[(4-chloro-6-methoxy-2-pyrimidinyl) amino]carbonyl]amino]sulfonyl]benzoic acid] are widely used in peanut fields in Georgia. Thus, field studies were initiated in 1991 to determine if chloroacetamide herbicides and chlorimuron affect the incidence of stem rot in peanut.

Materials and Methods

Field experiments were conducted in 1991, 1992, and 1993 at the Southwest Georgia Branch Station in Plains, GA on a Greenville sandy clay loam (clayey, kaolinitic, thermic Rhodic Paleudults). The experimental site had peanut planted continuously for >25 yr. As a result, the incidence of stem rot had developed to high levels. Land was tilled with a disk harrow to a depth of 15 cm and moldboard plowed 23 cm deep 3 mo prior to planting. A tractor-powered vertical action tiller was used to shape seedbeds and incorporate herbicides 8 cm deep.

Cultivar Florunner peanut was planted in paired rows, spaced 71 cm apart with 91 cm between pairs of rows. Seeds were planted 6 cm deep at 112 kg ha^{-1} to produce a final density of approximately 28000 plants ha^{-1}. Planting dates were 24 May 1991, 4 May 1992, and 13 May 1993.

The experimental design was a split plot with four replications. Main plots were a factorial arrangement of 14 combinations of chloroacetamide herbicides and chlorimuron, each applied at registered rates and times of application. Chloroacetamide herbicides included alachlor [2-chloro-*N*-(2,6-diethylphenyl)-*N*-(methoxymethyl)acetamide] applied preplant incorporated (PPI) at 3.4 kg ai ha^{-1}, vegetative emergence (VE) at 2.2 kg ha^{-1}, or sequentially (PPI followed by VE); metolachlor [2-chloro-*N*-(2-ethyl-6-methylphenyl)-*N*-(2-methylethyl)acetamide] applied PPI, VE, or sequentially (PPI followed by VE) at 2.2 kg ai ha^{-1}; and a nontreated control). Chlorimuron treatments included one application 60 d after emergence (DAE) at 0.009 kg ai ha^{-1} and nontreated. X-77, a nonionic surfactant containing alkylarylpolyoxy-ethylene glycols, free fatty acids, and isopropanol (Valent USA, San Ramon, CA 94583-0947), was included with all chlorimuron applications at 0.25% by volume.

Sub-plots were two levels of treatment with a fungicide to control stem rot. Thifluzamide (2',6'-dibromo-2-methyl-4'-trifluoromethoxy-4'-trifluoromethyl-1,3-thiazole-5-carboxanilide) effectively controls stem rot with one application. Fungicide treatments included one application of thifluzamide 60 DAE at 0.6 kg ai ha^{-1} and a nontreated control.

Herbicide and fungicide treatments were applied with a CO_2-pressurized backpack sprayer calibrated to deliver 281 L ha^{-1} at 138 kPa. Pest management and cultural practices were based on recommendations from the Univ. of Georgia Cooperative Extension Service. No herbicides were used other than those applied as treatments. All plots were maintained weed-free by handweeding throughout the season. No fungicides or insecticides were used that would provide incidental control of stem rot.

Peanut canopy development was measured at biweekly intervals with a hand-held multispectral radiometer (Model MRS87, Cropscan Inc., Fargo, ND) set to measure percentage reflectance of 800+13 nm wavelength radiation 2 m above the leaf canopy. The radiometer estimates healthy leaf area (1) or degree of canopy deterioration from foliar disease (18). In addition, Adcock *et al.* (2) reported that readings from a multispectral radiometer were better correlated with herbicide injury on soybean [*Glycine max* (L.) Merr.] than visual evaluations. Similarly, Johnson *et al.* (14) used radiometer readings to quantify peanut growth reductions throughout the season from applications of alachlor plus paraquat.

Stem rot assessment was made twice each season using a procedure developed by Rodriguez-Kabana *et al.* (20). Midseason counts were made 90, 108, and 85 DAE in 1991, 1992, and 1993, respectively and late-season counts were made 123, 134,and 131 DAE in 1991, 1992, and 1993, respectively. Stem rot loci (infected area ≤30 cm) were counted in each plot and converted to percentage infection based on the plot length. Peanut yields were measured by digging and inverting individual plots, followed by threshing 1 wk later with a peanut combine. Yield samples were cleaned to remove foreign material.

Data for stem rot assessment, reflectance, and yield were subjected to analysis of variance to determine significant sources of variation. Differences in stem rot and yield means were determined by using Fisher's Protected Least Significant Difference test. Reflectance data taken at biweekly intervals throughout the season were subjected to regression analysis. Only significant differences ($P \leq 0.05$) will be discussed unless otherwise stated.

Table 1. Effect of chloroacetamide herbicides on stem rot and peanut yield at Plains, GA, 1991-93.

Treatment	Rate	Time of application	Midseason stem rot[a,b]			Late-season stem rot[a,c]			Peanut yield		
			1991	1992	1993	1991	1992	1993	1991	1992	1993
	kg ha^{-1}		%			%			kg ha^{-1}		
Metolachlor	2.2	PPI[d]	21.5	8.3	11.5	25.8	16.7	25.9	4110	6340	3700
Metolachlor	2.2	VE[d]	16.3	11.0	10.3	24.5	16.1	25.1	4000	6160	3540
Metol./metol.[e]	2.2/2.2	PPI/VE	19.2	11.4	8.4	27.2	18.1	23.4	3890	6160	3950
Alachlor	3.4	PPI	17.5	11.2	13.9	26.3	15.8	23.9	4150	6210	3830
Alachlor	2.2	VE	16.9	9.7	8.1	26.3	16.4	24.5	3980	6110	3760
Ala./ala.	3.4/2.2	PPI/VE	14.4	8.5	9.4	17.5	18.9	33.9	4090	6090	3850
Nontreated	-------	------	14.8	9.1	15.1	26.4	17.2	27.2	4260	5450	3840
LSD (0.05)			ns	ns	5.3	ns	ns	7.2	ns	590	400

[a]*Sclerotium rolfsii*. Percentage of 30.5 cm sections of linear row per plot with at least one disease locus.

[b]Midseason stem rot assessments were made 90, 108, and 85 DAE in 1991, 1992, and 1993, respectively.

[c]Late-season stem rot assessments were made 123, 134, and 131 DAE in 1991, 1992, and 1993, respectively.

[d]Abbreviations: PPI = preplant incorporated; VE = vegetative emergence.

[e]Designation for sequential applications.

Table 2. Effect of chlorimuron and thifluzamide on stem rot and peanut yield at Plains, GA, 1991-1993.

Treatment	Rate	Midseason stem rot[a,b]			Late-season stem rot[a,c]			Peanut yield		
		1991	1992	1993	1991	1992	1993	1991	1992	1993
	kg ha^{-1}	%			%			kg ha^{-1}		
Herbicide										
Chlorimuron[d]	0.009	17.3	9.8	11.0	26.4	16.7	26.3	3940	6080	3710
Nontreated	--	16.7	9.9	10.9	23.3	17.4	26.3	4200	6070	3850
LSD(0.05)		ns	ns	ns	ns	ns	ns	ns	ns	ns
Fungicide										
Thifluzamide[d]	0.6	1.0	0.5	0.4	2.2	8.4	10.8	4460	6540	4450
Nontreated	--	33.0	19.3	21.6	47.4	25.7	41.8	3670	5610	3120
LSD 0.05)		3.7	3.7	2.8	3.4	3.4	3.9	310	310	210

[a]*Sclerotium rolfsii*. Percentage of 30.5-cm sections of linear row per plot with at least one disease locus.

[b]Midseason white mold disease assessments were made 90, 108, and 85 DAE in 1991, 1992, and 1993, respectively.

[c]Late-season white mold disease assessments were made 123, 134, and 131 DAE in 1991, 1992, and 1993, respectively.

[d]Chlorimuron and thifluzamide were applied 63, 60, and 60 DAE in 1991, 1992, and 1993, respectively.

Results and Discussion

Analysis of variance indicated significant main effects of chloroacetamides, chlorimuron, and thifluzamide on mid- and late-season stem rot counts, peanut yield, and reflectance. There were no significant interactions for any of the parameters. Data were not analyzed across years due to differences in growing conditions.

Mid- and Late-Season Stem Rot. None of the chloroacetamide herbicide treatments affected mid- or late-season stem rot in 1991 and 1992 (Table 1). In 1993, the nontreated control contained the most stem rot at midseason. Conversely, peanut treated with sequential applications of alachlor, metolachlor, or alachlor VE had the least midseason stem rot. Late-season stem rot counts in 1993 were different. Sequential applications of alachlor had the most late-season stem rot, but did not differ from the nontreated control.

Chlorimuron applied 60 DAE did not affect stem rot in any year, regardless of the rating period (Table 2). Thus, under the production parameters used in this study chlorimuron applications do not increase stem rot in peanut.

Thifluzamide effectively controlled stem rot. Stem rot incidence in the nontreated control averaged 25 and 38% at mid- and late-season, respectively. Thifluzamide controlled stem rot an average of 97.5 and 78.8% at mid- and late-season, respectively. In contrast, efficacy from the standard treatment of PCNB (pentachloronitrobenzene) is rarely greater than 40% (G. B. Padgett, pers. commun. 1994).

Reflectance. Reflectance from the peanut leaf canopy was not affected by chloroacetamide herbicides or chlorimuron throughout the duration of the study (data not presented). These results indicate that none of the herbicides significantly injured peanut by causing stunting or chlorosis. Alachlor, metolachlor, and chlorimuron were applied to irrigated peanut at registered rates, stages of crop growth, and on a soil that was not unusually coarse textured. Under these conditions, injury from these herbicides would be highly unusual.

Thifluzamide did not affect reflectance (data not presented), although stem rot was effectively controlled. Reflectance is not an accurate measurement of stem rot in peanut due to the clumped distribution of this disease in the field and the tendency to kill some plants and not affect adjacent plants. Thifluzamide does not control foliar diseases of peanut, and plant growth regulating (PGR) properties have not been reported. Apparently this is the case on peanut since any PGR or phytotoxic effects would have been apparent in our reflectance readings.

Peanut Yield. Chloroacetamide herbicides had no effect on peanut yield in 1991 (Table 1). In 1992, all chloroacetamide herbicides increased yields when compared with the nontreated control. The reason for the nontreated control having the lowest yield in a weed-free experiment is unknown. In 1993, plots treated with metolachlor PPI followed by metolachlor VE had the greatest yield, while those treated with metolachlor VE had the lowest yield. Other chloroacetamide treatment combinations had no effect on yield. When compared to the nontreated control, none of the chloroacetamide herbicide treatments affected peanut yield in 1993.

Chlorimuron applied 60 DAE did not affect yield in any year of the study (Table 2). These results further support the claim that chlorimuron applied according to registered instructions under optimum growing conditions will not injure peanut.

Peanut treated with thifluzamide produced greater yields than the nontreated control (Table 2). Controlling stem rot with thifluzamide increased yields an average of 27% over the nontreated control.

Chloroacetamide herbicides have a short residual life, especially in the southeastern coastal plain. For example, the half-life of metolachlor in the southern U.S. is 15 to 25

d (12). Symptoms of stem rot of peanut are usually apparent beginning midseason, with infection occurring during pegging and pod formation, 40 to 90 DAE (2,13). At this time, alachlor and metolachlor have likely dissipated. Therefore, it is unlikely that chloroacetamides would have any measurable effect on stem rot of peanut. Our results verify this hypothesis. Chlorimuron injury on peanut is characterized by temporary stunting and slight chlorosis. Chlorimuron applications often coincide with initial observations of stem rot, thus giving the appearance of being loosely correlated. However, our results could find no correlation between stem rot of peanut and applications of chlorimuron.

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