CHAPTER 4. EFFECT OF PLANT GROWTH AND CANOPY MODIFIERS ON SCLEROTINIA BLIGHT OF PEANUT

ABSTRACT

Plant growth modifiers and canopy desiccants were applied to peanut in 1995, 1996 and 1997 to determine their effect on plant architecture and Sclerotinia blight. The effect of natural defoliation was assessed by withholding insecticide treatments for corn earworm each year, and fungicide treatments for leaf spot control in 1996 and 1997. Chlorimuron (8.8 g a.i/ha) applied at 15 cm to row closure was the most consistent in significantly reducing mainstem height and delaying row closure when compared to other growth and canopy modifiers. Prohexadione Ca (140 g a.i./ha) applied at 15 cm to row closure and again 3 wk later (72 g a.i./ha) partially reduced mainstem height and delayed row closure while producing a compact canopy with lateral limbs appressed to the soil surface. Paraquat (105 g a.i./ha) produced foliar burn and thinned the upper plant canopy which resulted in <95% of the soil surface being shaded in 1995. Paraquat applied 1 wk after row closure at 52.5 g a.i./ha (air temp. > 30 C) or 79 g a.i./ha (air temp. ≤30 C) resulted in <75% of the soil surface shaded in 1996. Leaf spot defoliation resulted in <75% of the soil surface being shaded in 1996 while corn earworm defoliation did not change the percentage of soil surface shaded in any year. Drought stress inhibited vine growth and suppressed development of disease in 1997 and no differences in plant growth or disease were found in comparisons of plant growth/canopy modifiers. Treatments with chlorimuron and paraquat resulted in significant reductions in disease incidence at harvest (DIH) and area under the disease progress curve (AUDPC) when compared to the untreated check in 1995. Paraquat significantly reduced yield below that of other treatments in 1995 and produced a similar trend in 1996. Prohexadione Ca had no significant effect on DIH or AUDPC in 1995, but did significantly reduce disease incidence at two location s in 1996 . Yield was not significantly affected by prohexadione Ca for any year. Of the plant growth/canopy treatments, chlorimuron and treatments receiving no leaf spot control gave the most significant reductions in disease

incidence, DIH and AUDPC in 1996. Chlorimuron and treatments without leaf spot control significantly improved yield 724 and 705 kg/ha, respectively, when compared to the untreated check in 1996. Fluazinam alone and superimposed on plant growth/canopy modification treatments increased yield 768 and 1,281 kg/ha in 1995 and 1996, respectively. Iprodione increased yield an average of 337 and 605 kg/ha in 1995 and 1996, respectively. Synergism between fungicides and plant growth/canopy modification treatments was not evident in any year. These results suggest that withholding leaf spot control and use of chlorimuron show potential as disease management tools for reducing losses to Sclerotinia blight.

INTRODUCTION

Sclerotinia blight of peanut, caused by *Sclerotinia minor* Jagger, was first recognized as a pathogen of peanut (*Arachis hypogaea* L.) in 1922 in Argentina (21) and was subsequently found in the United States in 1971 (29). This disease has since spread to the peanut-producing areas of Oklahoma (46) and Texas (50). By 1986, Sclerotinia blight was the most destructive disease affecting peanut in Virginia and Oklahoma (30, 41). Field trials in Virginia over a 4-yr period showed yield losses to this disease were ca. 33% or 1598 kg/ha in naturally infested fields without fungicide treatment (38).

Cool, wet conditions have long been associated with outbreaks of diseases caused by *Sclerotinia* species (10, 16, 18, 26, 31, 48). Dow and Porter (10) reported that the optimum temperature for infection of peanut tissue by *S. minor* lies between 20 and 25 C while 95 to 100% relative humidity were necessary for sclerotial germination. Results from a 16-yr study concluded that rainfall accumulations were highest 6 to 15 days prior to outbreaks of Sclerotinia blight (26). This study also indicated that maximum and minimum air temperatures within the 15-day period prior to disease onset averaged ca. 32 and 20 C, respectively, while maximum and minimum soil temperatures at the 10-cm depth averaged ca. 30 and 25 C, respectively. Lee et al. (19) determined that *S. minor* was inactive when soil

temperatures exceeded 28 C at the 10-cm depth.

The density of the foliar plant canopy directly affects the temperature and moisture conditions in the microclimate beneath the plant canopy and may also interfere with the delivery of fungicide to infection sites (20, 25). Plant canopy density and growth habit have also been implicated in the onset and severity of Sclerotinia diseases (1, 4, 5, 6, 12, 17, 33, 39, 40). According to the aforementioned 16-yr study, the initial onset of disease always occurred when vines were within 15 cm of touching or when vines had overlapped between rows (26). Both Dow et al.(11) and Bailey and Brune (2) reported that mechanical pruning suppressed the incidence of Sclerotinia blight. However, both studies reported yield losses associated with mechanical pruning.

The purpose of this study was to determine the effect of plant growth and canopy modifiers on the onset and severity of Sclerotinia blight of peanut. Natural defoliation brought about by leaf spot and insect feeding were investigated along with chemicals that alter plant architecture.

MATERIALS AND METHODS

Fields in southeastern Virginia with histories of severe losses to Sclerotinia blight were planted to peanut cultivars commonly grown in Virginia (35) and managed according to recommended practices (44). Seeding rates were ca. 123 kg/ha each year. Tests were conducted at the Tidewater Agricultural Research and Extension Center (TAREC) Farm in Suffolk, Virginia in 1995. Field trials in 1996 in were located in Southampton Co., Sussex Co., and the TAREC Farm in Suffolk. Trials in 1997 utilized two locations in Southampton Co. (Statesville and Courtland), one location in Surry Co., and one at the TAREC Farm in Suffolk. All field trials were conducted on soils suited to peanut production (35). Esfenvalerate at 0.017 kg a.i./ha was used for control of corn earworm (13) and chlorothalonil at 1.26 kg a.i./ha was used for control of early leaf spot according to the Virginia leaf spot advisory program (8). Trials at the TAREC Farm in Suffolk were irrigated with 3.8 cm of water on 4 and 25 August in 1995 and 2.5 and 2.0 cm of water on 15 August

and 2 September, respectively, in 1997. The 1996 test was not irrigated. Recommended practices for field evaluation of fungicides were followed in all trials (28). The experimental design consisted of four randomized complete blocks. Plots were four, 10.7-m rows spaced 0.91 m apart. Treatments were applied to the two center rows of each plot while the adjacent outside rows served as buffers. The test at the TAREC Farm in 1996 and 1997 utilized a split-plot design with fungicide treatments in main plots and plant growth/canopy modifiers in subplots.

Iprodione [3-(3,5-dichlorophenyl)-N-(methylethyl)-2,4-dioxo-1-imidazolidine carboxamide] at 1.12 kg a.i./ha and fluazinam [3-chloro-N-(3-chloro-5-trifluoromethyl-2-pyridyl)-2,2,2-trifluoro-2,6-didnitro-p-toluidine] at 0.58 kg a.i./ha, respectively, were applied with one 8010LP nozzle centered over each row to provide complete coverage of plants according to the Virginia Sclerotinia Blight Advisory program (27). Sprays of iprodione included pinolene at 0.16% (v/v). Nozzles were calibrated to deliver 374 L/ha at 234 kPa and a ground speed of 6.28 km/hr. Both iprodione and fluazinam were applied on 9 August, 29 August, and 15 September in 1995 at the TAREC Farm in Suffolk. At the TAREC Farm location in Suffolk in 1996, iprodione and fluazinam were applied 25 July, 16 August, and 12 September. Chlorimuron treated plots received the initial fungicide application at an FDI threshold of 20 (25 July) in 1996. Plots sprayed with paraquat and those not receiving leaf spot control did not receive the 12 September application because disease risk did not reach the FDI 32 threshold. Fluazinam and iprodione were applied on 17 July, 12 August, and 9 September at the Southampton location and fluazinam was applied on 26 July, 20 August, and 13 September at the Sussex location in 1996. In 1997, iprodione and fluazinam were applied on 27 August and 22 September at the TAREC Farm. In Southampton Co., iprodione and fluazinam were applied on 15 September at the Statesville location and on 11 August and 5 September at the Courtland location in 1997. At the Surry location, iprodione and fluazinam were applied on 1 and 26 August and 19 September.

Chlorimuron (2-[[[[(4-chloro-6-methoxy-2-pyrimid-inyl)amino] carbonyl]amino]sulfonyl]benzoic acid), paraquat (1,1'-dimethyl-4,4'bipyridinium ion) and

prohexadione Ca (calcium salt of 3,5-dioxo-4-propionylcyclohexanecarboxylic acid) were applied using three, $D_3$23 (disc-core combination) nozzles per row calibrated to deliver 140 L/ha at 331 kPa and 6.28 km/hr. Sprays of chlorimuron included 0.1 L/ha of Latron B1956® (a non-anionic spray adjuvant). Sprays of prohexadione Ca included 0.4 L/ha of 28% UAN with 0.12 and 0.2 L/ha of Agridex® crop oil concentrate in 1995 and 1996, respectively. Paraquat applications included 0.2 L/ha of SoyOil 937® (a crop oil concentrate).

Chlorimuron at 8.8 g a.i./ha was applied when vines in adjacent rows were 15 cm from row closure each year. Application dates for chlorimuron were as follows: 13 July for the 1995 test; 8, 16, and 18 July for Southampton, TAREC Farm, and Sussex locations in 1996, respectively; 31 July for the TAREC Farm and Courtland location in Southampton Co., 7 August for the Statesville location in Southampton Co, and 30 July for the Surry location in 1997 . Prohexadione Ca at 140 g a.i./ha was applied when vines in adjacent rows were 15 cm from row closure and again 3 wk later at a rate of 72 g a. i./ha. A third application of prohexadione Ca at 72 g a.i./ha was made if excessive vegetative regrowth occurred after the second application. Spray timing for prohexadione Ca was: 13 July and 4 August in 1995; 16 July, 31 July, and 19 August at the Southampton location, 16 July and 7 August at the TAREC Farm location, and 18 July, and 8 August and 29 August at the Sussex location. In 1997, prohexadione Ca was applied on 31 July and 22 August at the TAREC Farm, 7 and 27 August at the Statesville location in Southampton Co, 31 July and 19 August at the Courtland location in Southampton Co., and 30 July and 20 August at the Surry location. In 1995, paraquat was applied at 105 g a.i./ha when vines in adjacent rows were 15 cm from touching between rows. In 1996, paraquat was applied 1 wk after row closure at 79 g a.i./ha if the air temperature was $\leq$30 C and 52.5 g a.i./ha if the temperature was >30 C. Paraquat was not tested in 1997 due to the deleterious yield effects observed in 1995 and 1996 (Fig. 7, 9). Paraquat sprays were applied in 1995 on: 13 July and 4 August. In 1996, paraquat was applied on 26 July and 7 August at the Southampton location, 31 July at the TAREC Farm, and 8 August in Sussex.

Treatments without insecticide for control of corn earworm (*Helicoverpa zea* Boddie)

were tested in 1995, 1996, and 1997, while treatments without fungicide for control of early (*Cercospora arachidicola* Hori) and late (Cercosporidium personatum (Berk. and Curtis) Deighton) leaf spot were evaluated in 1996 and 1997.

Measurements of the mainstem and distance between vines of adjacent rows were recorded at ca. 3-wk intervals in 1995 and 1-wk intervals in 1996 and 1997. Mainstem measurements were recorded from four randomly selected plants per plot. The distance between vines of adjacent rows was measured at the ends of plots. Visual estimates (%) of the soil surface shaded by foliage were recorded weekly. Fields were monitored weekly for initial symptoms or signs of Sclerotinia blight. Upon detection, disease incidence was recorded as the number of infection foci in the two center rows of each plot at ca. 2-wk intervals until harvest. Infection foci exhibited symptoms and/or signs of Sclerotinia blight and included 30.5 cm of row length. Disease incidence data was used to calculate the area under the disease progress curve (AUDPC) using the procedure reported by Shaner and Finney (34). Yield at 7% moisture (w/w) was determined by weighing peanuts harvested from the two center rows of each plot. Statistical analysis of plant measurements, disease incidence, disease incidence at harvest (DIH), AUDPC, and yield were analyzed at $P \leq 0.05$ according to the Waller-Duncan *k*-ratio *t* test (SAS Institute, Inc., Cary, NC). A split-plot analysis was used for certain tests in 1996 and 1997 where fungicide treatments were in main plots and plant growth/canopy treatments were subplots.

RESULTS

1995 Field Trials. The 1995 growing season was hotter and drier than normal for southeastern Virginia. Rainfall in May and June was 2.9 and 2.3 cm above normal, while levels in July, August and September were 7.3, 7.1, and 2.9 cm below normal. Disease onset was first detected on 29 August and steadily increased to epidemic levels prior to harvest (Fig. 6).

Plant growth measurements indicated that one application of chlorimuron significantly suppressed mainstem height and increased the distance between vines in adjacent rows by

24 July which was 11 days after treatment (Table 11). Two sprays of prohexadione Ca significantly suppressed mainstem height by 11 August which was 29 days after the first application and 7 days after the second application. Chlorimuron resulted in an overall inhibition of growth whereas prohexadione Ca allowed growth to continue but shortened internodes. This produced a compact foliar canopy with lateral limbs appressed to the soil surface. Paraquat caused foliar burn and defoliation which resulted in <95% of the soil surface shaded by foliage on 11 August or 29 days after application. This defoliation allowed sunlight to reach the soil surface around the crown of plants until harvest. Disease risk and progress was reduced by defoliation with paraquat (Fig. 6B). Defoliation due to corn earworm feeding resulted in <10% defoliation and did not reduce disease risk or contribute significantly to disease suppression. Chlorimuron and prohexadione Ca did not reduce disease risk in 1995.

Among the plant growth/canopy modification treatments, chlorimuron and paraquat alone significantly suppressed DIH and AUDPC when compared to the untreated check. The effect appeared to be mediated by changes in plant canopy architecture and density (Fig. 7). These changes were believed to allow greater penetration of sunlight to the soil surface, improve air movement inside the foliar canopy, and thereby suppress infection processes by *S. minor*. Disease incidence and AUDPC were similar to the untreated check when prohexadione Ca was used alone. Although defoliation due to corn earworm feeding was light and did not change disease risk, AUDPC was significantly less than the untreated check when treatments were withheld for corn earworm control. Fluazinam, alone or superimposed on plant growth/canopy modification treatments, was generally more effective in reducing DIH and AUDPC than similar applications of iprodione. Fluazinam was the only treatment that significantly improved yield, whereas all paraquat treatments significantly reduced yield.

1996 Field Trials. Overall, the 1996 growing season was characterized as cool and wet throughout southeastern Virginia. Rainfall at the TAREC Farm location in May, June, July and September was 0.5, 0.5, 8.3 and 9.7 cm above normal, respectively. Disease onset occurred in mid-July at all locations following heavy rainfall associated with hurricane

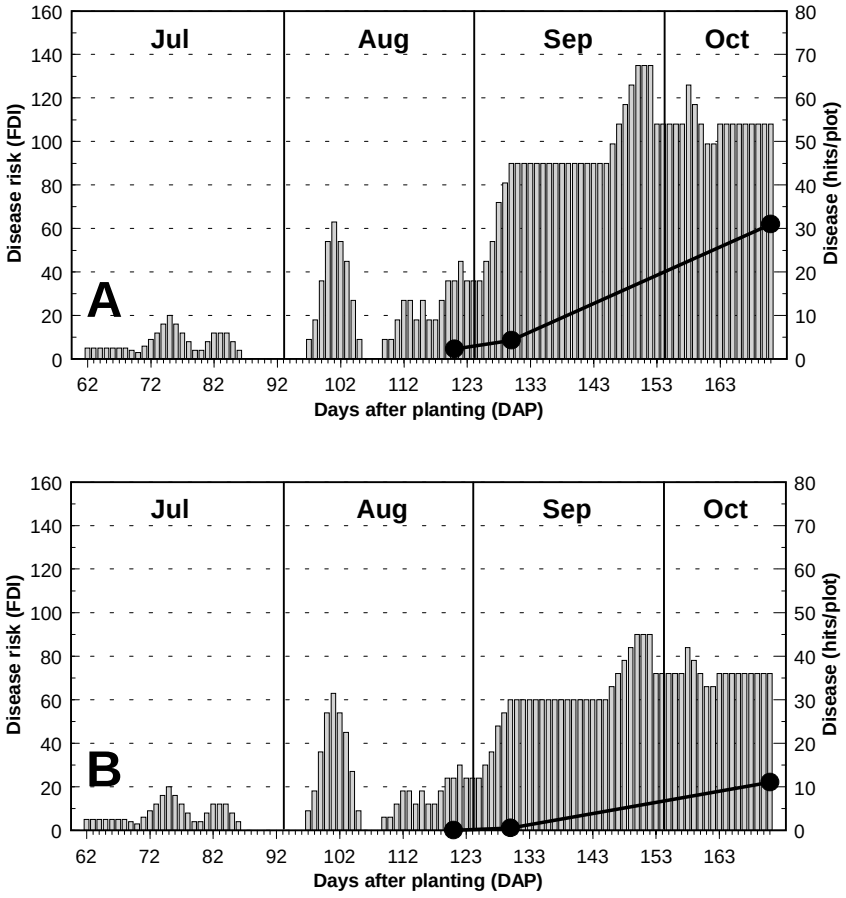

Figure 6. Relationship between disease risk (FDI), as determined by the Virginia Sclerotinia Blight advisory algorithm, to the onset and development of Sclerotinia blight at the Tidewater Agricultural Research and Extension Center Farm (TAREC) in 1995. A) Untreated check and B) paraquat treated.

Table 11. Effect of herbicides and plant growth regulators on peanut mainstem height and the distance between vines in adjacent rows at the Tidewater Agricultural Research and Extension Center (TAREC) Farm in 1995.

Measurement, treatment (a.i./ha) and spray timing[1]	Sampling dates[4]			
1995	3 Jul	24 Jul	11 Aug	24 Aug
Mainstem height[2] (cm)				
Prohexadione Ca 140 g (13 Jul) + 72 g (4 Aug)	13.0 a	22.1 a	25.2 b	29.2 b
Chlorimuron 8.8 g (13 Jul)	11.9 a	18.0 b	21.8 c	24.6 c
Paraquat 105 g (13 Jul, 4 Aug)	12.7 a	21.1 a	30.5 a	32.0 ab
Untreated	13.2 a	23.4 a	31.5 a	34.3 a
Distance between vines in adjacent rows[3] (cm)				
Prohexadione Ca 140 g (13 Jul) + 72 g (4 Aug)	34.3 b	1.2 b	0.0 a	0.0 a
Chlorimuron 8.8 g (13 Jul)	37.3 ab	15.7 a	0.0 a	0.0 a
Paraquat 105 g (13 Jul, 4 Aug)	35.4 b	2.5 b	0.0 a	0.0 a
Untreated	37.9 a	0.0 b	0.0 a	0.0 a

[1] Sprays of prohexadione Ca were applied when vines in adjacent rows were 15 cm from touching and again 3 wk later and included 0.4 L/ha of 28% UAN + 0.12 of Agridex® crop oil concentrate. Chlorimuron was applied when vines between adjacent rows were 15 cm from touching and included 0.1 L/ha of Latron B-1956® (a non-anionic spray adjuvant). Sprays of paraquat were applied when vines in adjacent rows were 15 cm from touching and again 3 wk later and included 0.2 L/ha of SoyOil 937® crop oil concentrate.

[2] Mainstem height recorded from four randomly selected plants per plot.

[3] Determined by measuring the distance between vines in adjacent rows on each end of the two center plot rows.

[4] Means in columns within the same year, date and location with the same letter(s) are not significantly different at $P \leq 0.05$ according to the Waller-Duncan *k*-ratio *t* test.

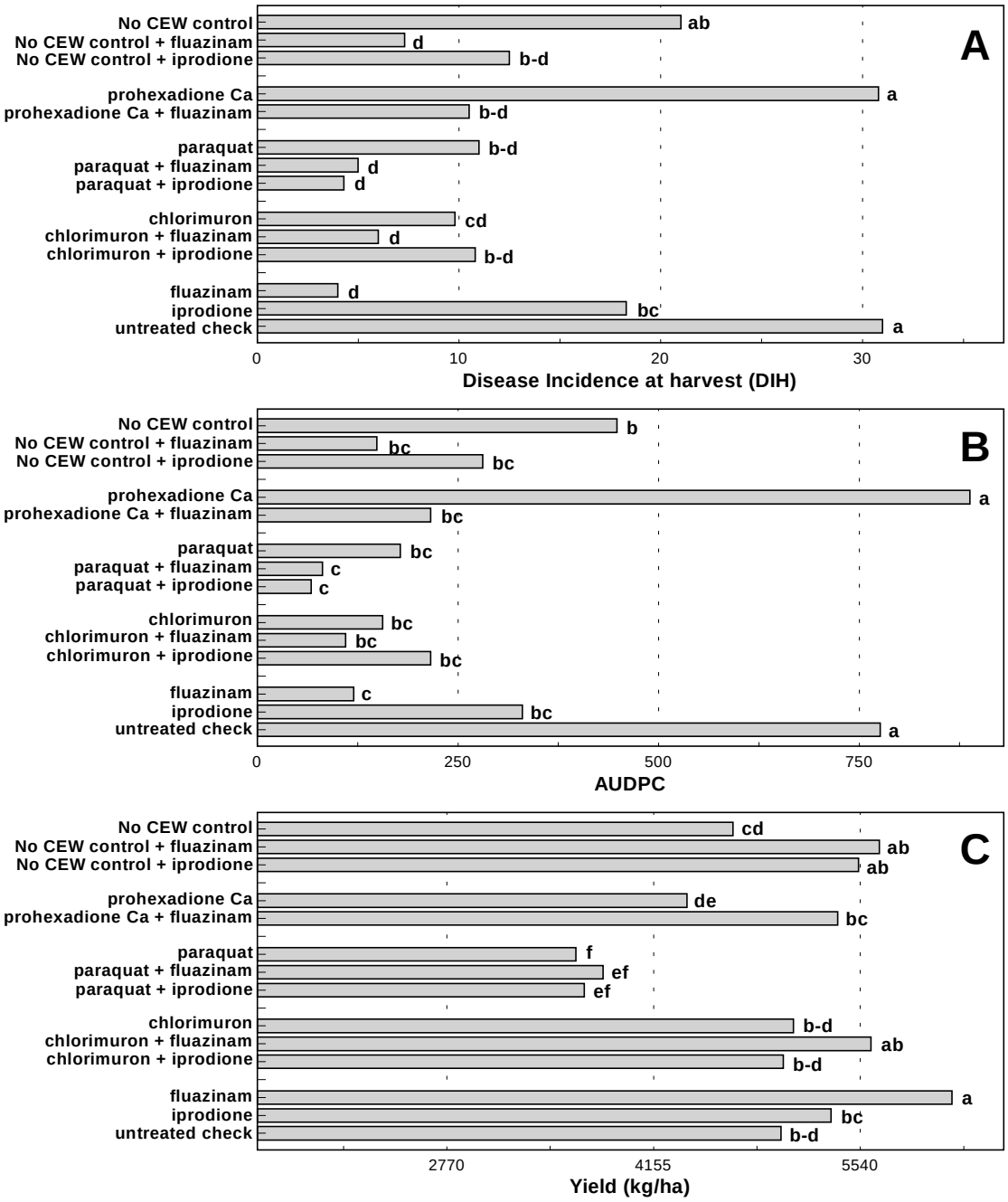

Figure 7. The effect of plant growth/canopy modifiers with and without fluazinam and iprodione on A) disease incidence at harvest (DIH), B) area under the disease progress curve (AUDPC), and C) yield at the Tidewater Agricultural Research and Extension Center Farm (TAREC) in 1995. Bars with the same letter(s) are not significantly different at $P \leq 0.05$ according to the Waller-Duncan *k*-ratio *t* test.

Bertha between 12 and 15 July.

Prohexadione Ca and chlorimuron significantly suppressed mainstem height at the Southampton location by 24 July which was 16 days after the initial applications were made (Table 12). The distance between vines in adjacent rows was significantly greater in plots sprayed with prohexadione Ca and chlorimuron compared to untreated plots by 16 July, which was 8 days after their initial application. Only the chlorimuron treatment resulted in a significantly greater distance between vines in adjacent rows by 24 July. Defoliation by paraquat resulted in <75% of the soil surface shaded by 14 August at the Southampton site. Both prohexadione Ca and chlorimuron significantly suppressed mainstem height by 25 July at the Sussex location, which was only 7 days subsequent to the initial application. By 25 July, chlorimuron was the only treatment to result in a significantly greater distance between vines in adjacent rows at the Sussex location. Paraquat reduced the percentage of the soil surface shaded to <75% by 15 August, which was only 7 days after application at Sussex. Plant growth measurements at the TAREC Farm demonstrated that chlorimuron and prohexadione Ca significantly suppressed mainstem height below that of the untreated check and paraquat treated plots on 25 July, 2 August, and 9 August which was 9, 17, and 24 days after the initial treatment on 16 July. The 9 August observation came 7 days after the second application of prohexadione Ca.

Both chlorimuron and prohexadione Ca applications resulted in a significantly greater distance between vines in adjacent rows by 25 July. Only chlorimuron significantly maintained this condition between vines in adjacent rows on 2 and 9 August at the TAREC Farm. The growth suppression observed with chlorimuron reduced disease risk by 42% and suppressed disease below that of untreated plots (Fig. 8A and B). Treatments with prohexadione Ca suppressed lateral growth of vines, however, the distance between vines in adjacent rows was significantly more than those of plots not receiving plant growth/canopy modifiers only on 24 July and no impact on disease risk progress was detected. Both paraquat and treatments receiving no leaf spot control showed defoliation which reduced the percent soil surface shaded from ≥95% to <75% by 30 August. This reduced disease risk

Table 12. Effect of herbicides and plant growth regulators on peanut mainstem height and the distance between vines in adjacent rows in 1996.

Location, measurement, treatment rate (a.i./ha), and spray timing[1]	Sampling dates[4]			
Southampton	6 Jul	16 Jul	24 Jul	2 Aug
Mainstem height[2] (cm)				
Prohexadione Ca 140g (8 Jul) + 72g (31 Jul) + 72 (19 Aug)	14.6 a	15.5 c	19.9 b	24.5 b
Chlorimuron 8.8 g (8 Jul)	15.9 a	16.8 bc	19.1 b	24.6 b
Paraquat 52.5 g (26 Jul) + 52.5 g (7 Aug)	14.7 a	18.4 b	28.6 a	29.4 a
Untreated	15.5 a	20.1 a	28.9 a	31.4 a
Distance between vines in adjacent rows[3] (cm)				
Prohexadione Ca 140 g (8 Jul) + 72 g (31 Jul) + 72 (19 Aug)	23.1 a	12.1 a	4.1 b	0.0 a
Chlorimuron 8.8 g (8 Jul)	21.3 a	12.7 a	7.3 a	0.0 a
Paraquat 52.5 g (26 Jul) + 52.5 g (7 Aug)	22.2 a	1.3 b	0.0 c	0.0 a
Untreated	22.9 a	1.3 b	0.0 c	0.0 a
Sussex	6 Jul	16 Jul	25 Jul	2 Aug
Mainstem height[2] (cm)				
Prohexadione Ca 140 g (18 Jul) + 72g (8 Aug) + 72 g (29 Aug)	14.0 a	16.9 ab	26.2 b	28.1 c
Chlorimuron 8.8 g (18 Jul)	13.8 a	16.9 ab	25.6 b	27.6 c
Paraquat 79 g (8 Aug)	13.9 a	15.5 b	29.2 a	34.5 b
Untreated	14.3 a	18.2 a	31.6 a	36.4 a
Distance between vines in adjacent rows[3] (cm)				
Prohexadione Ca 140 g (18 Jul) + 72g (8 Aug) + 72 g (29 Aug)	40.3 a	17.8 a	4.1 ab	0.0 a
Chlorimuron 8.8 g (18 Jul)	38.4 a	17.8 a	8.3 a	0.0 a
Paraquat 79 g (8 Aug)	39.7 a	14.9 a	0.0 b	0.0 a
Untreated	42.5 a	18.7 a	1.6 b	0.0 a
TAREC Farm (Suffolk)	16 Jul	25 Jul	2 Aug	9 Aug
Mainstem height[2] (cm)				
Prohexadione Ca 140 g (16 Jul) + 72 g (7 Aug)	16.8 a	16.6 b	19.1 b	22.3 b
Chlorimuron 8.8 g (16 Jul)	17.1 a	16.9 b	19.7 b	22.5 b
Paraquat 79 g (31 Jul)	16.1 a	22.8 a	27.1 a	33.5 a
Untreated	17.3 a	22.7 a	27.2 a	34.0 a
Distance between vines in adjacent rows[3] (cm)				
Prohexadione Ca 140 g (16 Jul) + 72 g (7 Aug)	19.1 a	10.5 a	3.5 b	1.0 b
Chlorimuron 8.8 g (16 Jul)	21.0 a	17.8 a	9.8 a	6.7 a
Paraquat 79 g (31 Jul)	20.3 a	0.0 b	0.0 b	0.0 b
Untreated	18.1 a	0.0 b	0.0 b	0.0 b

[1] Sprays of prohexadione Ca were applied when vines in adjacent rows were 15 cm from row closure and again 3 wk later. A third application of prohexadione was applied if excessive vegetative regrowth occurred after the second application. Prohexadione sprays included 0.4 L/ha of 28% UAN + 0.2 L/ha of Agridex® crop oil concentrate. Chlorimuron was applied when vines in adjacent rows were 15 cm from row closure and included 0.1 L/ha of Latron B-1956® (a non-anionic spray adjuvant). Sprays of paraquat were applied 1 wk after row closure and included 0.2 L/ha of SoyOil 937® crop oil concentrate. A second application of paraquat was needed at the Southampton location because the first spray did not result in substantial foliar burn.

[2] Mainstem height recorded from four randomly selected plants per plot.

[3] Determined by measuring the distance between vines in adjacent rows at each end of the two center plot rows.

[4] Means in columns within the same location with the same letter(s) are not significantly different at $P \leq 0.05$ according to the Waller-Duncan *k*-ratio *t* test.

65% which suppressed disease progress below that of untreated plots at the TAREC Farm (Figs 9A and 9C).

At the Southampton location, all plant growth/canopy modifiers significantly reduced disease incidence on 14 and 27 August when compared to the untreated check which corresponded to significant reductions in AUDPC (Table 13). However, the disease suppression offered was only temporary and significant differences in disease incidence were not detected between plant growth/canopy modifiers and the untreated check at the final rating on 4 October. Both fluazinam and iprodione reduced disease significantly below that of plant growth/canopy modifiers and significantly improved yield. A significant yield reduction was observed with the use of paraquat at the Southampton location.

A similar trend was observed at the Sussex location in that both prohexadione Ca and chlorimuron treated plots, resulted in significant reductions in disease incidence on 15 and 27 August, and AUDPC (Table 13). However, neither treatment reduced disease incidence below that of the untreated check at the final rating. The use of paraquat did not provide significant disease suppression at the Sussex location, but did reduce yield significantly. Fungicides gave both significant disease suppression and yield improvement when compared to plant growth/canopy modification treatments at this site.

A split-plot analysis of data from the TAREC Farm showed significant effects of fungicide treatments and plant growth/canopy modifiers for DIH, AUDPC and yield in 1996. The interaction of fungicide treatment with plant growth/canopy modifier treatments was significant for yield. Significant disease suppression was observed as early as 14 August in plots treated with chlorimuron, paraquat, and those receiving no leaf spot control (Table 13). Chlorimuron and no leaf spot control were the only plant growth/canopy treatments which significantly reduced DIH and AUDPC at the TAREC Farm (Fig. 9 A and B, Table 13). Both fluazinam and iprodione consistently reduced DIH and AUDPC in 1996 when compared to treatments not receiving these fungicides. Chlorimuron and treatments receiving no leaf spot control significantly improved yield 724 and 705 kg/ha, respectively, when compared to the untreated check. Overall, fluazinam increased yield an average of

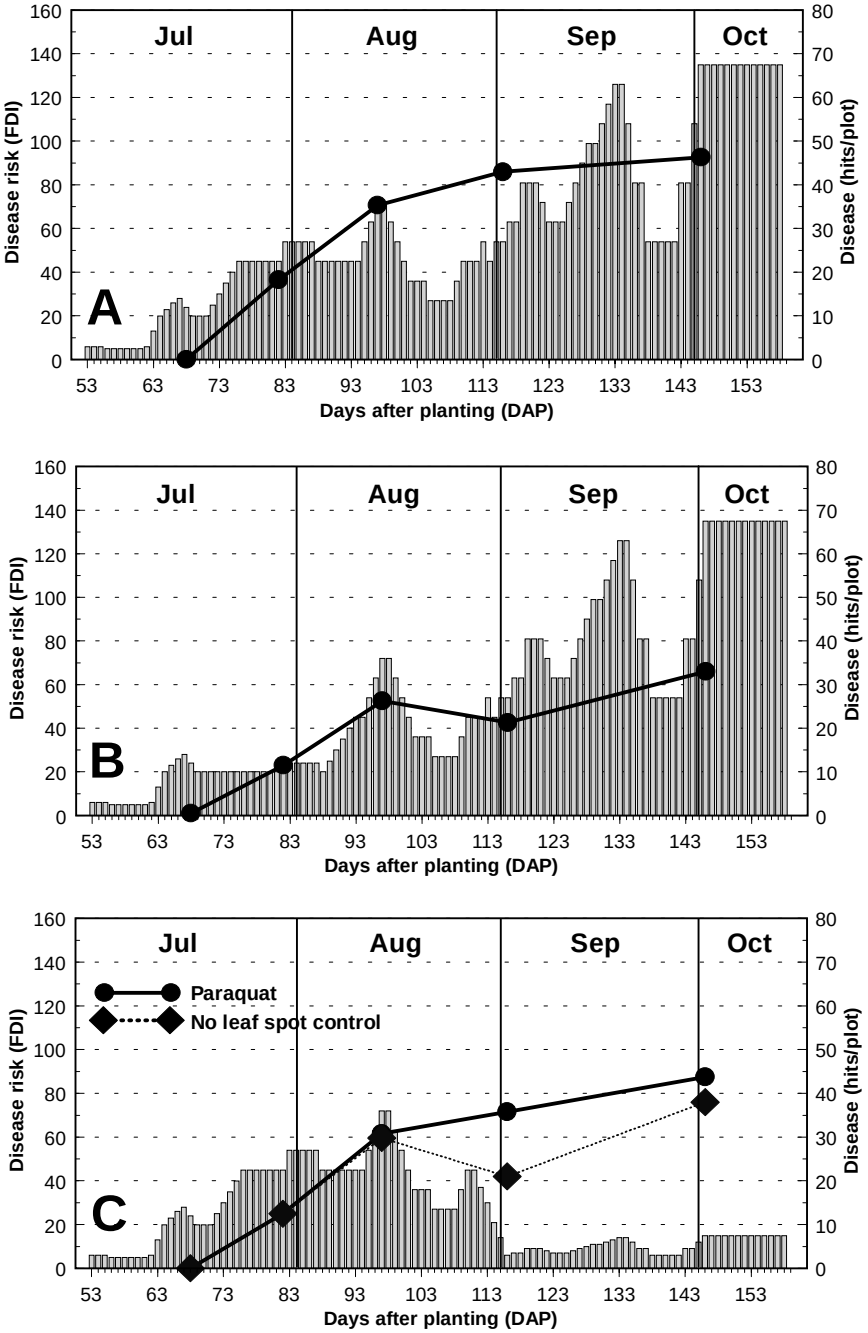

Figure 8. Relationship between disease risk (FDI), as determined by the Virginia Sclerotinia blight advisory algorithm, to the onset and development of Sclerotinia blight at the Tidewater Agricultural Research and Extension Center Farm (TAREC) in 1996. A) Standard leaf spot control and no plant growth/canopy modifiers, B) chlorimuron and C) paraquat and no leaf spot control.

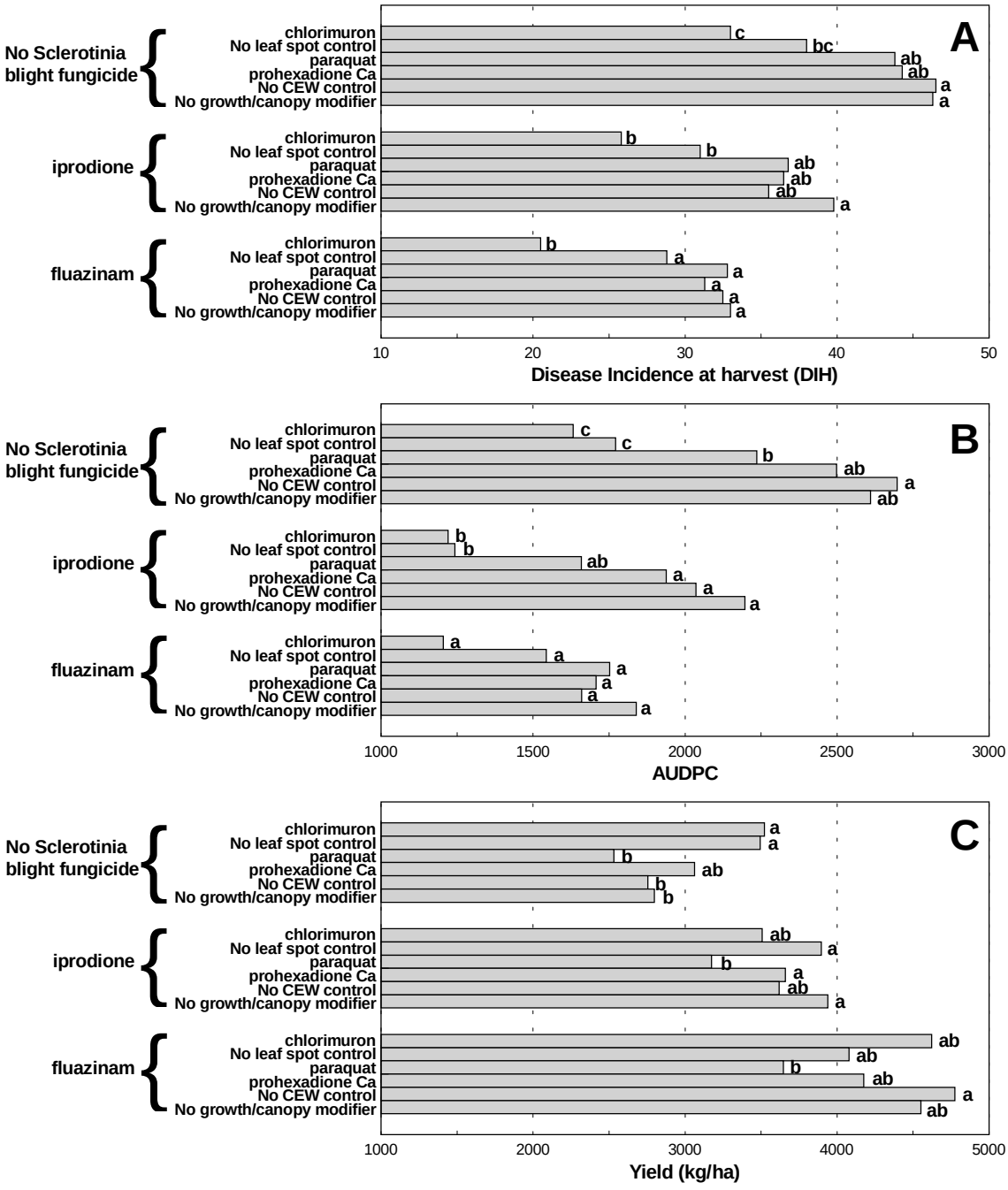

Figure 9. The effect of plant growth/canopy modifiers with and without fluazinam and iprodione on A) disease incidence at harvest (DIH), B) area under the disease progress curve (AUDPC), and C) yield at the Tidewater Agricultural Research and Extension Center (TAREC) Farm 1996. Bars in a fungicide treatment group with the same letter(s) are not significantly different at $P \leq 0.05$ according to the Waller-Duncan *k*-ratio *t* test.

Table 13. Effect of plant growth/canopy modifiers on disease incidence, area under the disease progress curve (AUDPC), and yield of peanut in 1996.

Location, treatment (a.i./ha), and spray timing[1]	Sclerotinia blight[2]						AUDPC	Yield (kg/ha)[3]
Southampton	17 Jul	30 Jul	14 Aug	27 Aug	13 Sep	4 Oct		
Prohexadione Ca 140 g (8 Jul) + 72 g (31 Jul, 19 Aug)	4.8 a	18.3 a-c	31.5 bc	44.3 b	50.0 a	50.8 a	2874 b	2893 c
Chlorimuron 8.8 g (8 Jul)	8.8 a	17.0 a-c	36.0 b	43.5 b	50.5 a	49.8 a	2933 b	2968 c
Paraquat 52.5 (26 Jul, 7 Aug)	4.8 a	22.0 ab	35.8 b	41.0 b	49.8 a	50.0 a	2925 b	2189 d
Fluazinam 0.58 kg (17 Jul, 12 Aug, 13 Sep)	3.8 a	9.5 c	14.8 d	24.0 d	25.8 c	28.3 c	1510 d	5127 a
Iprodione 1.12 kg (17 Jul, 12 Aug, 13 Sep)	5.5 a	14.8 bc	24.5 c	30.8 c	42.5 b	45.5 b	2332 c	3778 b
Untreated check	8.5 a	26.0 a	45.0 a	51.0 a	49.3 a	53.3 a	3309 a	2773 c
Sussex	18 Jul	31 Jul	15 Aug	27 Aug	13 Sep	27 Sep		
Prohexadione Ca 140 g (18 Jul) + 72 g (8 Aug, 29 Aug)	1.3 a	20.8 ab	37.3 b	42.0 b	45.3 a	45.0 ab	2562 b	1754 b
Chlorimuron 8.8 g (18 Jul)	1.0 a	18.0 ab	38.3 b	37.8 b	44.0 a	45.8 a	2459 b	2093 b
Paraquat 79 g (8 Aug)	0.3 a	17.8 ab	48.5 a	43.8 ab	48.3 a	52.8 a	2808 ab	1032 c
Fluazinam 0.58 kg (26 Jul, 20 Aug, 13 Sep)	1.5 a	13.5 b	20.8 a	23.8 c	30.8 b	36.0 b	1652 c	4497 a
Untreated check	1.5 a	21.3 a	44.0 c	49.0 a	53.5 a	46.3 a	2914 a	1655 b
TAREC Farm (Suffolk)	16 Jul	30 Jul	14 Aug	2 Sep	2 Oct			
Prohexadione Ca 140 g (16 Jul) + 72 g (7 Aug)	0.5 a	13.0 a	32.0 bc	45.0 a	44.3 ab	-	2499 ab	3063 cd
Chlorimuron 8.8 g (16 Jul)	0.5 a	11.5 a	26.3 cd	21.3 c	33.0 c	-	1632 e	3522 bc
Paraquat 79 g (31 Jul)	0.0 a	12.5 a	30.8 bc	35.8 ab	43.8 ab	-	2236 bc	2534 d
No corn earworm control	0.8 a	19.5 a	39.5 a	42.5 a	46.5 a	-	2698 a	2756 d
No leaf spot control	0.0 a	12.5 a	29.8 bc	21.0 c	38.0 bc	-	1772 de	3494 bc
Fluazinam 0.58 kg (25 Jul, 16 Aug, 12 Sep)	0.5 a	16.5 a	21.3 d	30.3 bc	33.0 c	-	1840 c-e	4552 a
Iprodione 1.12 kg (25, Jul, 16 Aug, 12 Sep)	0.5 a	11.5 a	29.0 bc	38.3 ab	39.8 a-c	-	2197 b-d	3940 b
Untreated	0.0 a	18.3 a	35.3 ab	43.0 a	46.3 a	-	2611 ab	2798 d

[1] Prohexadione Ca treatments were applied as vines reached 15 cm from row closure and again 3 wk later. Sprays were tank mixed with 0.4 L/ha of 28% UAN and 0.2 L/ha of Agridex® crop oil concentrate. A third application of prohexadione Ca was made if excessive vegetative growth occurred after the second application. Chlorimuron was applied as vines reached 15 cm from row closure and included 0.1 L/ha of Latron B-1956® (a non-anionic spray adjuvant). Paraquat was applied 1 wk after row closure and included 0.2 L/ha of SoyOil 937® crop oil concentrate. A second paraquat application was needed at the Southampton to cause substantial foliar burn and subsequent thinning of the plant canopy.

[2] Disease incidence data are counts of disease foci in the two centers rows of each plot.

[3] Yields are based on weight of peanuts with a 7% moisture content (w/w).
Means in a column with letter(s) in common and within the same location are not significantly different ($P \leq 0.05$) according to the Waller-Duncan *k*-ratio *t* test.

1,280 kg/ha, while iprodione increased yield an average of 605 kg/ha at Suffolk in 1996. Disease incidence at the TAREC Farm location showed that only chlorimuron and no leaf spot control resulted in significant disease suppression while improving yield significantly to levels which were similar to iprodione treated plots.

1997 Field Trials. The 1997 growing season was cool and dry when compared to normal for all test sites. The rainfall deficit totaled 16.8, 20.5, 27 and 29.5 cm for the TAREC Farm, Statesville (Southampton Co.), Courtland (Southampton Co.), and Surry locations, respectively. Drought stress suppressed vine growth, yield, and development of Sclerotinia blight. Plant growth/canopy modifiers only partially affected plant growth and only minor differences were recorded between treatments with no effect on disease incidence or yield when compared to the untreated check (data not shown). Defoliation by corn earworm and leaf spot were also minimal with no effect on disease or yield of peanut. Defoliation due to leaf spot in plots receiving no leaf spot fungicides only averaged 15% at 7 days prior to harvest.

DISCUSSION

Prohexadione Ca is a plant growth regulator that inhibits internode elongation by suppression of gibberellic acid synthesis (24). This product is thought to be an effective replacement for daminozide which was used previously as a growth regulator for improving row visibility at harvest (23). Results from this study showed that prohexadione Ca inhibits internode elongation which significantly reduced mainstem height but only partially delayed row closure when compared to the untreated check. This resulted in a more compact plant with lateral limbs appressed to the soil surface. This compact plant architecture appeared to allow for decreased air flow, increased moisture retention, and reduced sunlight penetration. More limbs appressed to the soil surface may increase the number of infection sites for *S. minor*. Several studies have demonstrated that plants with an upright growth habit are less susceptible to Sclerotinia diseases (1, 5, 6, 12, 17). This may account for the high levels of disease incidence and AUDPC where prohexadione Ca was applied in 1995. These

observations were similar to that of an earlier study which showed that the use of the growth regulator TIBA (2, 3, 5-triiodobenzoic acid), which also reduces internode elongation, caused white mold of Great Northern dry beans to be more severe (7). Although disease levels tended to be higher with prohexadione Ca in 1995, yield was not significantly lower than that of other plant growth/canopy modifiers. However, in 1996, the Southampton and Sussex locations showed significant disease suppression with prohexadione Ca use which may have been due to differences in vine growth between years and locations. In 1995, prohexadione Ca did not significantly increase the distance between vines in adjacent rows when compared to the untreated check, while in 1996 a significant increase was observed.

Chlorimuron is a sulfonylurea herbicide which reduces plant growth through the inhibition of mitosis (49). Like prohexadione Ca, this compound has been investigated in North Carolina and Virginia (22, 42, 43) for maintaining row visibility until harvest. However, reductions in yield and grade have been observed when chlorimuron was applied less than 65 days after emergence (22, 36, 42). Data presented in this study indicated that chlorimuron applied at 74 and 69 days after planting did not adversely affect yield when compared to plots not receiving plant growth/canopy modifiers. In both years, one application of chlorimuron produced an overall growth reduction by significantly inhibiting elongation of the mainstem and lateral limbs more effectively than prohexadione Ca. This reduction in growth did not, however, increase plant canopy density as seen with prohexadione Ca. This difference resulted in a reduction in disease risk in 1996 (Fig. 8) and helped explain the significantly lower disease levels in chlorimuron-treated plots compared to those treated with prohexadione Ca. The more open canopy produced by applications of chlorimuron was thought to create a more unfavorable environment for the development of Sclerotinia blight. Open canopies have been shown to suppress the severity of Sclerotinia diseases of other crops (4, 40).

Paraquat is a contact herbicide for early season weed control in peanuts (45). Applications can be phytotoxic to peanut foliage and reports have shown reductions in canopy width, yield and grade (14 , 47). In this study, paraquat was used to alter the canopy

density through defoliation. This represents the first report to date of the late season defoliating effects of paraquat on peanut. Defoliation by paraquat resulted in <95% of the soil surface shaded in 1995 and <75% of the soil surface shaded at all locations in 1996. Since paraquat is a contact herbicide, leaves in the upper canopy were damaged more than lower leaves. This defoliation corresponded to a significant reduction in disease incidence in 1995 and only a partial reduction in 1996. However, applications of paraquat significantly reduced yields each year. Boote et al. in 1980 (3) reported that the upper 42% of the canopy leaf area was responsible for 74% of light interception and fixes 63% of CO_2. The upper canopy contains younger, more photosynthetically active leaves than those of the lower canopy. Reduced yield prompted the discontinued testing of paraquat as a disease management tool after 1996.

Corn earworm is a late season insect pest of peanut, and larva can consume 175 to 200 cm^2 of peanut foliage before pupation (15). However, these pests rarely reach damaging levels and insecticide treatment is usually unwarranted in Virginia (Herbert, *personal communication*). Data from this study demonstrated a significantly lower AUDPC when corn earworm sprays were not applied in 1995 only. The lack of a disease response in other years was primarily due to the low level of corn earworm infestations encountered. Defoliation by insect feeding may be a useful disease control option in years with excessive vine growth and when corn earworm infestations cause heavy defoliation.

Leaf spot can cause severe defoliation which may result in up to a 50% loss of yield in years with heavy disease pressure (37). However, yield losses are not likely if 40% or less defoliation occurs late in the season (Phipps, *personal communication*). Leaf spot defoliation was evaluated in 1996 and 1997 as a possible mechanism for suppression of Sclerotinia blight. In the 1996 test, withholding fungicide sprays for control of leaf spot significantly reduced DIH and AUDPC while improving yield. Leaf spot defoliation averaged only 15% in 1997 and did not affect disease incidence or yield. This type of defoliation differs from that by paraquat in that leaf spot defoliation occurs primarily on older, lower leaves. The loss of these leaves would be less detrimental to CO_2 fixation and photosynthetic activity (3).

Applications of fungicides, fluazinam or iprodione, improved disease control and yield when used alone or superimposed on plant growth/canopy modification treatments for each location and year. There appeared to be no synergistic effects when the combination of fungicide and plant growth/canopy modifier treatments were used together.

Currently, iprodione is the only registered fungicide for control of Sclerotinia blight of peanut. Studies have shown that this fungicide provides only 31% disease suppression (38). The experimental fungicide, fluazinam, provides up to 60% disease suppression but this product has not been approved for commercial use in the U. S. In this 3-yr study, iprodione and fluazinam averaged 18 and 35% disease suppression, respectively. The use of plant canopy modification by withholding leaf spot fungicide applications has the potential to reduce losses to Sclerotinia blight as well as reduce cost due to reduced fungicide input. However, this method may only be useful in years when the losses to Sclerotinia blight may be more severe than losses to leaf spot. Chlorimuron also shows promise of being an effective disease management tool for Sclerotinia blight. However, it too is of minimal value unless conditions allow for excessive vine growth to be a factor in development of Sclerotinia blight.

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