

The Effect of 2,4-Dichlorophenoxyacetic Acid (2,4-D) on Peanut when Applied During Vegetative Growth Stages

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ABSTRACT

The development of 2,4-D-resistant cotton and soybean cultivars has created great concern about the potential off-target movement of 2,4-D onto sensitive broadleaf crops. Peanut is often grown in close proximity to cotton and soybean. Therefore, field studies were conducted during 2012 and 2013 at Plains, Ty Ty, and Attapulgus, GA to evaluate peanut response to 2,4-D at 67, 133, 266, 533, and 1066 g ae ha⁻¹ applied at preemergence (PRE), 10, 20, or 30 d after planting (DAP), corresponding to PRE, V2, V3, and V5 peanut growth stages. Nontreated controls (NTC) were included for comparison. Treatment timing by rate interactions were significant ($P < 0.0001$). As 2,4-D rate increased peanut injury increased. There was variation in yield loss response dependent on peanut growth stage at application timing. Peanut that was treated preemergence and at the V2 growth stage did not have yield loss at any of the 2,4-D evaluated rates (67 to 1066 g ha⁻¹) relative to the NTC. When peanut was treated at V3 and V5 growth stages with 2,4-D, injury estimates were 5 to 32% from the 67 to 1066 g ha⁻¹ rates respectively, and peanut canopy diameter was stunted 5 to 35% at the same rates. The resulting peanut yield loss was 23 and 36% from 533 and 1066 g ha⁻¹ of 2,4-D applied at V3 and V5 growth stages; in part due to reproductive growth being initiated during that time-frame and peanut had less time to recuperate before harvest. Linear regression models were used to evaluate peanut injury and peanut yield results. Significant correlations were established for V3 and V5 treatments between injury and yield, injury and canopy diameter, and canopy diameter and yield ($P < 0.0001$), with correlation coefficients of -0.48, -0.76, and 0.51, respectively. Growers and extension agents will be able to use these peanut injury estimates and canopy diameter data to make improved predictions of potential peanut yield loss where off-target movement of 2,4-D or sprayer contamination has occurred.

Key Words: *Arachis hypogaea*, peanut, herbicide-resistance, 2,4-D, drift, crop injury, herbicide injury, sprayer contamination, accidental exposure, vegetative growth stages, synthetic auxin, dose response.

Peanut (*Arachis hypogaea* L.) is an important crop to Georgia and many other southern states of the US. Georgia produces the most peanut in the US, with 314,400 ha harvested in 2015 (Anonymous, 2016). Peanut is regularly grown near cotton (*Gossypium hirsutum* L.) and soybean [*Glycine max* (L.) Merr.] across the southeast (Lassiter *et al.*, 2007; Prostko *et al.*, 2011; Prostko *et al.*, 2013). A large percentage of cotton and soybean cultivars have herbicide resistance technology to assist in weed management, and the increased use of glyphosate and glufosinate herbicides throughout the growing season increased the occurrence of accidental injury to peanut crops (Grey and Prostko, 2010; Johnson *et al.*, 2012a; Lassiter *et al.*, 2007). When spray systems are not properly cleaned prior to other applications to different crops, crop injury can occur from herbicide residue remaining in the spray system (Grey and Prostko, 2010; Prostko *et al.*, 2011).

Peanut injury can also occur when the herbicide becomes suspended in air, never reaching the target site, then moving onto nearby peanut (Auch and Arnold, 1978; Grey and Prostko, 2010). Herbicide drift causes and severity can be attributed to many different factors: nozzle type, boom height, pressure, wind speed, spray formulation, volume per area, sprayer speed, and other environmental variables (Wolf *et al.*, 1993). Grover *et al.* (1978) reported that herbicide drift can be 1 to 8% of the spray solution, and Wolf *et al.* (1993) indicated off-target spray drift can reach 16% when no shielding is used.

When applied 75, 90, and 105 d after planting (DAP), 240, 320, and 470 g ai ha⁻¹ of glyphosate caused peanut yield reductions of 12 to 36% (Grey and Prostko, 2010). In another study, glyphosate applied to peanut at 560 g ai ha⁻¹, 4 weeks after planting, resulted in yield loss greater than 50% and peanut injury was directly correlated with yield loss (Lassiter *et al.*, 2007). Glufosinate trials indicated yield loss to peanut when less than

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normal rates of 538 g ai ha⁻¹ were applied. Glufosinate at 135 and 269 g ai ha⁻¹ applied approximately three weeks after emergence (WAE) reduced peanut yield 14 and 51% respectively in North Carolina (Jordan *et al.*, 2011). A significant negative correlation with visual estimates of peanut injury and peanut yield was reported.

The frequency of herbicide-resistant weeds, especially weeds with resistance to acetolactate synthase inhibitor (ALS) (Wise *et al.*, 2009) and glyphosate herbicides (Culpepper *et al.*, 2006) have increased to the extent that some weed management programs are now less effective. Thus, there is an interest in developing other types of herbicide-resistant crops (Johnson *et al.*, 2012b; Subramanian *et al.*, 1997). Agricultural seed and chemical companies have reacted by developing alternative weed management systems that use 2,4-Dichlorophenoxyacetic acid (2,4-D) postemergence (POST) on 2,4-D-resistant crops including cotton and soybean (Egan *et al.*, 2014; Johnson *et al.*, 2012b; Leon *et al.*, 2014). 2,4-D-resistant crops have been genetically altered so that they metabolize the 2,4-D herbicide more readily, thus no injury occurs from application (Johnson *et al.*, 2012b). The proposed 2,4-D-resistant crops will also include herbicide-resistance technology for other herbicide mechanisms of action (MOA) (Johnson *et al.*, 2012b). This benefit will permit the use of several MOA as a means of reducing the selection pressure on a single MOA (Vencill *et al.*, 2012), as well as assist in management of current herbicide-resistant weeds.

2,4-D is known to cause significant injury to sensitive broadleaf crops when off-target exposure occurs as a result of tank contamination, herbicide drift, and movement due to volatilization (Egan *et al.*, 2014; Grover *et al.*, 1972; Johnson *et al.*, 2012b). There is great concern with the introduction of 2,4-D-resistant crops, as the use of 2,4-D in amplified quantities throughout the growing season would likely increase the occurrence of accidental crop injury to sensitive broadleaf species grown in the same vicinity, including peanut (Egan *et al.*, 2014; Grover *et al.*, 1972; Johnson *et al.*, 2012a; Leon *et al.*, 2014). It is important to note, herbicide drift rates up to 16% of the spray solution have been reported to move off-target (Wolf *et al.*, 1993). The 2,4-D and 2,4-D choline registrations for use in common and Enlist-Duo[®] crops of the southeast can be approximately 1066 g ae ha⁻¹, and 16% of that rate is 171 g ha⁻¹. If 171 g ha⁻¹ of 2,4-D can move off target in a controlled experiment, higher rates of 2,4-D could drift off-target if applicators did not follow all herbicide label

directions. Exposure rates of 2,4-D from tank contamination could range considerably.

Prostko *et al.* (2003) reported that 2,4-D applied to peanut preplant or preemergence (PRE) at 250 to 1000 g ha⁻¹ caused peanut injury of 8% or less for all applications and tillage systems with no yield loss. Leon *et al.* (2014) reported that peanut treated at 21 and 42 DAP had injury of 0 to 35% when 2,4-D was applied at 70 to 1120 g ha⁻¹, respectively, and 1120 g ha⁻¹ caused 41% maximum yield loss. Merchant *et al.* (2012) applied 2,4-D to peanut at 30, 60, and 90 DAP at rates of 105 to 1680 g ha⁻¹ and reported yield loss of 7 to 24%. The earlier applications (30 and 60 DAP) were more injurious than the 90 DAP treatments.

These studies are of great benefit to growers that face the possibility of peanut crop injury from accidental 2,4-D exposure, because in such an unfortunate situation the grower, extension agent, and crop consultant would be able to make an informed decision about the appropriate plan of action (Leon *et al.*, 2014; Prostko *et al.*, 2011). In an effort to link previous data, it is necessary to quantify the injury and yield response of peanut to 2,4-D across various rates applied at preemergence up to beginning bloom (R1) (Boote, 1982). Therefore, research was conducted to determine the sensitivity of peanut to 2,4-D at four early-season application timings during the vegetative growth stages, and to establish correlations between visual estimates of injury, canopy diameter, and peanut yield.

Materials and Methods

Field trials were conducted during 2012 and 2013 at three locations that represent the peanut growing regions in Georgia. The first location was the University of Georgia (UGA) Southwest Georgia Research and Education Center in Plains, GA, which had a Greenville sandy loam (fine, kaolinitic, thermic Rhodic Kandiudult) soil with 3.8% organic matter (OM), 60% sand, 10% silt, and 30% clay; the second location was the UGA Coastal Plains Experiment Station (Tifton campus) Ponder Farm in Ty Ty, GA, which had a Tifton loamy sand (fine-loamy, kaolinitic, thermic Plinthic Kandiudult) soil with 1% OM, 90% sand, 6% silt, and 4% clay; and the third location was the UGA Atapulgus Research and Education Center in Atapulgus, GA, which had an Orangeburg loamy sand (fine-loamy, kaolinitic, thermic Typic Kandiudult) soil with 1.5% OM, 86% sand, 6% silt, and 8% clay. Soil pH was 5.56, 5.63, and 6.0, respectively. The soil was prepared using a till-

Table 1. Rainfall and irrigation received monthly with planting, digging, and harvest date for each experiment^a

Site year	Rainfall and Irrigation per month (cm)												Total
	May		June		July		August		September		October		
	R ^b	I	R	I	R	I	R	I	R	I	R	I	
	cm												
Plains 2012	2.4	3.2	6.8	1.0	10.2	2.7	4.7	1.0	10.1	0	1.5	0	43.6
Plains 2013	1.1	1.0	13.3	0	19.7	0	15.0	0	5.5	2.0	0.9	0	58.5
Ty Ty 2012	5.8	1.0	9.9	1.0	15.4	2.0	26.7	0	8.3	0	6.1	0	76.2
Ty Ty 2013	4.4	1.6	29.7	0.4	15.2	0	18.3	0	5.9	1	0.6	0	77.1
Attapulugus 2013	0.1	3.0	11.0	2.5	29.5	0.5	15.8	1.5	8.3	2.0	0.4	0	74.6
	Planting date						Digging Date			Harvest Date			
Plains 2012	May 7						Sept. 27			October 7			
Plains 2013	May 20						October 18			October 24			
Ty Ty 2012	May 9						Sept. 27			October 8			
Ty Ty 2013	May 13						Sept. 25						
Attapulugus 2013	May 10						October 3			October 8			

^aRainfall and irrigation are reported in centimeters from planting date to harvest date.

^bAbbreviations: rainfall, R; irrigation, I

ovator to loosen the soil and make it more suitable for planting. Peanut cultivar ‘Georgia-06G’ (Branch, 2007) was planted when soil conditions were warm enough for proper germination (around the 2nd week of May in Georgia). Specific planting dates are shown in Table 1. Peanuts were planted with 91 cm row spacing, two rows per plot, using vacuum style planters adjusted to 20 seed m⁻¹ row⁻¹. Plots were 1.8 m wide and 7.6 m or 9.1 m long with the same size border on either side of each plot.

Preemergence herbicides were applied to suppress weeds early in the season, and included 1066 g ai ha⁻¹ of pendimethalin, 71 g ai ha⁻¹ of flumioxazin, and 27 g ai ha⁻¹ of diclosulam. Irrigation was applied to incorporate the herbicide into the soil and to firm up the soil around the peanut seed. Applications of POST herbicides were used as needed throughout the season which included acifluorfen, clethodim, and imazapic (Anonymous 2013). Hand-weeding was used as necessary. Supplemental irrigation was applied throughout the growing season as needed, which is shown in Table 1 with monthly rainfall received. Fertilizer, fungicides, and insecticides were applied by on-site farm management for the duration of the trials based on University of Georgia recommendations (Anonymous, 2013).

Trials were conducted using a randomized complete block design with 4 treatment timings and 6 herbicide rates, which included a non-treated control. 2,4-D (Weedar[®] 64, dimethylamine salt, 456 g ae L⁻¹, Nufarm Inc., 150 Harvester Drive, Burr Ridge, IL) at rates of 0, 67, 133, 266, 533, and 1066 g ae ha⁻¹ were applied preemergence (PRE),

10, 20, and 30 DAP. Treatments were replicated 4 times at Plains and Ty Ty and 3 times at Attapulugus. All treatments were applied using a CO₂ pressurized backpack sprayer calibrated to deliver 140 L ha⁻¹ at 152 kPa using TeeJet[®] XR8002VS nozzles (TeeJet[®] Technologies, Spraying Systems Company, Wheaton facility, P.O. Box 7900, Wheaton, IL).

PRE treatments were applied within three d of planting. The 10, 20, and 30 DAP treatments coincided with V2, V3, and V5 vegetative growth stages respectively (Boote, 1982). The treatments will be discussed as PRE, V2, V3, and V5 treatments. When the V5 treatments were applied, approximately 25% of the peanut plants had blooms, making them very close to what is considered beginning bloom for a population of peanut plants or the first reproductive stage (R1) (Boote, 1982).

Peanut plant populations were recorded 20 d after planting. Visual estimates of peanut injury were determined based on a combination of plant chlorosis, necrosis, stem epinasty, leaf cupping, plant stunting, and lack of emergence with a scale of 0 (no injury relative to NTC to 100% (plant death). Crop injury was evaluated throughout the growing season every 10 d until 80 DAP. Canopy diameter measurements were documented (3 per plot) every 10 d until 80 DAP.

At 130 DAP, non-2,4-D-treated sections of peanut were checked for maturity using the hull-scrape method (Williams and Drexler, 1981). Based on maturity tests, peanuts were dug and inverted. Digging dates and harvest dates are presented in Table 1. Peanuts were partially dried in bright

sunlight for 5 to 10 d, and then each peanut plot crop was harvested with a two-row peanut harvester, bagged, and weighed. Moisture content was recorded and adjusted to 10% for final yield determination. Yield data were not collected for the Ty Ty 2013 site year due to animal damage late in August of that season. Peanut pod samples (500 g) from each plot were stored in small paper or mesh bags. Within three wk of harvest, each plot sample was shelled with a mechanical peanut sheller, and the weight of 100 randomly selected whole seed was measured from each sample.

Data were subjected to ANOVA using PROC GLMMIX (SAS Institute, Inc. 2012) to determine interactions between main factors ($\alpha = 0.05$). Location, treatment timing, and herbicide rate were considered fixed effects, whereas random effects included years, repetitions and the associated interactions. Visual estimates of peanut injury, crop population density, peanut canopy diameter, peanut yield, and kernel weight were analyzed. Linear and nonlinear regression models were evaluated to determine associations between herbicide rate and all dependent variables. Pearson correlation analysis was conducted to define relationships between peanut injury, canopy diameter, and yield.

Results and Discussion

Location effects indicated no interactions with peanut response by herbicide rate or treatment timing ($P = 0.30$). Treatment timing by herbicide rate interactions were highly significant ($P < 0.0001$) for peanut injury, canopy diameter as a percentage to the NTC, and yield as a percentage NTC. Peanut plant population at 20 d after treatment (DAT) was not affected by herbicide rate ($P = 0.87$), and peanut kernel weights indicated no mass reduction ($P = 0.78$) (data not shown).

Preemergence treatments. All visual estimates of injury and canopy diameter measurements reported were observed 20 DAT as compared to the NTC. This timeframe from application coincides with the amount of time a grower may need when determining the outcome of an injured peanut crop. Additionally, if a peanut crop was exposed to an auxin herbicide, waiting 20 d after the initial injury would give indications of which herbicide caused the injury. 2,4-D injury on peanut has been reported to be higher at 1 week after treatment (WAT) when compared to 3 WAT, and dicamba injury on peanut indicated to increase up to 3 WAT (Leon *et al.*, 2014).

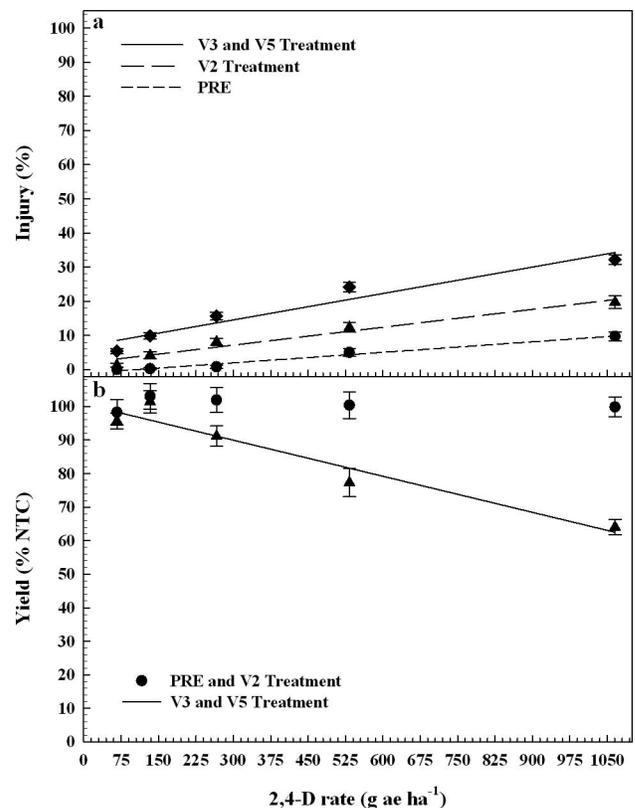


Fig. 1. Peanut injury 20 DAT (a) and yield (b) as a percentage compared to the nontreated control by 2,4-D rate. Plains, Ty Ty, and Attapulgus data are combined by treatment timing. Treatment timings are combined if there were no differences in response to herbicide rate. Data points represent the observed means with standard error.

$$\text{Injury, PRE treatments } y = 0.01x - 1.1, R^2 = 0.53$$

$$\text{Injury, V2 treatments } y = 0.02x + 1.9, R^2 = 0.58$$

$$\text{Injury, V3 and V5 treatments } y = 0.03x + 6.8, R^2 = 0.61$$

$$\text{Yield, V3 and V5 treatments } y = -0.04x + 101, R^2 = 0.39$$

Linear regression indicated minimal peanut injury for PRE 2,4-D treatments across all rates (Figure 1a). When compared to the NTC, peanut injury estimates were 10% at the highest rate of 2,4-D (1066 g ae ha⁻¹), but injury was transient and not observed later in the season (data not shown). As previously mentioned, peanut plant stand at 20 DAT were not different than the NTC, but a short delay in emergence was noted at 10 DAT from 2,4-D at 533 and 1066 g ha⁻¹ (data not shown). Peanut canopy diameter was stunted 8 to 10% with all rates of 2,4-D (67 to 1066 g ha⁻¹) relative to the NTC, but stunting was transient.

When 2,4-D is applied PRE it quickly dissipates in the soil and has negligible residual activity. Data indicates that 2,4-D degrades more rapidly in well drained soils; where sandy loam soils had 2,4-D half-lives of 3 to 7 d (Smith, 1980). It has a low binding affinity in mineral soils and sediment, with half-lives averaging 6.2 d according to the EPA (2005). Therefore, the lower concentrations of 2,4-

Table 2. Pearson Product Moment Correlation of peanut injury estimates, canopy diameter, and peanut yield for 2,4-D treatments made at V3 to V5 peanut growth stages in Georgia^a

		Canopy diameter	Yield
		———%NTC ^b ———	
Peanut injury	Correlation coefficient ^c	-0.76	-0.48
	P value	1.3E-037	8.8E-010
Canopy diameter	Correlation coefficient	———	0.51
	P value	———	8.7E-011

^cCorrelations are observed by the point of intersection between variables.

^bAbbreviations: nontreated control, NTC

^cPositive correlation coefficients and P values below 0.050 tend to increase together, negative correlation coefficients and P values below 0.050, one variable tends to decrease while the other increases.

D, applied PRE, degraded before injury occurred to peanut, but higher concentrations resulted in noticeable leaf cupping and stem epinasty. Within a few d after initial peanut injury, there was a quick recovery, due to peanut tolerance to low doses of 2,4-D. Since peanut grew out of the initial injury from PRE treatments, there was no evidence of yield loss when compared to the NTC for any 2,4-D rate (Figure 1b). In a similar study, 2,4-D was applied preplant or PRE at 250 to 1000 g ha⁻¹ causing peanut injury of 8% or less for all applications and tillage systems, with no yield losses (Prostko *et al.*, 2003).

V2 growth stage treatments. Peanut injury estimates 20 DAT were higher for V2 growth stage (Boote, 1982) treatments when compared to PRE treatments (Figure 1a). Peanut plants received a direct application of 2,4-D for V2 treatments but was soil applied for PRE treatments. Peanut injury for V2 treatments was 1 to 20% from 2,4-D rates of 67 to 1066 g ha⁻¹. When studying Virginia Bunch peanuts, Rawson (1963) reported that visible crop injury to be lowest on peanuts treated at 1 week after emergence (WAE) with rates up to 680 g ha⁻¹ of 2,4-D as compared to treatments of 1 to 16 WAE. In the present study, V2 treatments were less injurious to peanut when compared to the later treatments of 2,4-D at V3 to V5 growth stages (Figure 1). The majority of injury at 20 DAT was attributed to overall peanut stunting relative to the NTC and old necrotic tissue, but at that time peanut plants had new healthy growth after the initial 2,4-D injury (leaf cupping, stem epinasty, etc). Peanut canopy diameter was stunted 4 to 24% at 2,4-D rates of 67 to 1066 g ha⁻¹ when applied at

the V2 growth stage (Data not shown). After correlation analysis, peanut canopy diameter as a % NTC and injury estimates as compared to the NTC, were highly significant ($P < 0.0001$) with a correlation coefficient of -0.56 (Table 2), which is evidence that peanut canopy diameter can be a useful parameter when determining injury to peanut from 2,4-D.

Visual estimates of injury and canopy diameter stunting were not indicators of peanut yield loss when 2,4-D was applied at the V2 growth stage. With a maximum of 20% injury and 24% peanut canopy diameter stunting from 1066 g ha⁻¹ of 2,4-D, the peanut recovered to have no yield loss at harvest as compared to the NTC. Thus, peanut yield data were combined for the PRE treatments and for the V2 treatments since both treatment timings resulted in no yield loss (Figure 1). This result is another indicator that peanut has the ability to tolerate high levels of 2,4-D up to V2 growth stage, while having enough time to recover before harvest.

V3 to V5 growth stage treatments. Since there were no differences in response of peanut to 2,4-D treatments made at V3 and V5 growth stages ($P = 0.86$), data were combined and will be referred to as V3 and V5 treatments. Linear regression indicated peanut injury was 5 to 32% from 2,4-D rates of 67 to 1066 g ha⁻¹ after V3 and V5 treatments (Figure 1), which increased by 2,4-D rate. Johnson *et al.* (2012a) reported that 2,4-D treatments made to peanut approximately 3 WAE, planted 15 to 20 cm wide, caused 30 to 40% injury at 1 and 2 WAT, respectively. Leon *et al.* (2014) indicated that peanut injury, averaged over three rating dates, from 2,4-D at 70, 140, and 280 g ha⁻¹ applied 21 and 42 DAP was not different when compared to the NTC, but rates of 560 and 1120 g ha⁻¹ caused 15 to 35% injury, which is similar to previously described results. The present study indicated peanut canopy diameter was stunted 5 to 35% from 67 to 1066 g ha⁻¹ of 2,4-D relative to the NTC (Figure 2). After correlation analysis, peanut canopy diameter 20 DAT as a % NTC and visual estimates of injury 20 DAT relative to the NTC indicated to be highly significant ($P < 0.0001$) with a correlation coefficient of -0.76 (Table 2).

When peanut was treated at the V5 growth stage, peanut was in 25% bloom (data not shown). According to Boote (1982), the first reproductive growth stage is beginning bloom (R1), which begins when 50% of the peanut population has blooms, or had a bloom. Thus, the peanut trials had not entered the R1 growth stage at the time of treatments, but did within 10 d. Peanut had greater injury and increased canopy diameter stunting after V3 and V5 treatments of 2,4-D, and since

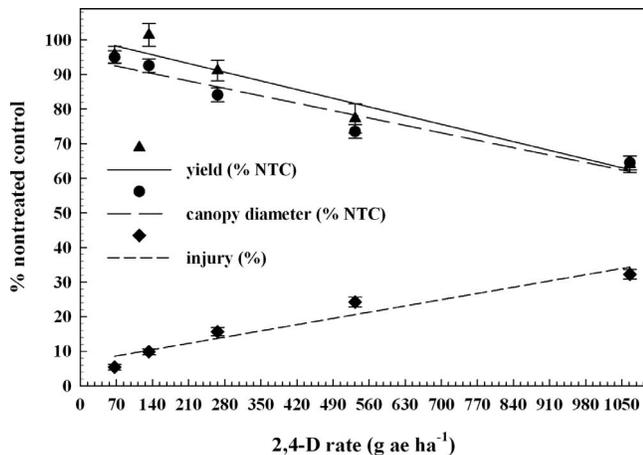


Fig. 2. Peanut yield, canopy diameter 20 DAT, and injury 20 DAT as a percentage compared to the nontreated control by 2,4-D rate. Plains, Ty Ty, and Attapulgis data are combined by V3 and V5 treatment timing. Data points represent the observed means with standard error.

$$\text{Yield } y = -0.04x + 101, R^2 = 0.39$$

$$\text{Canopy diameter } y = -0.03x + 95, R^2 = 0.45$$

$$\text{Injury } y = 0.03x + 6.8, R^2 = 0.61$$

reproductive tissues were forming, there was increased yield loss when compared to V2 treatments. Peanut pod yield loss was 9, 23, and 36% at 266, 533, and 1066 g ha⁻¹ of 2,4-D respectively (Figure 1) after V3 and V5 treatments. Johnson *et al.* (2012a) reported that peanut had considerable tolerance to sublethal rates of 2,4-D, but at 269 g ha⁻¹ yield loss occurred at one location in 2009. Leon *et al.* (2014) reported peanut yield loss of 11 to 41% at 70 to 1120 g ha⁻¹ of 2,4-D when applied 21 and 42 DAP. V3 and V5 treatments resulted in significant correlations ($P < 0.0001$) of peanut yield as % NTC with injury estimates 20 DAT and canopy diameter 20 DAT as % NTC; having correlation coefficients of -0.48 and 0.51 respectively (Table 2).

This research and similar studies were conducted to provide growers and extension agents with usable information. In the unfortunate situation of off-target exposure or accidental treatments of 2,4-D due to tank contamination, growers need to know how the injury will affect peanut yield. As shown in Figure 2, the previously described correlations indicate that the peanut injury estimates and canopy diameter measurements obtained 20 d after 2,4-D exposure can be very helpful when determining yield loss to peanut when injured at V3 to V5 growth stages.

Summary and Conclusions

Peanut injury, canopy diameter, and peanut yield loss varied due to 2,4-D treatments made at

different peanut growth stages. PRE and V2 growth stage treatments injured peanut, but peanut recovered to have no yield loss. There were significant increases in peanut injury as 2,4-D rate increased, especially after V3 and V5 treatments. Generally, 2,4-D was more injurious to peanut when applied closer to beginning bloom (approximately 30 DAP); the first reproductive growth stage (R1). Linear regression models were accurate representations of injury estimates and peanut yield as % NTC by herbicide rate. Visual estimates of injury and canopy diameter measurements of peanut in the present study provided practical and easily obtained information that a grower could do with little to no cost. Visual estimates of peanut injury at 20 DAT were correlated with peanut yield when 2,4-D was applied at V3 to V5 growth stages. Highly significant correlations ($P < 0.0001$) were observed for peanut injury 20 DAT, canopy diameter 20 DAT, and peanut yield % NTC.

2,4-D resistant crops will likely have widespread use in the near future, so great care should be taken to prevent 2,4-D drift, sprayer contamination, and volatilization. Applicators should always read the herbicide label and follow all directions and restrictions. Due to the sensitivity of peanut to 2,4-D, growers and applicators should avoid applying 2,4-D in the proximity of peanut during V3 to V5 growth stages and early reproductive growth stages. In the unfortunate situation where peanut injury from accidental 2,4-D exposure occurs, these data could assist the grower in determining peanut yield loss estimates and a possible plan of action.

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