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Establishing the Geographical Distribution and Level of Acetolactate Synthase Resistance of Palmer Amaranth (*Amaranthus palmeri*) Accessions in Georgia

Aaron M. Wise, Timothy L. Grey, Eric P. Prostko, William K. Vencill, and Theodore M. Webster*

Palmer amaranth resistance to acetolactate synthase (ALS)–inhibiting herbicides was first identified in Georgia in 2000. Since then, complaints from peanut producers have increased concerning failure of ALS herbicides in controlling Palmer amaranth. Because efficacy of ALS herbicides can be compromised under adverse conditions, seeds from Palmer amaranth plants that escaped weed control were collected across the peanut-growing region in Georgia to investigate the cause of these reported failures. Greenhouse and growth-chamber studies were conducted using these seeds to evaluate whether weed escapes were a result of Palmer amaranth resistance to ALS herbicides. Each of the 61 accessions collected across Georgia exhibited varying levels of resistance to imazapic applied POST (< 55% control, relative to ALS-susceptible Palmer amaranth). Subsamples of the accessions were evaluated for their response to imazapic rates, which indicated variable levels of resistance across Palmer amaranth accessions. The rate of imazapic that provided 50% reduction in Palmer amaranth plant biomass (I_{50}) for the known susceptible biotype was 0.9 g/ha of imazapic. Of the 10 accessions evaluated, 8 of them had I_{50} values that ranged from 3 to 297 g/ha of imazapic. The other two accessions could not be fit to the log-logistic dose–response curve and had undeterminable I_{50} values because of high levels of ALS resistance (> 1,400 g/ha of imazapic). Herbicide cross-resistance experiments indicated that 30 accessions were resistant to the ALS herbicides imazapic, chlorimuron, pyrithiobac, and diclosulam at the recommended field-use rates. However, each of these 30 accessions was susceptible to glyphosate. These data demonstrate that ALS-resistant Palmer amaranth occurs throughout the peanut-growing region of Georgia. Growers in Georgia will need to alter their weed-control programs in peanut to include herbicides with multiple modes of action that do not rely on ALS herbicides for effective Palmer amaranth control.
Nomenclature: Chlorimuron; diclosulam; imazapic; pyrithiobac; Palmer amaranth, *Amaranthus palmeri* L; peanut, *Arachis hypogea* L.
Key words: Herbicide resistance, acetolactate synthase, dose–response, weed resistance.

Many pigweeds (*Amaranthus* spp.) are native to the United States, but only a few have become significant weed problems (Steckel 2007). In Georgia, pigweeds rank as the third most-troublesome weed across all agronomic crops, the second most troublesome weed in cotton (*Gossypium hirsutum* L.) and vegetables, and among the top five most troublesome weeds in corn (*Zea mays* L.), soybean [*Glycine max* (L.) Merr.], and tobacco (*Nicotiana tabacum* L.) (Webster and MacDonald 2001). Before the detection of Palmer amaranth resistant to acetolactate synthase (ALS)–inhibiting herbicides in Georgia in 2001 (Heap 2009; Vencill et al. 2002), pigweeds were ranked as the ninth most troublesome species in peanut (Webster and MacDonald 2001). A more recent survey indicated that Palmer amaranth was the most troublesome weed in peanut (Webster 2005), likely because of the occurrence of herbicide resistance.

Palmer amaranth has invaded the lower Midwest (Kansas, Nebraska, Missouri) and the southeast United States (Alabama, Georgia, Florida, North Carolina, South Carolina, and Tennessee) from its native habitat in the southwest United States (southern California and west Texas) (Steckel 2007) and has become the dominant pigweed species in Georgia. Palmer amaranth is an annual herbaceous weed, capable of rapid growth (0.18 to 0.21 cm/growing degree day), high rates of photosynthesis (80 μmol/m^2/s), and high fecundity (up to 400,000 seeds/plant produced under crop competition) (Bensch et al. 2003; Ehleringer 1983; Horak and Loughin 2000; MacRae et al. 2008). Season-long Palmer amaranth interference has resulted in corn, soybean, and cotton yield reductions up to 90, 68, and 86%, respectively (Klingaman and Oliver 1994; Massinga et al. 2001; Morgan et al. 2001; Rowland et al. 1999). Because of their biomass and woody stems at the conclusion of the growing season, pigweeds can also impede harvest; Palmer amaranth interference caused a twofold to threefold increase in time required for harvest relative to that of weed-free cotton (Smith et al. 2000).

Palmer amaranth has developed resistance to four herbicide modes of action. Palmer amaranth resistance to pendimethalin was noted in South Carolina in 1989 (Gossett et al. 1992). Pendimethalin is widely used for PRE weed control in Georgia cotton, peanut, and soybean. Resistance of Palmer amaranth to ALS-inhibitor herbicides was discovered in Kansas in 1991, Arkansas in 1994, North Carolina in 1995, South Carolina in 1997, and Georgia in 2000 (Burgos et al. 2001; Heap 2009; Horak and Peterson 1995; Vencill et al. 2002). ALS-inhibiting herbicides are commonly applied in all agronomic crops in Georgia. Resistance of Palmer amaranth to triazine herbicides was initially reported in Texas in 1993 and Kansas in 1995 (Heap 2009; Peterson 1999). The fourth mode of action to which Palmer amaranth has developed resistance is glyphosate. Glyphosate provides broad-spectrum weed control but is also used in glyphosate-resistant crops.

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Palmer amaranth resistance to glyphosate was first reported in Georgia in 2005 and has been confirmed in North Carolina, Arkansas, and Tennessee (Culpepper et al. 2006; Norsworthy et al. 2008; Steckel et al. 2008).

The ALS herbicides prevent the synthesis of valine, leucine, and isoleucine by affecting the first common enzyme in the branched-chain amino acid biosynthetic pathway (Saari et al. 1994; Shaner et al. 1984; Zhao et al. 1999). The triazines or auxins have been used since the early 1950s and have 67 and 26 resistant weed species, respectively (Heap 2009). In contrast, ALS herbicides have been in commercial production since the early 1980s and have 95 resistant weed species (Heap 2009). Weed resistance to ALS herbicides occurs because repetitive use of this mode of action in multiple crops increases selection pressure for one of the many known mutations that hinder herbicide binding for this class of herbicides (McNaughton et al. 2005; Tranel and Wright 2002).

In 2005, an increase in the number of weed control failures in peanut associated with ALS herbicides was reported for Palmer amaranth in Georgia (personal observations). Herbicide resistance was suspected, and studies were initiated to corroborate this hypothesis. The objectives of this study were to (1) determine whether field-sampled Palmer amaranth in Georgia was resistant to imazapic, (2) determine the geographical distribution of ALS-resistance, and (3) determine whether cross-resistance or multiple resistance to other herbicides existed.

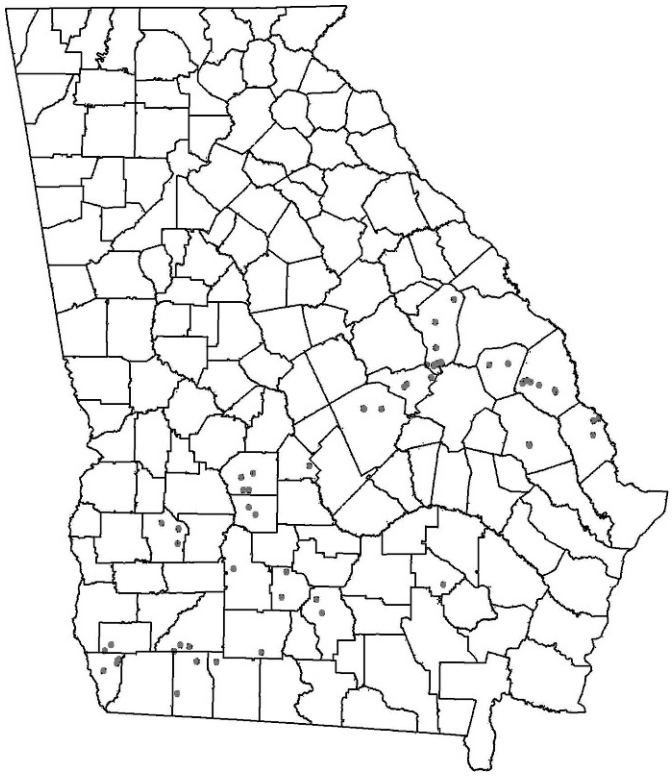

Figure 1. Sample sites for Georgia Palmer amaranth studies.

Materials and Methods

Collection and Cleaning. Palmer amaranth seeds were collected from across the peanut-growing region of Georgia in autumn of 2005 (Figure 1). Sites with potential ALS-resistant Palmer amaranth were selected, based on information provided by local county extension agents. Agents identified fields within their assigned county where suspected herbicide failures occurred in fields that had one or more ALS-herbicide applications in 2003, 2004, or 2005. Sixty-one locations were identified across 21 counties (Table 1). For each location, global positioning system (GPS) coordinates were recorded.[1] Locations were labeled with a county lettering and numbering system value (Bond et al. 2006).

Thyrses were collected from at least 30 female plants per site to obtain a representative sample of each field population. Representative samples were obtained by randomly selecting plants while walking across suspected fields. Immature seed were separated during the cleaning process. Seed heads were hand-harvested and stored in paper bags at 4 C at 20% relative humidity for 6 to 7 wk to allow adequate drying time before cleaning. Thyrses were hand-threshed with a sifter,[2] and the seed was separated from the chaff using a sieve series[3] and forced air. Cleaned seeds were then placed into a scintillation vial,[3] labeled with the appropriate GPS coordinates, and returned to the controlled environment storage, where they remained until testing. A Palmer amaranth accession susceptible to ALS herbicides from the University of Georgia, Ponder Research Farm (Ty Ty, GA), was used as a susceptible standard for the study.

Imazapic Screening. Palmer amaranth seed were sown in 54- by 28- by 7.5-cm flats,[4] containing a commercial potting mixture,[5] which were then placed inside of a growth chamber.[6] Diurnal settings in the growth chambers were 16/8 h light/dark at 30/20 C day/night temperatures, respectively. A combination of fluorescent and incandescent lights provided 400 μmol/m^2/s photosynthetically active radiation. While in the growth chamber, the flats were watered twice daily, and upon emergence, a solution of fertilizer[7] was applied weekly. Flats remained in the growth chamber until plants reached the cotyledon growth stage. Flats were then transferred to a greenhouse with diurnal settings of 32/25 C day/night temperature (± 5 C), with supplemental light provided by metal halide growth lights for 16 h/d. Plants were watered twice daily and fertilized weekly. After 7 d, seedlings were thinned to 15 plants/flat. For each location, five flats were planted for replication.

Herbicide treatments were applied when Palmer amaranth was 5 to 10 cm tall with four to eight leaves. Treatments were imazapic at 70 and 700 g ai/ha, combined with a nonionic surfactant[8] (0.25% v/v), and included a nontreated control for each accession. Treatments were applied using a spray chamber, calibrated to deliver 140 L/ha at 207 kPa through 8004 flat-fan spray tips.[9] After treatment, flats were returned to the greenhouse. Herbicide efficacy was rated 14 to 21 d after treatment (DAT) on a scale of 0 (no plant injury) to 100% (plant death). Treatments were arranged in a randomized complete-block design, and the study was repeated.

Table 1. Origin of Georgia Palmer amaranth accessions listed by county, coordinates from which they were collected in 2005, and response to imazapic.[a]

Accession	County	Latitude	Longitude	Control[b]
		°N	°W	%
CR1	Crisp	31.91	83.77	26 *
CR2	Crisp	31.96	83.82	22 *
DO1	Dooly	32.06	83.86	18 *
DO2	Dooly	32.06	83.82	11 *
DO3	Dooly	32.16	83.79	3 *
DO4	Dooly	32.14	83.88	17 *
P01	Pulaski	32.21	83.37	12 *
T01	Terrell	31.83	84.34	4 *
T02	Terrell	31.86	84.46	28 *
T03	Terrell	31.73	84.34	19 *
W01	Worth	31.58	83.94	17 *
CT1	Colquitt	31.07	83.73	14 *
TM1	Thomas	31.02	84.06	7 *
M01	Mitchell	31.07	84.37	19 *
M02	Mitchell	31.12	84.32	38 *
M03	Mitchell	31.12	84.25	15 *
G1	Grady	30.82	84.35	14 *
G2	Grady	31.02	84.20	12 *
S1	Seminole	31.03	84.77	10 *
S2	Seminole	31.01	84.79	18 *
S3	Seminole	31.02	84.79	14 *
S4	Seminole	30.96	84.89	9 *
ML1	Miller	31.09	84.88	28 *
ML2	Miller	31.12	84.83	16 *
TT1	Tift	31.41	83.58	15 *
TT2	Tift	31.56	83.54	7 *
B1	Berrien	31.40	83.33	8 *
B2	Berrien	31.32	83.28	3 *
BN1	Bacon	31.49	82.39	11 *
J1	Jefferson	32.84	82.41	15 *
J2	Jefferson	32.82	82.46	36 *
J3	Jefferson	32.82	82.51	42 *
J4	Jefferson	32.83	82.40	13 *
J5	Jefferson	32.92	82.44	33 *
J6	Jefferson	33.08	82.45	34 *
J7	Jefferson	33.22	82.31	15 *
SC1	Screven	32.67	81.57	11 *
SC2	Screven	32.66	81.56	14 *
SC3	Screven	32.70	81.69	11 *
SC4	Screven	32.72	81.75	7 *
SC5	Screven	32.73	81.77	11 *
SC6	Screven	32.71	81.81	4 *
SC7	Screven	32.72	81.80	6 *
EM1	Effingham	32.50	81.25	21 *
EM2	Effingham	32.48	81.29	4 *
EM3	Effingham	32.40	81.29	15 *
BL1	Bulloch	32.34	81.76	18 *
BL2	Bulloch	32.33	81.75	5 *
BL3	Bulloch	32.34	82.76	12 *
LN1	Laurens	32.56	82.98	11 *
LN2	Laurens	32.56	82.98	19 *
LN3	Laurens	32.55	82.97	55 *
LN4	Laurens	32.55	82.84	11 *
J8	Jefferson	32.83	82.44	12 *
J9	Jefferson	32.82	82.44	19 *
J10	Jefferson	32.82	82.46	14 *
JS1	Johnson	32.74	82.48	9 *
JS2	Johnson	32.70	82.66	5 *
JS3	Johnson	32.69	81.68	26 *
JK1	Jenkins	33.66	82.91	52 *
JK2	Jenkins	32.82	82.05	16 *
Susceptible	Worth	31.47	83.65	100

[a] Expressed as a percentage of the deceased biomass compared with live plants ($n = 30$ plants or greater per location). No significant interaction for rate within an accession allowed the combination of data across imazapic rates of 70 and 700 g ai/ha.

[b] Means followed by an asterisk (*) are significantly different at $P \leq 0.05$ from the susceptible control using Dunnett's procedure.

Dose–Response. Ten sites exhibiting some level of ALS resistance were selected based on a cross-section of the peanut-growing region of Georgia. Imazapic treatments included 0.71, 7.1, 71, 710, and 1,420 g/ha, with a nonionic surfactant (0.25% v/v) and a nontreated control. Seeds from the ALS-susceptible population and from an advanced generation obtained from plants surviving the original screening were also evaluated for ALS-resistance. Plants were grown in a topsoil mix[10] in 11.5- by 15-cm pots, watered daily, and fertilized weekly, as previously described. Seedlings were thinned to five plants/pot, with four replications per treatment. The treatment was applied using a CO_2 backpack sprayer, calibrated to deliver 140 L/ha at 193 kPa, equipped with 8002 flat-fan nozzles. Aboveground biomass was harvested 14 DAT, dried in 9- by 15-cm paper-coin envelopes[11] at 50 C for 72 h, and weighed. Experimental units were arranged in a randomized complete-block design and repeated.

Cross-Resistance Screen. Seeds from 30 accessions and the known ALS-susceptible population were grown as previously described for the dose–response study. At the two-leaf growth stage, plants were thinned to five plants/pot. Treatments consisted of the ALS herbicides, imazapic at 70 g ai/ha, diclosulam at 26 g ai/ha, chlorimuron-ethyl at 13 g ai/ha, and pyrithiobac at 89 g ai/ha. These ALS herbicides were selected based on their frequent use in Georgia cotton (pyrithiobac), peanut (chlorimuron, diclosulam, and imazapic), and soybean (chlorimuron). Other treatments included glyphosate at 840 g ae/ha and a nontreated control. Treatments were applied when plants were 5 to 10 cm in height, as previously described in the dose–response study. The number of surviving plants/pot was recorded and then harvested by clipping at the soil surface. Plant material was dried and weighed. Dry weight data were expressed as a percentage of the nontreated control. The study was a randomized complete-block design with three replications and was repeated.

Statistical Analysis. All data were subjected to ANOVA. In the imazapic screening study, treatment means of herbicide response at each location were compared with a nontreated control using the Dunnett's test ($P < 0.05$). Because the experiments were repeated, all data were combined for analysis to test for experiment and treatment interactions. For cross-resistance experiments, the dry weight reduction (percentage) data were analyzed using ANOVA in SAS (SAS 1999).

Data from dose–response studies were subjected to nonlinear regression, in addition to ANOVA. Palmer amaranth dry weight data, expressed as a percentage of the nontreated control, were regressed against the $\log_{10}$ of the imazapic rate (SAS 1999). The log-logistic regression equation was used to describe the dose–response:

$$y = C + \left[\frac{D - C}{1 + \left(\frac{X}{I_{50}}\right)^{b}}\right] \quad [1]$$

where C is the lower limit, D is the upper limit, b is the slope, and I_{50} is the dose–response giving 50% response (Seefeldt et al. 1995).

Table 2. Herbicide rate that reduces biomass by 50% (GR_{50}) values and resistance factors of 12 Georgia Palmer amaranth accessions for imazapic.

Accession	Imazapic I_{50}[a]	Resistance factor
	g ai/ha	
Susceptible	0.9	1
SC2	32	36
J2	6.5	7
BL3	14.2	16
LN1	> 1,400[b]	> 1,555
CR2	6.9	8
DO3	7.1	8
TO1	179	199
MO1	2.6	3
G2	> 1,400[b]	> 1,555
BN1	6.8	8
F2	297	330

[a] Based on regression using log-logistic dose-response curve.

[b] Analysis of data indicated a non-fit for log-logistic dose response model.

[c] F2 generation seed collected from initial screening of greenhouse grown accessions.

Results and Discussion

Imazapic Screening. There was no imazapic rate interaction; therefore, data were combined across imazapic rate and were presented by location (Table 1). Resistance to imazapic was confirmed by comparing phytotoxicity of the suspected ALS-resistant accessions with a known ALS-susceptible population of Palmer amaranth.

Imazapic injury symptoms on susceptible Palmer amaranth included stunting, epinasty, and chlorosis during the first week, with plant death by 21 DAT. Response of the suspected ALS-resistant accessions ranged from no injury to slight injury to plant death. The control ratings ranged from 3 to 55% across accessions, with some accessions having different proportions of dead plants. The surviving, resistant plants produced new growth on the auxiliary buds at 21 DAT. These symptoms were similar to those observed in other ALS-resistant weed species (Blair and Martin 1988; Falk et al. 2005; Fletcher et al. 1993). Plants from all 61 locations showed some level of tolerance to imazapic. Patzoldt et al. (2002) reported similar results, indicating that the response of *Amaranthus* species to herbicides varies. Common waterhemp (*Amaranthus rudis* Sauer) and Palmer amaranth are dioecious species that allow for a high level of variability within any given accession (Franssen et al. 2001).

Dose–Response. The imazapic I_{50} value for the known ALS-susceptible Palmer amaranth population was 0.9 g/ha (Table 2). High levels of resistance to imazapic were confirmed in the suspected accessions. For two (G2 and LN1) of the 10 locations evaluated, imazapic at 1,400 g/ha failed to reduce biomass by 50% (Table 2). These two locations did not exhibit any significant growth reduction from imazapic applied at the maximum rate in this study (20 times the

Table 3. Cross-resistant screening for susceptible and acetolactate synthase (ALS)–resistant Georgia Palmer amaranth.[a]

Herbicide	Biomass[b]	
	Resistant	Susceptible
	%	
Nontreated	100 a	100 a
Imazapic	115 a	20 bc
Chlorimuron	113 a	64 b
Diclosulam	118 a	21 bc
Pyrithiobac	93 a	17 c
Glyphosate	64 b	43 bc

[a] No significant interaction for accession within a herbicide allowed the combination of data across accessions for presentation by herbicide.

[b] Values followed by the same letter do not significantly differ using Fischer's Protected LSD at $P \leq 0.10$.

registered field-use rate of 70 g/ha), which implies a resistance factor in excess of 1,500 times. Eight locations had variable imazapic dose–responses, although all accessions demonstrated some level of resistance to imazapic. Relative to the ALS-susceptible Palmer amaranth populations, 5 sites had resistance factors between 3 and 8 times greater, 2 sites had resistance 16 and 36 times greater, and 1 site had resistance 199 times greater. The greenhouse advanced population had a level of resistance of 330 times, indicating ALS-resistance was maintained from F1 ascensions to an F2 mixed population.

This is the first study, to our knowledge, to report on ALS-resistant Palmer amaranth in peanut. Previous studies have not specifically evaluated the influence of imazapic on ALS-resistant Palmer amaranth because peanut is the only agronomic crop in which imazapic is registered. Palmer amaranth resistance to imazaquin in Arkansas was determined to be 14 to 16 times greater for intermediate-resistant plants, with highly resistant populations having 141 to 196 times greater tolerance (Burgos et al. 2001). Palmer amaranth populations from Kansas were not affected by 560 g ai/ha of imazethapyr, 8 times the field-use rate of 70 g/ha (Gaeddert et al. 1997; Horak and Peterson 1995). Other pigweeds had variable resistance factors to imazethapyr that ranged between 3.8 and 3,438 times greater for Powell amaranth (*Amaranthus powellii* S. Wats), 13 to 168 times greater for redroot pigweed (*Amaranthus retroflexus* L.), and 74 to 2,000 times greater for tall water hemp (Ferguson et al. 2001; Patzoldt and Tranel 2007; Sprague et al. 1997).

Cross-Resistance. A combined ANOVA across treatments of the ALS-resistant accessions did not indicate a significant treatment by accession interaction among the ALS herbicides tested. Therefore, data across accessions were combined for presentation by herbicide.

Biomass of the ALS-susceptible Palmer amaranth accession was 20, 64, 21, and 17% of the nontreated control for imazapic, chlorimuron, diclosulam, and pyrithiobac, respectively (Table 3). For chlorimuron, the susceptible accession was significantly different from the nontreated control at 64%, but this was the highest biomass among the group, indicating that some level of ALS-tolerance could be developing in this population. In contrast, the ALS-resistant Palmer amaranth accessions were cross-resistant to all the tested ALS herbicides, with plant biomass similar to the nontreated control. The incidence of *Amaranthus* species resistance to ALS herbicides has been well documented (Burgos et al. 2001; Diebold et al. 2003; Ferguson et al. 2001; Sprague et al. 1997) and recently reviewed (Vencill et al. 2008). The ALS mechanism of resistance in *Amaranthus* species is considered to be a point mutation to the ALS gene (Tranel and Wright 2002). Previous studies have found variable responses of ALS-resistant pigweeds to different ALS herbicides. An imazaquin-resistant Palmer amaranth accession from Arkansas was cross-resistant to diclosulam, chlorimuron, and pyrithiobac, with resistance factors of 39, 74, and 117 times, respectively (Burgos et al. 2001). Other pigweeds, including smooth pigweed (*Amaranthus hybridus* L.), tall waterhemp, redroot pigweed, and Powell amaranth, are also cross-resistant to imidazolinone and sulfonylurea herbicides (Ferguson et al. 2001; Maertens et al. 2004; Patzoldt et al. 2002; Sprague et al. 1997). However, accessions of Powell amaranth and redroot pigweed in Canada, waterhemp in Illinois, and redroot pigweed in Virginia were not cross-resistant to different ALS herbicides (Ferguson et al. 2001; Manley et al. 1999; Patzoldt and Tranel 2007). In fact, the Virginia imazethapyr-resistant accession had greater sensitivity to pyrithiobac (Manley et al. 1999; Poston et al. 2000). Since initial discovery in 2000, surveys were not conducted to confirm the distribution of ALS-resistant Palmer amaranth in Georgia. However, it was likely that other ALS-resistant biotypes were in the state, just never properly identified. Pollen movement and cross-pollination (Sosnoskie et al. 2007) could be a factor that increased the level of ALS resistance for the 2005 survey of 61 ascensions.

With the confirmation of glyphosate-resistant Palmer amaranth in Georgia (Culpepper et al. 2006), Tennessee (Steckel et al. 2008), and Arkansas (Norsworthy et al. 2008); multiple resistance (i.e., ALS and glyphosate resistance in the same plant) in Palmer amaranth is a concern to agronomic crop production in the southeast United States. In the current study, glyphosate-resistance was not found among the tested accessions (Table 3). A survey of Palmer amaranth populations from 10 states in 2002 and 2003 indicated no glyphosate resistance but did identify resistance to pyrithiobac ($<$ 50% control) in five states (Bond and Oliver 2006). However, the dioecious nature of Palmer amaranth and the confirmed transfer of herbicide resistance through pollen flow has already altered this status (Culpepper et al. 2006; Sosnoskie et al. 2007; Tranel et al. 2002).

In summary, initial ALS screening indicated there was ALS resistance in Palmer amaranth populations throughout the peanut-growing region in Georgia. However, data indicated heterogeneity in the levels of resistance both among and within the screened accessions. The soil seedbanks at many of these locations likely contain populations of both ALS-susceptible and ALS-resistant Palmer amaranth. In addition to imazapic resistance, accessions exhibited cross-resistance to diclosulam, pyrithiobac, and chlorimuron. Glyphosate resistance was not detected for any accession.

Although the level of resistance varied by accession, repeated applications of ALS herbicides without alternative modes of action will increase ALS-resistant Palmer amaranth

populations across the state. Application of ALS herbicides in Georgia peanut increased from 63% treated hectares in 1999 to 93% treated hectares in 2003 (University of Georgia extension survey 1997 to 2003; E. Prostko, personal communication). Alternatives to managing ALS-resistant Palmer amaranth are necessary for peanut producers to maintain profitability. Season-long interference of Palmer amaranth at a density of 1 plant/m of crop row reduced peanut yields 28% (Burke et al. 2007), a density that readily occurs in Georgia. The current University of Georgia recommendation for ALS-resistant Palmer amaranth management for peanut includes herbicides from multiple groups (Heap 2009), including dinitroanilines (K), chloroacetamides (K3), bipyridiliums (D), and protoporphyrinogen oxidase inhibitors (E) (Prostko 2008). Proper stewardship of these herbicides and management of resistant weeds species should include rotations of herbicide modes of action, both within a cropping season and across crop rotations in multiple years.

Sources of Materials

[1] Magellan Meridian Gold GPS Receiver, Thales Navigation Consumer Products, 960 Overland Court, San Dimas, CA 91773.

[2] Flour Sifter, Wal-Mart Stores, Inc., 702 S.W. 8th Street, Bentonville, AR 72716.

[3] Fischer Scientific, 2000 Park Lane, Pittsburgh, PA 15275.

[4] Belden Plastics, 2582 Long Lake Road, Roseville, MN 55113-2526.

[5] Brown Earth, Craven Pottery, 100 Pottery Road, Commerce, GA 30529.

[6] Controlled Environments Inc., 222 South 5th Street, Pembina, ND 58271.

[7] Scotts Miracle-Gro Products, Inc., P.O. Box 606, Marysville, OH 43040.

[8] Chem Nut, Inc., P.O. Box 3706, Albany, GA 31706.

[9] Spraying Systems Co., P.O. Box 7900, Wheaton, IL 60189.

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