HERBICIDE STRATEGIES FOR PALMER AMARANTH (*AMARANTHUS PALMERI*) MANAGEMENT IN ILLINOIS

BY

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THESIS

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ABSTRACT

Palmer amaranth has been spreading from the southern United States into the Midwest, and the extent of damage this weed can inflict to Illinois row crops is not yet known. Palmer amaranth is known for rapid biomass accumulation and multiple emergence events, making this weed difficult to control. Field and greenhouse experiments were conducted in 2015 and 2016 to evaluate the biology and management of Palmer amaranth in Illinois. Chapter 1 includes a literature review of herbicides pertinent to these experiments and a section of Palmer amaranth biology. Chapter 2 characterizes the growth accumulation and emergence patterns of Palmer amaranth in Illinois to determine when and how long this weed will compete with field crops. Results indicate that Palmer amaranth can germinate for at least 10 weeks in Illinois. Due to the rapid biomass and height accumulation of Palmer amaranth, post emergence herbicides were evaluated in Chapter 3 to determine the herbicide efficacy at different Palmer amaranth heights. Results showed chlorimuron (13 g ai ha^{-1}), imazethapyr (70 g ai ha^{-1}), and mesotrione (105 g ai ha^{-1}) did not achieve Palmer amaranth control ≥73%, regardless of application timing in all experiments. New technology has enabled extensive usage of synthetic auxin herbicides as an option for control in resistant-soybean varieties; therefore, Chapter 4 discusses dose response experiments of Palmer amaranth in Illinois to 2,4-D and dicamba. Results indicate that dicamba ratings did not exceed 86% of Palmer amaranth control under field conditions at the maximum labeled rate for dicamba-resistant soybean. Single or multiple applications of glufosinate, or glufosinate plus residual herbicides were compared in Chapter 5 to determine the overall control of biomass and emergence of Palmer amaranth in glufosinate-resistant soybean. Results indicate that Palmer amaranth biomass reduction is greatest when multiple applications of glufosinate are used, regardless of the addition of a residual herbicide. Preemergence herbicides were evaluated

in Chapter 6 to determine the length of control and reduction in biomass of Palmer amaranth. Further research is needed for evaluation of length of residual control.

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TABLE OF CONTENTS

CHAPTER 1

LITERATURE REVIEW

1.1 Herbicide Properties

1.1.1 ALS Inhibitors

The enzyme acetolactase synthase (ALS), also known as acetohydroxyacid synthase (AHAS), is the first enzyme of the branched chain amino acid biosynthetic pathway (Hirai et al. 2002; Tan et al. 2005). This pathway involves four enzymes (anabolic AHAS, ketol-acid reductoisomerase, hydroxyacid dehydratase, and a transaminase) in parallel steps to produce valine, leucine, and isoleucine. Pyruvate is a common precursor for these amino acids as well as a second precursor for isoleucine, 2-ketobutyrate (Duggleby and Pang 2000).

There are five classes of ALS inhibiting herbicides based on chemical structure (Shaner 2014). Sulfonylureas and imidazolinone herbicides have partially overlapping binding sites that are within and overlay the channel leading to the active site for this enzyme (McCourt et al. 2005). Common characteristics of ALS-inhibiting herbicides include low use rates and low mammalian toxicity (Hirai et al. 2002). The activity of sulfonylureas as herbicides was discovered in mid-1970s by DuPont, which coincided to the time American Cyanamid was developing imidazolinones (Duggleby and Pang 2000; Shaner et al. 1984). Commercialization of ALS herbicides occurred in 1982 for broadleaf control in small grains (Saari et al. 1994). ALS-inhibiting herbicides are systemically transported in the xylem and phloem and accumulate in the actively growing regions of the plant. Necrosis occurs in sensitive plants after several weeks due to depletion of essential amino acids (Shaner 2014). Crop tolerance to ALS herbicides is based on the crop's ability to rapidly metabolize the herbicide. Certain crops are able to naturally detoxify the ALS herbicides through a variety of hydroxylation, conjugation, hydrolytic, and cleavage reactions (Duggleby and Pang 2000).

Sulfonylureas (SUs) have a basic structure of X-SO2-NH-CO-NH-Y, where X is typically a phenyl group and Y is a substituted pyrimidine or triazine ring (Duggleby and Pang 2000). This group has substantial biological activity, as SUs are typically applied at a rate of 10 to 100 g per hectare (Duggleby and Pang 2000). Mammalian toxicity also is low with high LD_{50} values (e.g., chlorosulfuron LD_{50} is 6 grams per kilogram of biomass in rats) (Duggleby and Pang 2000). These herbicides are degraded in soil by a combination of non-enzymatic hydrolysis and microbial degradation (Brown and Kearney 1991). SU herbicides depend on crop selectivity, which results from a plant species' ability to convert the herbicide to non-toxic forms via hydroxylation, conjugation, hydrolytic, and cleavage reactions (Brown 1990). SU herbicides inhibit root and shoot growth in sensitive weed species. Symptoms of injury are slow to develop and include vein reddening, leaf chlorosis, terminal bud death, and eventual tissue necrosis (Brown 1990). The biochemical site-of-action was discovered when the inhibition of bacterial growth by a SU on a medium containing valine was reversed by the addition of isoleucine, demonstrating that SUs inhibit the enzyme acetolactate synthase (Larossa and Schloss 1984).

Imidazolinone (IMI) herbicide structures consist of a 4-isopropyl-4methyl-5oxo-2-imidzolin-2-yl nucleus linked at the 2-position to an aromatic ring system, which is usually heterocyclic (Duggleby and Pang 2000). Herbicides in this chemical family are typically applied at rates ranging from 100 to 1000 g per hectare, as these herbicides are less potent than sulfonylureas. Residual soil activity is affected by organic matter and pH values lower than 6.0: conditions that favor soil microbial activity will enhance herbicide degradation (Kraemer et al. 2009).

The widespread use of ALS inhibitors has led to the evolution of resistance to a level that surpasses all other herbicide groups (Heap 2017). Currently, there are 92 dicot and 62 monocot

species that are resistant to ALS herbicides (Heap 2017). Most cases of ALS resistance are caused by changes in the base sequence of the ALS gene, resulting in an enzyme that is less sensitive to the binding of ALS-inhibiting herbicides (Burgos et al. 2001; Saari et al. 1994; Sprague et al. 1997; Tranel and Wright 2002). The mutations to the ALS base sequence are partially dominant (Tranel and Wright 2002); therefore, only single gene copies are necessary for resistance. Metabolism is another factor that allows plants to overcome ALS inhibition. Metabolic detoxification is similar to the process that confers crop selectivity to ALS inhibitors (Yu and Powles 2014). Some weed populations have been reported to have both mechanisms of resistance to ALS-inhibiting herbicides (Ma et al. 2013).

1.1.2 HPPD Inhibitors

4-hydroxy-phenylpyruvate-dioxygenase (HPPD**)** is a catalyst for the conversion of *p*-hydroxyphenylpyruvate to homogentisate in the biosynthesis pathway of plastoquinone (Hirai et al. 2002). This pathway produces plastoquinones, vitamin E, and carotenoids.

Herbicides that inhibit the enzyme HPPD comprises of three chemical classes: triketones, isoxazoles, and pyrazolones. When HPPD inhibitors block hydroxylation, no plastoquinone is formed, and without this acceptor of hydrogen, phytoene accumulates and carotenoid biosynthesis is compromised (Hirai et al 2002). HPPD herbicides interfere with the plant's ability to produce energy by inhibiting the production of plastoquinones, which are needed as electron carriers between carotenoid desaturase and the photosynthetic electron transport chain (van Almsick 2009). This indirect inhibition was confirmed by eliminating carotenoids and adding supplemental homogentisate to treated plants (Norris et al. 1995). Since the plant's energy supply from photosynthesis is inhibited, plant growth is inhibited. The ability to produce vitamin E is also disrupted, which is needed to protect against oxidative stress. Without the anti-

oxidant, the buildup of free radicals disturbs the function and structure of the chloroplast and thylakoid membranes. The prevention of carotenoid biosynthesis is caused by the loss of the ultraviolet (UV) shield that protects chlorophyll, which is destroyed by UV rays and excess light (van Almsick 2009). This loss of chlorophyll causes the plant to turn white, often described as bleaching symptoms on new growth (Shaner 2014).

HPPD-inhibiting herbicides have been used for selective weed control since the early 1990s (Hirai et al. 2002; van Almsick 2009). Currently, tall waterhemp *(Amaranthus tuberculatus)* and Palmer amaranth *(Amaranthus palmeri)* are the only species resistant to HPPD-inhibiting herbicides (Heap 2017). HPPD resistance was first reported in Stafford County, Kansas. Palmer amaranth was reported to be 7–11 times more resistant to pyrasulfotole and bromoxynil than a susceptible population (Thompson et al. 2012). The first case of HPPD-resistant waterhemp occurred in central Illinois, USA, which was found to have 10-fold resistance to mesotrione when compared to sensitive biotypes (Hausman 2011).

Metabolism of mesotrione was reduced following application of the cytochrome P450 monooxygenase inhibitors malathion or tetcyclacis, which suggests that enhanced oxidative metabolism contributes to mesotrione resistance (Ma et al 2013). Inheritance experiments reported metabolism-based atrazine resistance is conferred by a single major gene, while inheritance of mesotrione resistance is more complex (Huffman et al. 2015). Reduced translocation of mesotrione at high temperatures (40/30°C) has been demonstrated in HPPD-resistant Palmer amaranth (Godar et al. 2015), indicating another possible mechanism of resistance.

1.1.3 EPSP Inhibitors

The enzyme 5-enolpyruvylshikimate-3-phosphate synthase (EPSPS) catalyzes the transfer of PEP to shikimate-3-phosphate (S3P), to produce EPSP and inorganic phosphate (Powles and Preston 2006). These products are vital in the synthesis of the aromatic amino acids tryptophan, tyrosine, and phenylalanine, which are critical for production of hormones and other critical plant metabolites (Powles and Preston. 2006; Shaner 2014). Glyphosate is an inhibitor of EPSP synthase in the shikimate production pathway of plants and is competitive in regards to the binding site for phosphoenolpyruvate (PEP) (Amrhein 1980; Franz et al. 1997). Inhibition of EPSPS results in shikimic acid accumulation and the plant is deprived of amino acids and EPSP.

Glyphosate has been widely used for many years due to low mammalian toxicity, low weed management costs, and crops engineered to be resistant to this herbicide. Genetically modified crops for glyphosate resistance express a bacterial EPSPS that is insensitive to glyphosate and may express a gene endowing glyphosate metabolism (Padgette et al. 1996). Glyphosate-resistant soybean varieties were commercialized in the USA in 1996, and by 2004, 85 percent of all soybeans produced were herbicide resistant (Dill 2005). The herbicide is applied post-emergence and is rapidly inactivated by soil organisms. Sensitive plants display foliar symptoms of chlorosis and necrosis of the actively growing tissue 10 to 20 days after treatment (Shaner 2014).

Resistance mechanisms to glyphosate include gene amplification of the target site, altered translocation, and insensitive target site (Dill 2005; Powles and Preston 2006). The first target-site resistance for glyphosate was documented in Malaysian populations of goosegrass (*Eleusine indica*) (Powles and Preston 2006). A mutation from proline to serine at amino acid position106 resulted in a 5-times higher resistance to glyphosate than the susceptible biotype. A

proline-to-threonin mutation also confers glyphosate resistance in goosegrass (Comai et al. 1983; Ng et al. 2004; Powles and Preston 2006). Glyphosate-resistant Palmer amaranth was first reported in Georgia, and experiments on this population suggested an altered target site as a source of resistance (Culpepper et al. 2006). Glyphosate resistance in an Australian population of rigid ryegrass (*Lolium rigidum*) resulted from accumulated glyphosate in the tip of the treated leaf, with little translocation to the roots (Lorraine-Colwill et al. 2002). Biotypes of Palmer amaranth from a North Carolina population absorbed less glyphosate compared with the sensitive biotype (Whitaker et al. 2013). However, results also supported an increase in EPSPS gene copy number (Whitaker et al. 2013). Palmer amaranth populations resistant to glyphosate have been shown to produce multiple copies of the EPSPS gene to confer resistance (Gaines et al 2010). Genomic copy numbers between 30 and 50 are necessary to survive typical field applications rates between 0.47 and 0.99 kg ha^{-1} (Gaines et al. 2011). A total of 37 grass and broadleaf species are resistant to glyphosate (Heap 2017).

1.1.4 PPO Inhibitors

The porphyrin pathway synthesizes heme and chlorophyll, which is important for photosynthetic processes (Duke et al. 1994). The precursor of this pathway is 5-aminolevulinic acid (ALA), which is produced from glutamate then converted to Protoporphyrin IX (Jacobs et al. 1991). The enzyme protoporphyrinogen oxidase (Protox or PPO) converts protoporphyrinogen IX (Protogen IX) to Protoporphyrin IX (Proto IX) in the chloroplast and mitochondria (Duke et al. 1994). The Protox enzyme is the target site of many classes of PPO inhibitors including diphenyl ethers, cyclic imides, oxadiazoles, phenylphthalimides, triazolinones, and thiadiazolidines (Li et al. 2004; Matsumoto 2002).

PPO-inhibiting herbicides are competitive with Protogen IX to bind with the Protox enzyme and cause the Protogen IX substrate to "leak" into the cytoplasm via extraplastidic oxidation (Hirai et al. 2002; Lee and Duke 1994). The substrate is then converted to Proto IX, a photosensitive chemical, which then transfers the energy from light to oxygen to create a singlet oxygen (Shaner 2014). The conversion of Protogen IX to Proto IX in the cytoplasm keeps the singlet oxygen segregated from the antioxidants and enzymatic protective mechanisms (ascorbate, tocopherols, and reduced glutathione) that are in the chloroplast, which in turn does not trigger any feedback inhibition for this reaction (Duke et al. 1994). The singlet oxygen initiates a chain reaction of lipid peroxidation, which results in chlorophyll loss and leaky membranes, allowing cells to dry and disintegrate (Duke et al. 1994).

PPO-inhibiting herbicides can be applied to the soil or plant foliage. Many PPO-inhibiting herbicides have poor translocation, which can cause reduction in efficacy (Matsumoto 2002). These herbicides are light-induced, with foliage of susceptible plants becoming necrotic within days of herbicide application (Hirai et al. 2002; Shaner 2014). Crop selectivity is due to raped metabolic detoxification by GST enzymes and moderate leaf absorption (Grossman and Schiffer 1999). Soybeans are tolerant to this class of herbicides and may only exhibit "bronzing" on expanded leaves (Shaner 2014). Currently, 11 species, including Palmer amaranth, are resistant to PPO herbicides (Heap 2017).

The mechanism of PPO-inhibitor resistance was first discovered in a waterhemp biotype from Illinois that had a unique target-site codon deletion (Patzoldt et al. 2005). This resistance mechanism involves the loss of an amino acid codon for glycine at position 210 (ΔG210) of the PPX2 gene, which encodes the mitochondrial isoform of PPO (Patzoldt et al. 2005). Two new mutations of PPX2 were discovered at the R98 site (R98G and R98M), which likely confer

resistance to PPO-inhibitors in Palmer amaranth (Giacomini et al. 2017). These two mutations occurred alongside the widespread ΔG210 mutations, indicating high selection pressure (Giacomini et al. 2017).

1.1.5 Glufosinate inhibitors

Glufosinate is a structural analog of the amino acid glutamate, which is the substrate for the glutamine synthetase enzyme. The herbicide is a competitive inhibitor with glutamate for glutamine synthetase enzyme binding (Sauer et al. 1987). The herbicide inhibits the pathway responsible for assimilating ammonia and incorporating ammonia into a reduced organic form such as the amino acids glutamine and glutamate. In Palmer amaranth, as the ammonia accumulation increases, stomatal conductance decreases (Coetzer and Al-Khatib 2001). The buildup of ammonia is toxic to the plant, but the primary cause of death is due to the inhibition of transamination reactions in photorespiration (Sauer et al 1987; Wild et al. 1987). Without amino group donors, glyoxylate, glycolate, and phosphoglycolate accumulate and inhibit the enzyme RuBisCo and subsequent carbon fixation in the Calvin cycle, which indirectly leads to inhibition of photosynthetic electron transport (Timm et al. 2016; Wild et al. 1987). Development of singlet oxygen leads to lipid peroxidation resulting in cell death. Glufosinate is not translocated in plants and therefore mainly affects the foliage where the herbicide is applied (Coetzer and Al-Khatib 2001; Shaner 2014). Herbicide injury occurs rapidly on sensitive species and herbicidal activity has been reported to be enhanced by increased light intensity (Köcher 1983).

There is no natural crop mechanism for glufosinate resistance, so crops are genetically modified using bacterial genes from *Streptomyces viridochromogenes* (Droge et al. 1992). The bar or PAT gene codes for phosphinothricin acetyltransferase (PAT enzyme) with detoxifies glufosinate by acetylating the herbicide molecule (Devine et al. 1993). Glufosinate is a non-

selective, non-systemic herbicide that has no residual activity in the soil (Shaner 2014). Symptoms of chlorosis and necrosis appear rapidly on sensitive plants treated with this herbicide. Currently, only 3 monocot species are resistant to this herbicide (Heap 2017).

1.1.6 Very-long-chain fatty acid inhibitors

Fatty-acids are created via fatty acid synthase located in the plastid at the Acyl-carrier protein (ACP) (Schmalfuβ et al. 2000). Malonyl-CoA is the two carbon unit donor involved in the synthesis of VLCFA (Cassange et al. 1994). VLCFAs are classified as possessing 20 or more carbon atoms, and the elongation process to VLCFAs occurs in the endoplasmic reticulum (ER) (Cassange et al. 1994; Schmalfuβ et al. 2000). The majority of VLCFA are located in the plasma membrane and when absent, the membrane loses stability and becomes leaky, which results in plant death (Matthes and Böger 2002). Inhibition of VLCFA elongation impedes cuticle synthesis and membranes, thus hindering the ability of a developing seedling shoot to emerge properly from the soil (Böger et al. 2000). Germination is not inhibited, but rather the shoot elongation of the germinated seed (Tanetani et al. 2009). Use rates are typically in low concentrations comparatively and weed resistance is rare (Götz and Böger 2004). Herbicide injury symptoms in grasses include improper unfurling of the leaf from the coleoptile (buggy-whipping), and in soybeans injury is evident by slow emergence and crinkled or cupped leaves (Fuerst 1987). Only 5 grass species are resistant to herbicides from this site-of-action group.

Chloroacetamide herbicides, such as acetochlor and *S*-metolachlor, impede VLCFA synthesis and have been commonly used preemergence (PRE) in corn and soybean (Fuerst 1987). Chloroacetamide herbicides are absorbed by shoot and root and translocated in both the xylem and the phloem (Fuerst 1987). Crop selectivity is possible by enhanced metabolism due to the ability to rapidly synthesize glutathione, maintaining glutathione in the reduced state, or a

glutathione S-transferase (GST) present in higher levels or with specificity for acetamide selectivity (Fuerst 1987). Plants are able to detoxify chloroacetamides by conjugation with glutathione or homoglutathione in legumes to a non-toxic form (Fuerst 1987).

Herbicides in the pyrazole family (pyroxasulfone) inhibit VLCFA elongases (Tanetani et al. 2011). Pyroxasulfone has a lower use rate than other VLCFA synthesis-inhibiting herbicides, ranging from 60–250 g ai ha^{-1} (Yamaji et al. 2014). Pyroxasulfone is a selective herbicide that is applied preemergence in corn, soybeans, wheat, cotton, and other crops (Yamaji et al. 2014). Selectivity is observed in corn and soybeans and may be due to GST detoxification (Tanetani et al. 2009). A metabolism study between susceptible and tolerant grass species determined that cleavage of a methylenesulfonyl linkage by glutathione conjugation plays a significant role in detoxification (Tanetani et al. 2013). This study indicates that there are differences in physiological activities controlling metabolism of pyroxasulfone (Tanetani et al. 2013). Injury by this herbicide results in slight twisting in corn and leaf cupping in soybeans, and both crops exhibited growth inhibition (Yamaji et al. 2014).

1.1.7 Auxin Inhibitors

Natural auxin (Indole-3-acetic acid) (IAA) causes phototropic movements and regulates growth in plants (Sterling and Hall 1997). This hormone is found in actively growing meristems and is transported through the phloem parenchyma cells. A receptor for auxin was discovered in 2005 and was identified as the F-box protein transport inhibitor response 1 (*TIR1*) (Guilfoyle 2007). Auxin/IAA bind to the *TIR1* receptor and either represses or activates gene expression. Gene expression activates most processes regulated by this plant hormone (Guilfoyle 2007).

Herbicides that mimic IAA are referred to as synthetic auxin herbicides. Also known as plant growth regulators (PGRs), these herbicides are translocated in the xylem and the phloem.

PGR herbicides regulate cell division and elongation, and developmental processes such as vascular tissue and floral meristem differentiation, leaf initiation, phyllotaxy, senescence, apical dominance, and root formation (Grossman 2010). These herbicides cause plant death due to uncontrolled, unregulated auxin activity in the plant. The auxin has a promiscuous binding site on the receptor and in turn acts as "molecular glue" that strengthens the substrate binding to the receptor (Guilfoyle 2007), rendering the plant unable to regulate auxin production. The deregulation of plant growth by PGRs has been described by Grossman (2010) in three phases following uptake of the PGR herbicide in a dicot plant. The first stage is stimulation, which occurs after herbicide application, and metabolic processes such as ethylene biosynthesis is stimulated through the induction of 1-aminocyclopropane-1-caboxylic acid (ACC) synthase (Grossman 2010). The second phase includes the inhibition of root and shoot growth, decreased internode elongation and leaf area, intensified pigmentation, stomatal closure, reduced transpiration, and overproduction of reactive oxygen species (ROS). The overproduction of these hormones along with ROS cause rapid leaf senescence followed by tissue decay, chlorosis, and eventual plant death (Grossman 2009; Grossman 2010).

Synthetic auxin herbicides cannot be metabolized, compartmentalized, or regulated in sensitive dicot species (Kelley and Riechers 2007). PGR herbicides have activity primarily in sensitive dicot species rather than grasses. One possible explanation for this selectivity is a difference in metabolism. Grasses rapidly convert synthetic auxins to inactive metabolites with irreversible ring hydroxylation. Sensitive dicots convert the synthetic auxin to amino acid conjugates, which can be converted back to the active auxin, so an active pool of herbicide remains (Kelley and Riechers 2007).

Sensitive plants exhibit symptoms of epinastic twisting of leaves, leaf cupping, and swollen nodes with necrosis occurring in two to four weeks (Shaner 2014). Roots become thickened (braced) and stunted, and adventitious roots can develop (Sterling and Hall 1997). Auxin herbicides have long been used to control broadleaf species in grain crops such as corn (*Zea may* L.), wheat (*Triticum aestivum* L.) and grain sorghum [*Sorghum bicolor* (L.) Moench] (Mithila et al. 2011). Previous evaluation of broadleaf weed management in dicamba-resistant soybean reported only 60% of Palmer amaranth controlled at preemergence using 0.25 lb ae acre^{-1} (Johnson et al. 2010). 2, 4-D (230–1060 g ae ha^{-1}) and dicamba (280–1120 g ae ha^{-1}) applied to 13–20-cm tall Palmer amaranth plants provided 68 to 80% and 59 to 83% control respectively (Merchant et al. 2013). Currently 26 broadleaf species have been documented resistant to synthetic auxin herbicides (Heap 2017).

An *Amaranthus* species, tall waterhemp (*Amaranthus tuberculatus*), has been documented to be 10-fold more resistant to 2, 4-D than a susceptible biotype (Bernards et al. 2012). Increasing doses of 2,4-D induced up-regulation or down-regulation of different genes in the ethylene and abscisic acid pathways, indicating several receptor sites may cause resistance in *Arabidopsis* (Raghavan et al. 2006). A kochia (*Kochia scoparia*) population is resistant to dicamba due to a quantitative trait resulting from a number of small changes to a gene (Cranston et al. 2001). However, another kochia population resistant to dicamba was reported to be caused by a single dominant allele (Preston et al. 2009). As demonstrated by the two conflicting studies, the mechanism of action of auxinic herbicides and resistance is difficult to determine due to the multiplicity of biochemical effects within the cell (Coupland 1994).

1.2 Palmer Amaranth Biology

Palmer amaranth (*Amaranthus palmeri*) is a summer annual, small-seeded broadleaf species that originates from the Sonoran desert of North America (Sauer 1957; Ward et al. 2013). This plant is in the Amaranthaceae family in the order Centrospermae, a group that contains anthocyanin pigments (Steckel 2007). Palmer amaranth has been expanding into the Midwest and farmers are concerned that this weed will have the same damaging effect on their fields as Palmer amaranth has had in areas of the South. Studies conducted by Davis et al. (2015) investigated the importance of genetics and environmental factors that would help determine the extent of the damage niche Palmer amaranth would inflict upon the Midwest. McDonald et al. (2009) hypothesized that increased temperatures would expand the northern damage niche of southern originating weed species. Temperature increase would be beneficial for Palmer amaranth because of this species' positive response to increased temperature (Ehleringer 1983; Guo and Al-Khatib 2003). The damage niche for Palmer amaranth is not reliant on the genotype of this weed, but rather the growing environment in which the seed is dispersed (Davis et al. 2015).

Palmer amaranth can be identified by a glabrous stem with petioles that are longer than the ovate leaf blade. The leaves are alternate and sometimes have a chevron (Ward et al. 2013). Palmer amaranth is dioecious species with the male plants producing pollen and the female plants producing seeds. Both inflorescences can grow up to a meter in length. The female inflorescence is distinguishable from the male inflorescence by stiff bracts that are sharp to the touch. The females produce a prolific amount of seed, often ranging between 200,000–600,000 seeds when the plant emerges from March to June (Ward et al. 2013). The utricle in 1.5 to 2 mm long with dark red to black seed 1 to 1.25 mm in diameter (Sauer 1955; Ward et al. 2013).

Palmer amaranth is able to overwhelm a field with multiple emergence events, predominantly after a soil disturbance or rainfall event, due to originating and evolving in frequently rewashed alluvium (Sauer 1957). The seeds germinate throughout the growing season, which necessitates multiple herbicide applications throughout the year (Ward et al. 2013).

Palmer amaranth has a photosynthetic rate around 80 μmol m^{-2} s^{-1} and therefore is able to rapidly accumulate biomass (Ehleringer 1983). Palmer amaranth also has a high photosynthetic rate due the diaheliotropic movement of the leaves (Ehleringer 1983). This weed has been recorded to be 10 centimeters tall 2 weeks after planting and 24 centimeters at 4 weeks (Sellers et al. 2003). Palmer amaranth can grow 0.18 to 0.21 centimeters per growing degree day (GDD), allowing this weed to quickly outcompete the crop in the field (Horak and Loughin 2000). Palmer amaranth competition with soybean can cause a 17–63% yield reduction at 0.33 – 10 plants per m^{-1} (Klingaman and Oliver 1994) or 79% yield reduction at 8 plants per m^{-1} (Bensch 2003) when this weed emerges shortly after soybean. Palmer amaranth can reduce corn yield 11–91 % when emerging the same time as corn at a density of 0.5–8 plants m^{-1}, while emergence after corn resulted in 7–35% yield reduction at the same plant densities (Massinga et al. 2001).

1.3 Research Objectives

Palmer amaranth is a devastating weed in southern areas of the United States. This weed has been expanding to northern states and is a concern for growers because of yield reduction and management challenges as Palmer amaranth has exhibited resistance to herbicides from six site of action groups. Evaluation of herbicide management strategies is necessary so that prevention strategies can be utilized to preserve the effectiveness of herbicide programs. The objectives of this project were to determine the best herbicide strategies to manage Palmer

amaranth in Illinois agronomic systems. Experiments were designed to determine the optimal postemergence herbicide application timing with respect to weed height. The effectiveness of multiple herbicide applications for Palmer amaranth control in a soybean system was evaluated. Growth parameters were measured to determine the duration of Palmer amaranth emergence and biomass accumulation throughout the growing season. Rate titrations of synthetic auxin herbicides were evaluated to determine the effective dose to control Palmer amaranth. Pre emergence herbicides were evaluated to determine their length of residual control.

1.4 Stratification Technique

The technique for stratifying small seeded *Amaranthus* species of using a 1:1 bleach and distilled water solution (Evans et al. 2015) was too intense for Palmer amaranth seeds from 2015. Approximately 58 percent of those treated seeds had a viable embryo when evaluated with a tetrazolium test. Experiments were conducted on the use of a dilution of gibberellic acid as well as an ethylene dilution. Germination was measured when the radicle was visibly protruding from the seed coat.

Palmer amaranth seed germination enhancement with gibberellic acid consisted of 50 seeds placed in a petri dish with one petri paper. A 10^{-3} dilution of gibberellic acid was applied at 9 milliliters (mL) of solution to the petri paper. The dish was sealed and placed in the greenhouse. Germination reached 90 percent, but stems appeared spindly and germination was not uniform.

Similar to the methods of Kępczyński and Sznigir (2013) a 10^{-4} M ethylene dilution was created with ethephon. The ethylene dilution was applied at a volume of 9 mL in each petri dish. Petri dishes were filled with 50 Palmer amaranth seeds and three petri papers, then sealed and stored at 4°C for two or four weeks. Both time periods for ethylene treatments produced 90

percent of seeds germinating and the treatment had a uniform germination pattern without the spindly stem growth from the gibberellic acid treatment.

A modified method of ethylene stratification consisted of the bleach stratification technique, but a 10^{-4} M ethylene dilution was used instead. Seeds were placed in an Eppendorf tube to the 0.05mL line. Then 0.95 mL of ethylene dilution was added to the tube. The seeds were vortexed periodically for two hours to ensure that all seeds had made contact with the solution. After that time period, the ethylene solution was removed and the seeds were rinsed with 0.95 milliliters of distilled water for ten minutes. This step was repeated twice and then 0.95 milliliters of 0.1% agarose was added to the tube for seeds to be stored. The treated seed was usable for planting immediately after stratification.

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CHAPTER 2

PALMER AMARANTH EMERGENCE DURATION AND GROWTH IN ILLINOIS

2.1 Abstract

Palmer amaranth has been spreading from the southern United States into the Midwest, and the extent of damage this weed can inflict to Illinois row crops is not yet known. Palmer amaranth is known for rapid biomass accumulation and multiple emergence events, making this weed difficult to control. The objectives of this study were to determine the duration of Palmer amaranth emergence and biomass accumulation during two Illinois growing seasons. Field experiments were conducted in 2015 and 2016 near Kankakee, Illinois and in 2016 in Urbana, Illinois. Quadrats were marked in untreated plots that had either no crop or planted with soybeans. Emerged Palmer amaranth were counted weekly and harvested for aboveground biomass. Individual Palmer amaranth plants were marked every 2 and 3 weeks in 2015 and 2016, respectively, and their height recorded weekly thereafter. Marked plants were harvested for above ground biomass at the end of the growing season. Palmer amaranth plants emerged for 10 weeks in 2015 and 9 weeks in 2016. A negative correlation was noted in 2015 with Palmer amaranth emergence and high amounts of rainfall. Biomass was also affected by the weather in 2015, with the biomass accumulation averaging 9 g per plant while in 2016 averaging 427.6 and 269.2 g per plant in Kankakee and Urbana, respectively. Similar to previous research, Palmer amaranth height accumulation was 0.2 cm per growing degree day. Plants in the earliest marked cohort accumulated the greatest biomass and height, but all marked Palmer amaranth plants flowered within a similar time frame.

2.2 Introduction

Palmer amaranth (*Amaranthus palmeri*) is a summer annual, small-seeded broadleaf species that originates from the Sonoran desert of North America (Sauer 1957; Ward et al. 2013). This plant is in the Amaranthaceae family in the order Centrospermae, a group that contains anthocyanin pigments (Steckel 2007). Palmer amaranth has been expanding into the Midwest and farmers are concerned that this weed will have the same damaging effect on their fields as Palmer amaranth has had in areas of the South. Davis et al. (2015) investigated the genetics and environmental factors that would help determine the damage niche of Palmer amaranth in the Midwest. They concluded the damage niche of Palmer amaranth is not reliant on genotype, but rather the growing environment in which the seed is dispersed. McDonald et al. (2009) hypothesized that increased temperatures would expand the damage niche of southern-originating weed species northward. This temperature increase would be beneficial for Palmer amaranth growing conditions because of this species' positive response to increased temperature (Ehleringer 1983; Guo and Al-Khatib 2003).

Palmer amaranth can be identified by a glabrous stem with petioles that are longer than the ovate leaf blade. The leaves are alternate and sometimes have a chevron mark on the leaf (Ward et al. 2013). Palmer amaranth is a dioecious species with male plants producing pollen and female plants producing seeds. Both inflorescences can grow up to a meter in length. The female inflorescence is distinguishable from the male inflorescence by stiff bracts that are sharp to the touch. The females produce a prolific amount of seed, often ranging between 200,000–600,000 seeds when the plant emerges from March to June (Ward et al. 2013). The utricle is 1.5 to 2 mm long with dark red to black seed 1 to 1.25 mm in diameter (Sauer 1955; Ward et al. 2013). Location on the inflorescence determines when the seed matures, and seeds on the top and

middle third of the inflorescence have a 67–78% greater germination rate than seeds lower on the inflorescence (Jha et al. 2010). Palmer amaranth is able to overwhelm a field because of multiple emergence events within a growing season, which attributes to the species originating and evolving in frequently rewashed alluvium (Sauer 1957). The seeds germinate at different times, which necessitates multiple herbicide applications throughout the year (Ward et al. 2013).

Seed germination is stimulated by natural or red light while far-red light inhibits germination (Jha et al. 2010), suggesting that when seed is under crop canopy, germination will be decreased due to less light. Germination occurs over a wide range of temperatures and the increase of temperature also leads to an increase in germination, with the peak occurring at approximately 30°C (Steckel et al. 2004). Research indicates a narrow window of opportunity to control Palmer amaranth before the plant is too large during periods of higher temperatures (Powell 2014).

Palmer amaranth is a C4 species with a photosynthetic rate around 80 μmol m^{-2} s^{-1} and rapid biomass accumulation (Ehleringer 1983). Palmer amaranth utilizes this high photosynthetic rate via diaheliotropic movement of the leaves (Ehleringer 1983). Previous research has reported Palmer amaranth to be 10 cm tall two weeks after planting and 24 cm two weeks later (Sellers et al. 2003). Palmer amaranth can grow 0.18 to 0.21 cm per growing degree day (GDD), allowing this weed to quickly outcompete many crops (Horak and Loughin 2000). Palmer amaranth competition with soybean can cause a 17–63% yield reduction at 0.33–10 plants per m^{-1} (Klingaman and Oliver 1994) or 79% yield reduction at 8 plants per m^{-1} (Bensch et al. 2003) when this weed emerges shortly after soybean. Palmer amaranth can reduce corn yield 11–91% when emerging at the same time as corn at a density of 0.5–8 plants m^{-1}, or 7 –35% when Palmer amaranth emerged after corn at the same densities (Massinga et al. 2001).

The objectives of this study were to determine the duration of Palmer amaranth emergence and the amount of biomass that can be accumulated throughout an Illinois growing season.

2.3 Materials and Methods

2.3.1 Field Design and Implementation

Field experiments were conducted in Kankakee County, IL in 2015 and 2016 and Urbana, IL in 2016. The soil at Kankakee was a Kankakee fine sandy loam (loamy-skeletal, mixed, superactive, mesic Typic Hapludolls) with a pH of 6.5 and organic matter of 2%. The Urbana soil type was a Flanagan silt loam (fine, smectitic, mesic Aquic Argiudolls) with a pH of 6.5 and organic matter of 4.9%. All plots were 3 meters wide and 7.6 meters long at Kankakee and 10 meters long at Urbana. Quadrats used in Kankakee were ½ m^2, while a 1 x ½ m^2 quadrat was used at Urbana. Differences in quadrat sizes was due to a much higher Palmer amaranth density at Kankakee compared with Urbana.

Tillage was implemented each spring to remove any existing vegetation and prepare the seedbed for planting. Glufosinate-resistant soybean cultivar "Credenz 3233 LL" was planted in 76-cm rows and soybean planting was simulated on bare ground trials beginning on May 19th, 2015 and May 26th, 2016 in Kankakee and June 8th, 2016 in Urbana. Experiments were organized in a randomized complete block design.

2.3.2 Duration of Palmer amaranth emergence

Quadrats were marked near the center of untreated plots with six repetitions in bare ground and four repetitions in soybean trials to determine Palmer amaranth emergence with and without crop competition. Palmer amaranth emergence was recorded weekly. Emerged plants in the quadrats were harvested for above ground biomass and dried for three days at 65°C. Biomass was recorded for analysis of weekly and cumulative biomass of Palmer amaranth.

2.3.2.1 Statistical analysis

Emergence data were analyzed in SAS 9.4[2] (SAS Institute, Cary, NC 27513, USA) using the PROC MIXED procedure. Analysis revealed a significant year and location interaction and, therefore, the data for location and years were not pooled. Fixed effects were year and treatment while random effects were block and replication within block. Above ground biomass and plant counts were evaluated in SAS using the PROC GLM procedure. Means of significant main effects and interactions were separated using Fischer's Protected LSD test at ($P \leq 0.05$).

2.3.3 Season-long Palmer amaranth biomass accumulation

Six individual Palmer amaranth plants were marked every third week in 2015 and every second week in 2016 to determine overall height at the end of the growing season. The total number of marked Palmer amaranth per field totaled 18 and 24 in of 2015 and 2016, respectively. Palmer amaranth seedlings were marked when the plant was at least 1cm tall and the first true leaf was developed. Plants were marked with a numbered stake on the right side of the plant and the area around the marked plant cleared of all other vegetation. When the weed was 60 cm in height, tags with the corresponding stake number were placed loosely around the stem. Plants were tagged so they were clearly visible. Height of each marked weed was recorded weekly for 14 weeks. Marked plants were harvested on August 21st in 2015 and August 26th and 23rd in 2016 Kankakee and Urbana, respectively. Plants were flowering and growth had slowed or stopped at the time of harvest. Harvested biomass was then dried for 30 days at 65°C and biomass then recorded.

2.3.3.1 Statistical Analysis

The accumulation of biomass per growing degree day (GDD) was calculated using a baseline temperature of 10°C (50°F). GDDs were calculated from cumulative thermal times following a modified model (Russelle et al. 1984):

$$GDD = ([\{ T_{max} + T_{min}\} /2] - T_B)$$

where T_{max} and T_{min} are the maximum and minimum daily temperatures of the given day, respectively, and T_B is the base temperature below which little or no growth occurs. A T_B of 10° C was followed based on minimum germination temperatures for these species (Horak and Loughlin 2000).

Biomass was evaluated in SAS using the PROC MIXED procedure and all possible main effects and interactions were tested. Due to interaction by year, years were kept separate, however, location was considered an environment as suggested by Carmer et al. (1989). Environments, replications (nested within environments), and all interactions containing either of these effects were considered random effects; all other variables (cohort timing) were considered fixed effects. Means of significant main effects and interactions were separated using Fischer's Protected LSD test at $P \leq 0.05$.

2.4 Results and Discussion

2.4.1 Duration of Palmer amaranth emergence

Rainfall amounts were high in 2015 (Table 2.1) and due to interaction by year, data were not pooled and data are separated by location and year. Flooding occurred and soil was often well saturated in 2015 at Kankakee. Palmer amaranth emerged for 10 weeks in 2015 and 9 weeks in 2016 at both locations. Comparisons between years indicated emergence was greater for bare ground emergence in 2016 (Table 2.2). Bare ground quadrats had abundant Palmer amaranth

emergence, likely due to favorable weather conditions in 2016 (Table 2.1). Emergence patterns followed a trend of decreasing after copious rainfall events that left soils overly saturated (Figure 2.1–2.3).

Palmer amaranth has a prolonged emergence pattern during the growing season. Observations corresponded with Powell (2014) that Palmer amaranth emerges in early May and continues until late September. Total Palmer amaranth emergence was lower in soybean quadrats for both locations in 2016, possibly due to crop shading the weed seed. Crop canopy can have a suppressive effect on emergence due to altering the light quality of red to far red ratio, which is vital for germination (Jha et al. 2010; Norsworthy 2004).

2.4.2 Season-long Palmer amaranth biomass accumulation

Two locations in 2016 were beneficial to determine year effect rather than genotype because the competitiveness of Palmer has been shown to vary based upon the site-year instead of weed genotype (Davis et al. 2015). Cohort 1 had biomass in 2015 that reached 9.1 g, while in 2016, this same cohort timing accumulated 348.8 g of biomass (Table 2.3). The lower accumulation of biomass in 2015 is similar to observations that growth is reduced when the temperature is 25°C, which results in half the rate of optimum photosynthesis (Ehleringer 1983). Previous research has concluded that Palmer amaranth height increases at a rate of 0.18 to 0.21 centimeters per growing degree day (Horak and Loughin 2000). Results herein were similar to this previous finding, however, growth became more linear after first true leaves were developed and decreased as maturity was reached (Figures 2.4–2.5). Earlier emerging plants accumulated the most biomass and attained greatest heights, however, by harvest all cohorts had reached the reproductive stage regardless of emergence date. Flowering for all marked Palmer amaranth occurred around similar time periods, which has been shown to be proportional to the amount of

thermal time and growing degree days (Davis et al. 2015). These results are expected since Palmer amaranth is a short-day species (Keeley et al. 1987).

2.4.3 Palmer amaranth emergence and biomass accumulation

Palmer amaranth has a long period of germination, which could cause concerns for row crop producers when planning herbicide applications. While 10 weeks was observed during the study, germination occurred before trials were implemented. Jha and Norsworthy (2009) observed long periods of germination from May to September and April to August. Germination throughout the season may necessitate multiple applications of postemergence herbicides to be utilized. Late season germination achieved flowering, which can then produce more Palmer amaranth seeds to germinate during the next growing season. Due to the rapid growth exhibited by this weed, the window of opportunity for timely herbicide application may be limited. Maximum relative growth rates observed by Horak and Loughin (2000) were greatest for Palmer amaranth and therefore herbicide application needs to be based on Palmer amaranth height. While the weather can greatly affect the emergence and growth, weed pressure was still abundant for both growing seasons, therefore making Palmer amaranth control critical in Illinois cropping systems.

2.5 Source of Materials

[1]Statistical Analysis Software (SAS) 9.4. SAS Institute, Inc., 100 SAS Campus Drive, Cary, NC 27513.

2.6 Tables and figures

Table 2.1. Weekly growing degree days and rainfall for Kankakee (2015 and 2016) and Urbana (2016)

	GDD50[a]			precipitation		
	Kankakee		Urbana	Kankakee		Urbana
Week[b]	2015	2016		2015	2016	
					(cm)	
0	117.9	197.8	67.5	0.1	2.5	1.5
1	217	132.7	141	1.1	0.5	1.1
2	136.5	185.4	190.5	2.0	1.3	1.7
3	231.2	177.5	185	2.7	1.9	2.7
4	170	149.5	135	6.9	0.1	0.1
5	169	138.5	137	17.4	1.3	1.6
6	195.3	181.1	180.5	1.5	0.7	0.7
7	143.2	177.9	186.5	2.8	3.4	0.5
8	196.8	150.5	169	0.1	0.6	1.6
9	132.1	187.7	128	0	0.3	0.1
10	194.1	169.5	186	0	0.04	0
11	179.5	227.1	213.5	0.4	2.4	2.5
12	17.4	240.8	157	0.1	2.4	0.6

[a] Growing Degree Day, GDD; calculated with a base temperature of 10°C (50°F)

[b] Weather data collected in Kankakee starting May 19th in 2015 and May 26th in 2016. Urbana weather data collected starting June 3rd in 2016

Table 2.2. Palmer amaranth mean emergence and biomass in Kankakee (2015 and 2016) and Urbana (2016) under field conditions. Means sharing the same letter within a column are not significantly different.

	Kankakee				Urbana	
	2015		2016		2016	
	emergence	biomass (g)	emergence	biomass (g)	emergence	biomass (g)
bare ground	171.3 a	1.8 a	318.5 a	9.7 a	61.8 a	1.6 a
soybeans[a]	162.8 a	1.8 a	216.8 b	5.7 b	8.5 b	0.2 b

[a] glufosinate-resistant soybean (*Glycine max*) variety "Credenz 3233 LL"

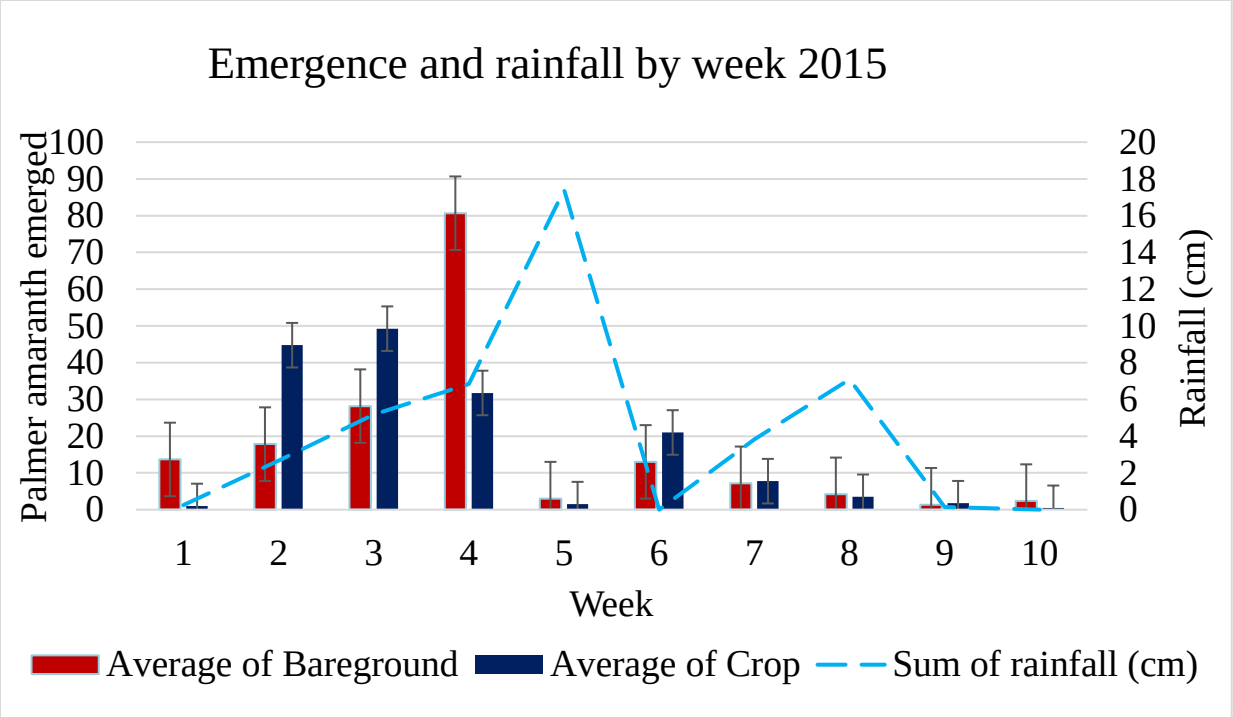
[b] Kankakee 2015 week 1 recorded on May 26; Kankakee 2016 week 1 recorded on June 2; Week 1 in Urbana recorded June 2nd.

Table 2.3. Palmer amaranth height and biomass means at harvest* in 2015 and 2016. Means sharing the same letter within a column are not significantly different.

	2015		2016	
cohort[a-d]	height	biomass	height	biomass
	(cm)	(g)	(cm)	(g)
1	95.3 a	9.1 a	195.3 a	348.8 a
2	43.3 b	2.7 a	124.3 b	27 b
3	48 b	3.4 a	61.7 c	2.4 b
4	-	-	35 c	0.8 b

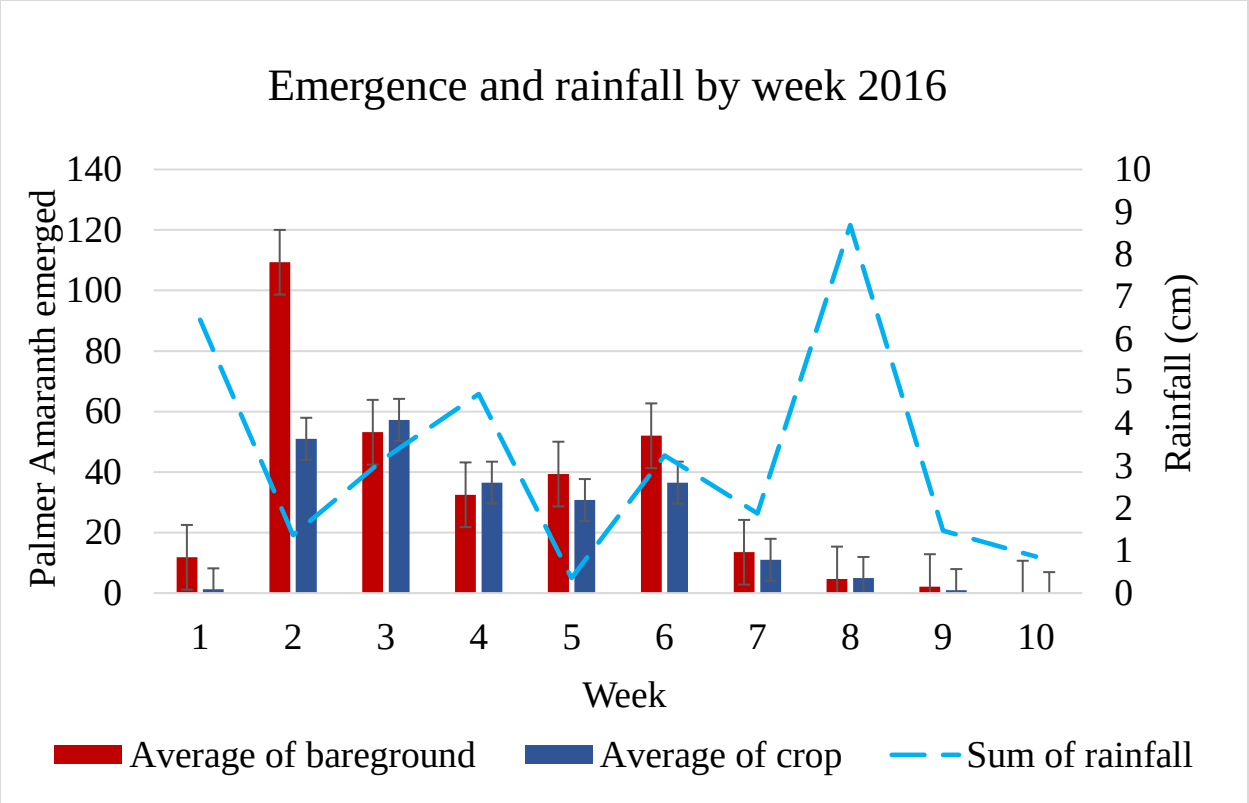
[a] Cohort 1 marked Kankakee 2015, June 2nd; Kankakee 2016, June 2nd; Urbana 2016, June 3rd.
[b] Cohort 2 marked June 23rd, Kankakee 2015; June 16th, Kankakee 2016; June 17th, Urbana 2016.
[c] Cohort 3 marked July 15th, Kankakee 2015; June 30th, Kankakee 2016; June 30th, Urbana 2016.
[d] Cohort 4 marked July 14th Kankakee 2016; July 14th Urbana 2016.
* Plants were harvested on August 21st in 2015 and August 26th and 23rd in 2016 Kankakee and Urbana, respectively

Figure 2.1. Palmer amaranth emergence and rainfall in Kankakee in 2015



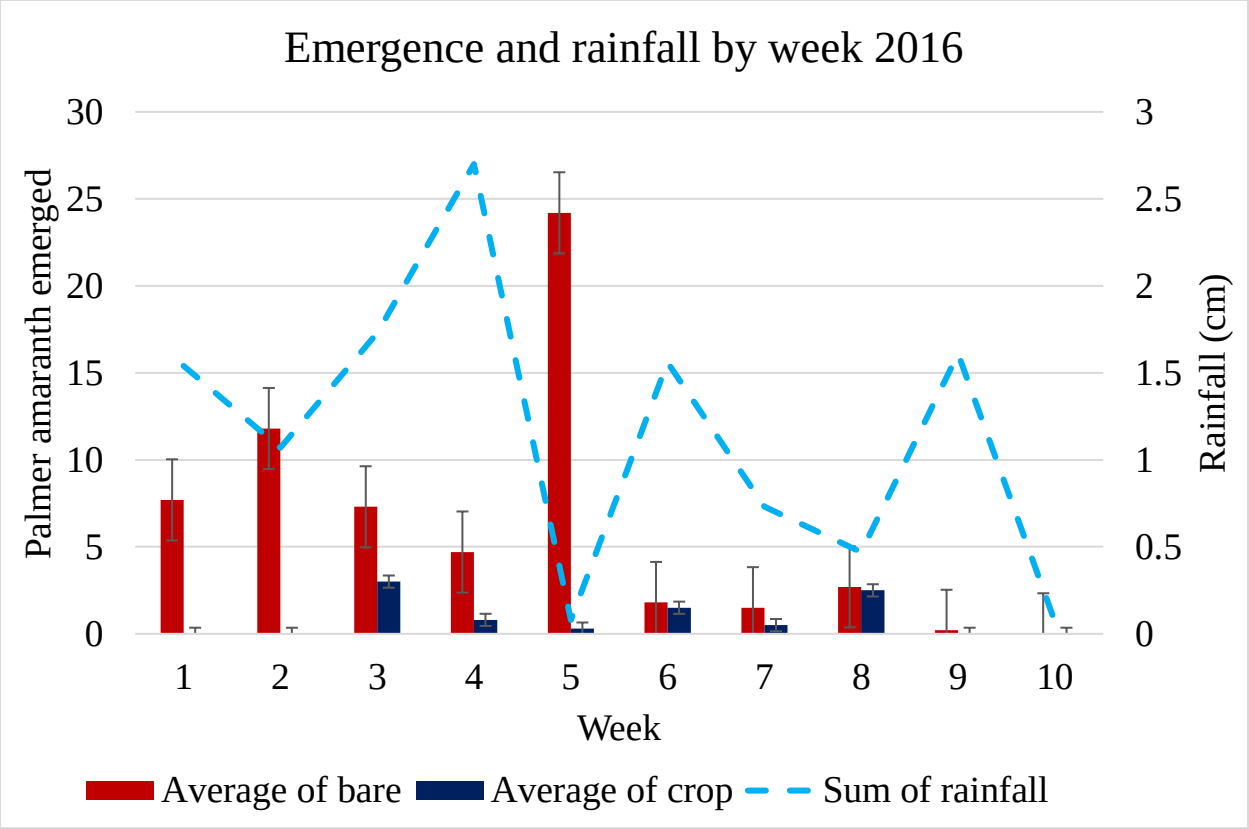
Kankakee 2015 week 1 was recorded from May 26 to August 21st in 2015

Figure 2.2. Palmer amaranth emergence and rainfall summarized by week in Kankakee 2016.



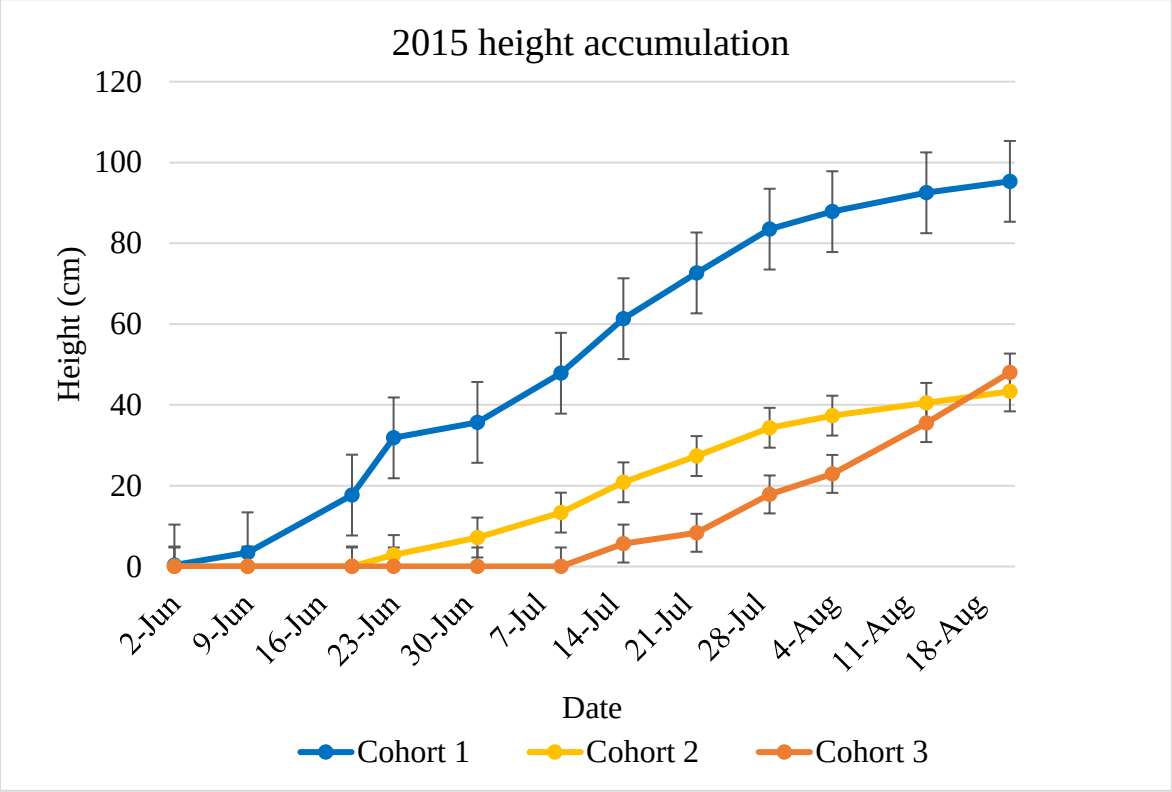
Kankakee 2016 week 1 was recorded from June 2 to August 26th

Figure 2.3. Palmer amaranth emergence and rainfall summarized by week in Urbana 2016.



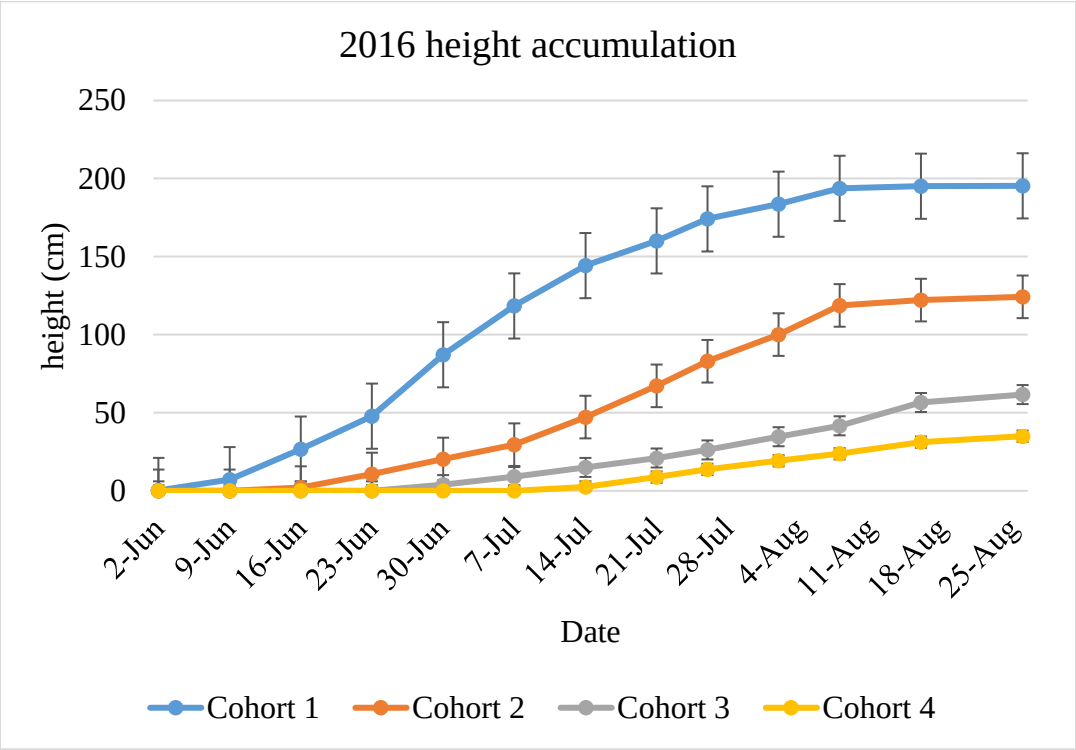
Week 1 in Urbana was recorded from June 2nd to August 23rd

Figure 2.4. Height summarized by week for six Kankakee (2015) Palmer amaranth under field conditions marked every third week starting on June 2nd, 2015.



Average dry biomass by cohort: 1, 9.1g; 2, 2.7g; 3, 3.4g

Figure 2.5. Height summarized by week for Palmer amaranth under 2016 field conditions marked every second week starting the first week in June.



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CHAPTER 3

FOLIAR HERBICIDE OPTIONS AND APPLICATION TIMING FOR PALMER AMARANTH MANAGEMENT IN ILLINOIS CROPPING SYSTEMS

3.1 Abstract

Palmer amaranth has been spreading from the southern United States into the Midwest, and concerns have been expressed about control options in Illinois cropping systems. Currently, Palmer amaranth has evolved resistance to herbicides from six site-of-action groups. The objectives of this study were to: 1) determine efficacy of foliar-applied herbicides from six site-of-action groups, and 2) determine optimal Palmer amaranth height for application of postemergence herbicides. Field and greenhouse experiments were conducted in 2015 and 2016 near Kankakee, Illinois and in 2016 in Urbana, Illinois. Postemergence herbicides were applied when Palmer amaranth plants were at three different heights: early-POST (EPOST) when plants were 5–8 cm, POST when plants were 9–13 cm, and late-POST (LPOST) when plants were 14–18 cm tall. In the field, chlorimuron (13 g ai ha^{-1}), imazethapyr (70 g ai ha^{-1}), and mesotrione (105 g ai ha^{-1}) controlled Palmer amaranth ≤ 73% regardless of application timing. Recovery of Palmer amaranth in 2016 was evident following field applications of glufosinate (594 g ai ha^{-1}), lactofen (218 g ai ha^{-1}), and fomesafen (347 g ai ha^{-1}). Herbicides applied LPOST did not achieve more than 83% control of Palmer amaranth 21 DAT. Under greenhouse conditions, fomesafen, 2,4-D, and glufosinate produced 100% Palmer amaranth mortality following all application timings. Palmer amaranth biomass was greatest with chlorimuron following the EPOST application timing and chlorimuron or imazethapyr following POST and LPOST application timings.

3.2 Introduction

Palmer amaranth (*Amaranthus palmeri*) is a dioecious, summer annual broadleaf species that originated in the Sonoran Desert (Sauer 1957; Ward et al. 2013). Palmer amaranth has a photosynthetic rate around 80 μmol m^{-2} s^{-1} and is capable of rapid biomass accumulation (Ehleringer 1983). Research examining the effects of genetics and environmental factors on Palmer amaranth revealed the extent Palmer amaranth damage in the Midwest would be limited only by seed dispersal (Davis et al 2015). The damage niche for Palmer amaranth is not reliant on the genotype of this weed, but rather the environment into which seed is dispersed (Davis et al. 2015). McDonald et al. (2009) hypothesized that increased temperatures would expand the damage niche of southern-originating weeds northward. Female Palmer amaranth plants produce a prolific amount of seed, often ranging between 200,000–600,000 seeds when the parent plant emerges from March to June (Ward et al. 2013). Palmer amaranth can overtake many crops with multiple germination events throughout a growing season (Sauer 1957). The rapid growth of Palmer amaranth can cause growers to miss the most effective size of Palmer amaranth for postemergence herbicide application, resulting in plant survival, crop yield loss, and increasing soil seed bank density.

Postemergence herbicides should be applied at the appropriate growth stage of Palmer amaranth to optimize control. Herbicides applied at an earlier growth stage have been shown to provide greater control of several broadleaf species (Hart et al. 1997), whereas delaying applications until plants are larger can decrease herbicide efficacy (Lee and Oliver 1982). Previous research has demonstrated a significant interaction between herbicide efficacy and application timing on Palmer amaranth control (Mayo et al. 1995; Rios et al. 2016).

Palmer amaranth and other *Amaranthus* species can be difficult to control with postemergence herbicides in conventional soybean. Systemic ALS-inhibiting herbicides provided acceptable control of plants larger than 10 cm (Mayo et al. 1995). Efficacy of PPO-inhibiting herbicides often decrease when herbicides are applied at later growth stages (Hager et al. 2003). Non-systemic herbicides often provide greater weed control when applied at early timings compared with a late stage (Grichar 1997; Jordan et al. 1993; Shaw et al. 1990).

Non-systemic herbicides, such as fomesafen and glufosinate, provided the greatest control when Palmer amaranth was 8 cm or less (Powell et al. 2014). When plant size increases, the rate of herbicide required for effective control also increases (Powell et al. 2014). Lactofen provided 99% control of Palmer amaranth plants, but control significantly decreased at later herbicide applications (Mayo et al. 1995). Variable control has been reported for *Amaranthus* species when using PPO herbicides in the field (Mayo et al. 1995) and control was reported as incomplete under heavy weed pressure (Hager et al. 2003).

Postemergence herbicides are widely used in modern agricultural practices, however data describing Palmer amaranth control with postemergence herbicides is lacking in Illinois cropping systems and requires evaluation. As Palmer amaranth height increases, attaining acceptable control becomes more challenging, therefore Palmer amaranth should be treated at early stages of growth (Klingaman et al. 1994). Currently in Illinois, Palmer amaranth has been documented resistant to herbicides with ALS, EPSPS, and PPO sites of action (Heap 2017). The first objective of this research was to determine the efficacy of herbicides with different sites of action for control of Palmer amaranth, while the second objective was to quantify the effects of plant size on herbicide efficacy.

3.3 Materials and Methods

3.3.1 Field design and implementation

Field experiments were established near Kankakee, Illinois in 2015 and 2016, and Urbana, Illinois in 2016. The soil at Kankakee was a Kankakee fine sandy loam (loamy-skeletal, mixed, superactive, mesic Typic Hapludolls) with a pH of 6.5 and organic matter of 2%. The soil type at Urbana was a Flanagan silt loam (fine, smectitic, mesic Aquic Argiudolls) with a pH of 6.5 and 4.9% organic matter. All plots were 3 meters wide and 7.6 meters long at Kankakee and 10 meters long at Urbana. The soil was tilled at the beginning of the season to control any existing vegetation. No crop was planted at any location. Plots were arranged in a randomized complete block design with three replication per treatment. All herbicides were applied with a backpack CO_2 sprayer equipped with XR8002[1] flat-fan spray nozzles spaced 51 cm apart on a 3 m boom. Spray volume was 187 L ha^{-1} and pressure was 276 kPa. Environmental conditions were recorded at each herbicide application timing. Selection of herbicides, applications rates, and additives was based on label recommendations and current Illinois production practices.

3.3.2 Herbicide application timing based on Palmer amaranth size under field conditions

Postemergence herbicides representing six site-of-action groups were selected for evaluation (Table 3.1). Each herbicide treatment was applied at the corresponding label-recommended rate and included any appropriate spray additive(s). Each treatment was applied to Palmer amaranth at the following plant heights: 5–8 cm (early postemergence (EPOST)), 9–13 cm (postemergence (POST)), and 14–18 cm (late postemergence (LPOST)). Applications were made when the majority of Palmer amaranth plants were within the listed height ranges.

Five Palmer amaranth plants at the specified height were marked in each plot (15 total per treatment) to quantify control and aboveground biomass accumulation after herbicide

treatment. Individual plants were also marked in non-treated plots. Individual plants were marked by placing a wooden garden stake to the immediate right of the weed. The area around the selected weed was cleared of other plants to ensure full spray interception by the marked plants. Mortality of marked Palmer amaranth plants was visually determined 7, 14, and 21 days after treatment (DAT). Plants were appraised as dead, likely to die, or alive. Plants were scored alive if they demonstrated actively growing tissue. At 21 DAT, marked plants were harvested for aboveground biomass, dried at 65°C for 7 days, and dry biomass recorded.

The effectiveness of each herbicide on a whole-plot basis also was visually determined 7, 14, and 21 DAT on a scale from 0 (no control) to 100 (complete control). The ratings were based on estimates of injury and biomass reduction when compared to non-treated plots.

3.3.3 Palmer amaranth population used for greenhouse experiments

Female inflorescences of Palmer amaranth from Kankakee were collected in fall 2015 when the seed was mature. Seed was cleaned and stratified using 10^{-4} M dilution of ethephon. The mixture was then shaken intermittently over two hours, after which the solution was removed and the seeds were rinsed with 0.95 mL distilled water for ten minutes. This rinse was repeated and then seeds were suspended in 0.1% agarose solution. Seeds not used immediately for planting were stored at 4°C.

3.3.4 Greenhouse plant culture

Following stratification, Palmer amaranth seed was placed in 12-cm by 12-cm flats filled with a commercial potting medium[2] of peat and perlite and covered with sifted soil. Emerged seedlings were transplanted at the second true leaf stage into 9 by 12 cm flats also filled with potting medium. Subsequently 4-cm tall, Palmer amaranth plants were transplanted to a 950 cm^3

pot containing a 3:1:1:1 mixture of potting mix: soil: peat: sand. A slow-release fertilizer[3] was added when needed.

Herbicides were applied to Palmer amaranth plants at the same plant heights indicated for field experiments. Treatments were applied using a compressed air research sprayer[4] calibrated to deliver 185 L ha^1 at 275 kPa. The experiment was arranged in a randomized block design with each treatment replicated four times and the experiment was repeated.

Mortality of treated Palmer amaranth plants was determined 7, 14, and 21 DAT as described previously. At 21 DAT, Palmer amaranth plants were harvested for aboveground biomass, dried at 65°C for 7 days and dry biomass recorded.

3.3.5 Statistical Analysis

Data were tested for the assumptions of ANOVA using the Shapiro-Wilk's test for normality and Levene's test for homogeneity of variance at α 0.05. Biomass data were log transformed [log10 (x+1)] (Gomez and Gomez 1984) to meet residual normality assumptions. Data were analyzed using the PROC MIXED procedure using the TYPE 3 model in SAS[5] 9.4 (SAS Institute Inc, Cary NC). Each year-location combination was considered an environment as suggested by Carmer et al. (1989). All possible main effects and interactions were tested. Fixed effects included herbicide site-of-action, treatment within the site-of-action group, and application timing. Random effects included environment and block nested within environment (field only) and all interactions containing either of these effects, or experimental run (greenhouse only). Mean estimates of plot control (field) and dried biomass (field and greenhouse) were separated with the use of the SAS macro *%pdmix800* (Saxton 1998). Means of Palmer amaranth application timing biomass were compared with the use of single-degree-of-freedom contrast statements.

3.4 Results and Discussion

3.4.1 Herbicide efficacy on Palmer amaranth under field conditions

Control of Palmer amaranth with chlorimuron and imazethapyr did not exceed 78% regardless of application timing (Table 3.2), and no difference was observed between these ALS-inhibiting herbicides. Tembotrione controlled Palmer amaranth more than mesotrione 7 and 14 days after the EPOST and POST application timings, but no differences in control were apparent by 21 days after these application timings nor at any evaluation of the LPOST application timing. No differences in Palmer amaranth control with lactofen and fomesafen were observed at any application timing, which is similar to the lack of difference in control between dicamba and 2,4-D. Additionally, control with the two PPO-inhibiting herbicides was greater than control of the two growth regulator herbicides only at the 7 DAT evaluation of the EPOST and LPOST application timings. The non-selective herbicides glyphosate and glufosinate controlled Palmer amaranth equally at all evaluation timings. Numerically, control values tended to decline between the first and last evaluation of each application timing, although control from dicamba and 2,4-D tended to trend upward at the latest evaluation timing of each application.

All treatments reduced biomass of marked Palmer amaranth plants compared with the nontreated control 21 days after each application timing (Table 3.3). A difference in biomass between chlorimuron and imazethapyr was apparent only following the EPOST application timing. Similar to control observation, tembotrione reduced Palmer amaranth biomass more than mesotrione following each application timing. Biomass was similar between lactofen and fomesafen for all application timings, and these PPO-inhibiting herbicides were consistently among those treatments that reduced biomass most regardless of application timing. Dicamba and 2,4-D reduced Palmer amaranth biomass similarly at each application timing, and no

differences in biomass were determined between glyphosate and glufosinate. Biomass data collected from plants marked prior to herbicides application generally support whole-plot control observations that the PPO-inhibiting and nonselective herbicides provided the greatest control and biomass reduction of Palmer amaranth. Plant mortality did not exceed 98% for any treatment. Numerically, mortality values were higher at the EPOST application timing with five treatments producing at least 82% mortality. Following the POST and LPOST application timings, mortality of marked plants did not exceed 74% and 50% respectively.

Contrast statements were generated to compare estimated differences and 95% confidence intervals in Palmer amaranth biomass by application timing (Table 3.4). Contrast statements comparing application timings at 21 DAT demonstrated that Palmer amaranth biomass was significantly less for EPOST than the POST or LPOST application timings.

3.4.2 Response of Palmer amaranth biomass and mortality to herbicide applications based on plant size under greenhouse conditions

Under greenhouse conditions, differences in Palmer amaranth biomass and mortality among treatments were less apparent compared to results from treatments applied under field conditions. Palmer amaranth biomass was greatest with chlorimuron following the EPOST application timing and chlorimuron or imazethapyr following POST and LPOST application timings (Table 3.5). No other differences in biomass among treatments applied EPOST or POST were observed. Mortality was zero following treatment with chlorimuron regardless of application timing. Subsequent molecular screening revealed this population contains an altered coding sequence for the ALS gene that is known to confer resistance to sulfonylurea and imidazolinone herbicides (data not presented). There were no differences in mortality between the two herbicides from each site-of-action group after any application timing, except between

ALS inhibitors and the nonselective herbicides at the EPOST and POST application timings, respectively. Fomesafen, 2,4-D, and glufosinate produced 100% Palmer amaranth mortality following all application timings. All treated biomass was significantly lower than the non-treated Palmer amaranth plants at each timing.

Previous studies have found ALS inhibitor herbicides to have incomplete Palmer amaranth control (Horak and Peterson 1995; Mayo et al. 1995; Sprague 1997). Control of Palmer amaranth with chlorimuron and imazethapyr did not exceed 78% and differences in biomass were only noted under greenhouse conditions after the EPOST application timing. Palmer amaranth populations have also been documented to be 4- to 23-fold resistant to HPPD-inhibiting herbicides (Jhala et al. 2014). Tembotrione controlled Palmer amaranth greater than mesotrione. Applications of glufosinate and glyphosate efficacy was reduced once Palmer amaranth size increases similar to findings by Rios et al. (2016), however no differences between herbicides at each application timing were noted. PPO inhibiting herbicides have reduced efficacy under field conditions as the plant size increased over 10 cm (Hager et al. 2003; Grichar 2007; Morechetti et al. 2012). POST and LPOST herbicide timings had control values that tended to decline between the first and last evaluation timings. Similar to research by Morechetti et al. (2012), plants did not completely die and regrowth was noted weeks after herbicide application. Results concur with findings by Klingaman et al. (1992), that Palmer amaranth should be controlled with herbicide at the early postemergence stage before this weed becomes too large for control. Post emergence herbicide treatments should be applied at the EPOST application height to ensure the greatest level of Palmer amaranth control.

3.5 Source of Materials

[1]TeeJet 80025EVS. TeeJet Technologies, P.O. Box 7900, Wheaton, IL 60187.

[2]LC1. Sun Gro Horticulture, 15831 N.E. 8th Street, Bellevue, WA 98008.

[3]Osmocote 13-13-13 slow release fertilizer. The Scotts Company, 14111 Scottslawn Rd., Marysville, OH 43041.

[4]Generation III Research Sprayer. DeVries Manufacturing, 28081 870th Ave., Hollandale, MN 56045.

[5]Statistical Analysis Software (SAS) 9.4. SAS Institute, Inc., 100 SAS Campus Drive, Cary, NC 27513.

3.6 Tables

Table 3.1 Herbicides and rates used for all postemergence applications in Kankakee and Urbana

herbicide[a]	Trade name	site of action[b]	rate[c]	manufacturer
			g ha^{-1}	
chlorimuron	Classic	ALS	13	DuPont Crop Protection, Wilmington, DE; http://www.dupont.com
imazethapyr	Pursuit	ALS	70	BASF Corporation Agricultural Products, Research Triangle Park, NC; http://www.agro.basf.com
mesotrione	Callisto	HPPD	105	Syngenta Crop Protection, Greensboro, NC; http://www.syngentacropprotection.com
tembotrione	Laudis	HPPD	91	Bayer CropScience, Research Triangle Park, NC: http://www.bayercropscience.com
dicamba	Clarity	Synthetic auxin	560	BASF Corporation Agricultural Products, Research Triangle Park, NC; http://www.agro.basf.com
2,4-D	Weedar 64	Synthetic auxin	1120	Nufarm Inc. Alsip, IL; http://www.nufarm.com
fomesafen	Flexstar	PPO	347	Syngenta Crop Protection, Greensboro, NC; http://syngentacropprotection.com
lactofen	Cobra	PPO	218	Valent U.S.A. Corporation, Walnut Creek, CA; http://www.valent.com
glyphosate	RoundUp Powermax	EPSP	1260	Monsanto Company, St. Louis, MO; http://www.monsanto.com
glufosinate	Liberty	GS	594	Bayer CropScience, Research Triangle Park, NC: http://www.bayercropscience.com

[a] Herbicide treatments, excluding the synthetic auxins, included a nonionic surfactant at 2.5% (v/v); herbicide treatments containing PPO, HPPD, and ALS inhibitors included a crop oil concentrate (COC 1% v/v); Synthetic auxin herbicide treatments included a nonionic surfactant at 0.25% (v/v).

[b] Abbreviations for site of action; GS, glutamine synthetase; EPSP, enolpyruvylshikimate-3-phosphate synthase; PPO, protoporphyrinogen oxidase; HPPD, hydroxyphenylpyruvate dioxygenase; ALS, acetolactate synthase.

[c] Rates expressed at active ingredient for all herbicides, excluding glyphosate, dicamba, and 2,4-D, which are expressed as acid equivalent.

Table 3.2. Visual estimates of whole-plot control of Palmer amaranth under field conditions by timing. Visual estimates of control sharing the same letter within a column are not significantly different at α=0.05 (separated by the SAS macro %pdmix800).

		application timing[a]								
		EPOST			POST			LPOST		
		----------DAT[b]----------								
herbicide	rate[c]	7	14	21	7	14	21	7	14	21
	g ha^{-1}	----------% control----------								
untreated	0	0 e	0 e	0 d	0 e	0 e	0 d	0 e	0 e	0 d
chlorimuron	13	78 bcd	71 bcd	65 c	70 cd	71 bcd	62 c	67 cd	62 d	58 c
imazethapyr	70	73 cd	69 cd	68 bc	65 d	69 cd	61 c	67 cd	67 bcd	59 bc
mesotrione	105	65 d	66 d	66 c	64 d	66 d	65 bc	60 d	63 cd	65 abc
tembotrione	91	87 ab	81 ab	87 abc	81 abc	81 ab	83 ab	68 cd	68 bcd	74 abc
dicamba	560	79 bc	80 abc	95 a	78 bcd	80 abc	86 a	76 bc	82 ab	80 ab
2,4-D	1120	81 bc	83 a	91 a	79 abc	83 a	87 a	77 bc	79 abcd	84 a
fomesafen	347	97 a	87 a	88 ab	96 a	87 a	83 ab	93 a	81 ab	79 abc
lactofen	218	96 a	87 a	89 ab	92 a	87 a	83 ab	93 a	80 abc	82 a
glyphosate	1260	86 abc	81 ab	76 abc	87 ab	81 ab	77 abc	88 ab	84 ab	77 abc
glufosinate	594	97 a	88 a	85 abc	92 a	88 a	77 abc	91 ab	88 a	77 abc

[a] Early postemergence, EPOST; postemergence, POST; late postemergence, LPOST.

[b] Days after treatment, DAT

[c] Rates expressed at active ingredient for all herbicides, excluding glyphosate, dicamba, and 2,4-D, which are expressed as acid equivalent.

Table 3.3. Mean separation of marked Palmer amaranth biomass and mortality ratings by timing at 21 DAT under field conditions. Means sharing the same letter within a column are not significantly different at α=0.05 (separated by the SAS macro %pdmix800).

herbicide	rate[a]	EPOST		POST		LPOST	
	g ai (ae) ha^{-1}	biomass	mortality	biomass	mortality	biomass	mortality
		(g)	(%)	(g)	(%)	(g)	(%)
untreated	0	3.18 a	-	5.98 a	-	5.48 a	-
chlorimuron	13	0.95 b	26.7c	1.44 b	19.7 b	1.60 b	6.7 d
imazethapyr	70	0.46 cde	33.3 c	4.81 b	15.7 b	1.65 b	11 cd
mesotrione	105	0.72 bc	35.7 bc	1.51 b	33.3 ab	1.59 b	15.7 cd
tembotrione	91	0.18 e	88.7 a	0.51 c	48.7 ab	0.41 d	44.7 bcd
lactofen	218	0.17 e	95.7 a	0.70 c	64.3 ab	0.71 cd	88 a
fomesafen	347	0.27 de	80 ab	0.54 c	42.3 ab	1.03 bcd	46.7 abcd
2,4-D	1120	0.31 de	82.3 a	0.76 c	46.7 ab	1.17 bc	44.3 bcd
dicamba	560	0.59 bcd	84.7 a	0.77 c	62 ab	1.27 bc	46.3 abcd
glyphosate	1260	0.76 bc	66.7 abc	0.80 c	80 a	1.52 b	80 ab
glufosinate	594	0.43 cde	97.7 a	0.62 c	73.3 a	1.02 bcd	49.7 abc

[a] Rates expressed at active ingredient for all herbicides, excluding glyphosate, dicamba, and 2,4-D, which are expressed as acid equivalent.

Table 3.4. Contrasts of Palmer amaranth biomass by treatment timing under field conditions at 21 DAT.

timing[a]	estimated difference[b]	95% confidence interval	standard error
EPOST vs POST	0.607	-1.419 – -0.001*	0.099
POST vs LPOST	0.205	-2.839 – -0.0002*	0.199
LPOST vs EPOST	0.812	-2.129 – -0.0003*	0.298

[a] Timings: early post emergence, EPOST; post emergence, POST; late post emergence, LPOST.

[b] Estimated difference of biomass means

* Significant at α=0.05.

Table 3.5. Mean separation of Palmer amaranth biomass at 21 DAT under greenhouse conditions. Means sharing the same letter within a column are not significantly different at α=0.05(separated by the SAS macro %pdmix800).

		EPOST		POST		LPOST	
herbicide	rate[a]	biomass	mortality	biomass	mortality	biomass	mortality
	g ai (ae) ha^{-1}	(g)	(%)	(g)	(%)	(g)	(%)
nontreated	-	3.04 a	-	4.03 a	-	3.54 a	-
chlorimuron	13	1.89 b	0 c	2.47 b	0 c	3.08 b	0 b
imazethapyr	70	0.90 c	75 ab	1.90 b	0 c	3.19 b	68 ab
mesotrione	105	0.60 cd	50 b	0.73 c	50 b	1.54 c	0 b
tembotrione	91	0.30 d	88 ab	0.55 c	63 ab	1.35 cd	75 ab
lactofen	218	0.22 d	100 a	0.82 c	63 ab	1.96 c	63 ab
fomesafen	347	0.20 d	100 a	0.46 c	100 a	0.67 e	100 a
2,4-D	1120	0.30 d	100 a	0.53 c	100 a	0.78 de	100 a
dicamba	560	0.36 d	100 a	0.33 c	100 a	0.89 de	75 ab
glyphosate	1260	0.29 d	100 a	0.50 c	50 b	0.75 de	75 ab
glufosinate	594	0.18 d	100 a	0.45 c	100 a	0.57 e	100 a

[a] Rates expressed as active ingredient for all herbicides, excluding glyphosate, dicamba, and 2,4-D, which are expressed as acid equivalent.

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CHAPTER 4

DOSE-RESPONSE OF PALMER AMARANTH TO SYNTHETIC AUXIN HERBICIDES

4.1 Abstract

The objectives of this study were to determine the effective herbicide rate of dicamba and 2,4-D to control Palmer amaranth in Illinois. Field dose-response experiments were conducted in 2015 and 2016 near Kankakee, Illinois and in 2016 in Urbana, Illinois. Dicamba or 2,4-D was applied to 10–12 cm tall Palmer amaranth plants at increasing rates equally spaced along a base 3.16 logarithmic scale. Greenhouse experiments compared the dose-response of Palmer amaranth, tall waterhemp and smooth pigweed to 2,4-D and dicamba applied when plants had 8-9 true leaves. Dicamba and 2,4-D rates ranged from 82 to 2242 g ae ha^{-1}, resulting in nine rates for each herbicide. By 21 DAT under field conditions, control of Palmer amaranth did not exceed 86% with 560 g dicamba ha^{-1}, however control exceeded 80% with 2,4-D at doses greater than or equal to 532 g ha^{-1}. Estimated doses for 50–90% of Palmer amaranth biomass reduction was at rates at 496–1332 g ae ha^{-1} of dicamba and 254–536 g ae ha^{-1} of 2,4-D. Under greenhouse conditions, doses resulting in visual assessment of 50% Palmer amaranth mortality were 404 g dicamba ha^{-1} and 388 g 2,4-D ha^{-1}, while 90% mortality occurred near rates of 885 g dicamba ha^{-1} and 1507 g 2,4-D ha^{-1}. Results indicate that rates for 90% mortality of Palmer amaranth exceeds the maximum in-crop application rate of dicamba when applied to dicamba-resistant soybean.

4.2 Introduction

Palmer amaranth (*Amaranthus palmeri*) is a dioecious, summer annual, broadleaf species indigenous to the Sonoran Desert (Sauer 1957; Ward et al. 2013). Palmer amaranth has a photosynthetic rate of approximately 80 μmol m^{-2} s^{-1}, which contributes to the plant's ability to

rapidly accumulate biomass (Ehleringer 1983). Research examining genetics and environmental factors of Palmer amaranth distribution determined the extent of Palmer amaranth damage in the Midwest would be limited only by seed dispersal (Davis et al 2015). McDonald et al. (2009) hypothesized that increased temperatures would expand the damage niche of southern-originating weed species northward. Female Palmer amaranth plants produce a prolific amount of seed, often ranging between 200,000–600,000 seeds when the plant emerges from March to June (Ward et al. 2013). Palmer amaranth can overtake many agronomic crops with multiple germination events within a growing season (Sauer 1957).

Natural auxin (Indole-3-acetic acid, IAA) causes phototropic movements in plants. The hormone is found in actively growing meristems and is transported through the phloem parenchyma cells. A receptor for auxin was discovered in 2005 and identified as the F-box protein transport inhibitor response 1 (TIR1) (Guilfoyle 2007). Auxin/IAA bind to the TIR1 receptor and either represses or activates gene expression. Gene expression activates most processes regulated by this plant hormone (Guilfoyle 2007).

Herbicides that mimic IAA are commonly referred to as synthetic auxin herbicides or plant growth regulators (PGRs). These herbicides are translocated in the xylem and the phloem and cause plant death due to uncontrolled, unregulated auxin activity in the plant. Auxin has a promiscuous binding site on the receptor and in turn acts as “molecular glue” that strengthens the substrate binding to the receptor (Guilfoyle 2007), rendering the plant unable to regulate auxin production. The increase of auxin promotes an increase in ethylene production, causing leaf senescence and excess production of abscisic acid. The overproduction of these hormones, along with radical oxygen species (ROS), cause rapid leaf senescence followed by eventual plant death. There are multiple sites of action due to the plant having many auxin receptors in cells.

Synthetic auxin herbicides cannot be metabolized, compartmentalized, or regulated in sensitive dicot species (Kelley and Riechers 2007).

PGR herbicides injure primarily sensitive dicot species rather than monocot species. One possible reason for this selectivity is differential metabolism. Grasses rapidly convert synthetic auxins to inactive metabolites with irreversible ring hydroxylation (Kelley and Riechers 2007). Sensitive dicots convert the synthetic auxin to amino acid conjugates, which can be converted back to the active auxin, so an active pool of herbicide remains (Kelley and Riechers 2007).

Auxin herbicides have long been used to control broadleaf species in grain crops such as corn (*Zea may* L.), wheat (*Triticum aestivum* L.) and grain sorghum [*Sorghum bicolor* (L.) Moench] (Mithila et al. 2011). Currently, 26 broadleaf species have documented resistance to synthetic auxin herbicides (Heap 2017). An *Amaranthus* species, tall waterhemp (*Amaranthus tuberculatus*), has been documented to be 10-fold more resistant to 2,4-D than the susceptible biotype (Bernards et al. 2012). Previous research evaluating broadleaf weed management in dicamba-resistant soybean reported only 60% of Palmer amaranth control when dicamba was applied preemergence at 113.4 g ae acre^{-1} (Johnson et al. 2010). 2,4-D applied at 230-1060 g ae ha^{-1} and dicamba applied at 280-1120 g ae ha^{-1} to 13–20-cm tall Palmer amaranth plants provided between 68 – 80% and 59 – 83% control, respectively (Merchant et al. 2013). The baseline sensitivity of a Nebraska Palmer amaranth population was reported to be below the recommended field rate for both dicamba (560 g ha^{-1}) and 2,4-D (800 g ha^{-1}) (Crespo et al. 2016). General field use rates of auxin herbicides in Illinois are 560 g ha^{-1} dicamba and 1120 g ha^{-1} 2, 4-D.

Concerns exist about the extent of damage Palmer amaranth could inflict upon Illinois agronomic crops, and what herbicide options are effective for control and suppression of this

weed. Currently in Illinois, Palmer amaranth has been documented to be resistant to herbicides from acetolactate synthethase (ALS), 5-enolpyruvyl-shikimate-3-phosphate (EPSP), and protoporphyrinogen oxidase (PPO) site-of-action inhibitors (Heap 2017). With new soybean varieties with resistance to dicamba or 2,4-D entering the market, synthetic auxin herbicides likely will be more extensively used in crop management programs. The objective of this study was to determine the appropriate use rate of synthetic auxin herbicides to control Palmer amaranth in Illinois. Additionally, greenhouse experiments were conducted to compare and quantify the response of three *Amaranthus* species, (*A. palmeri*, *tuberculatus*, *hybridus*) to dicamba and 2,4-D.

4.3 Materials and Methods

4.3.1 Field design and implementation

Field experiments were established near Kankakee, IL in 2015 and 2016 and Urbana, IL in 2016. The soil at Kankakee was a Kankakee fine sandy loam (loamy-skeletal, mixed, superactive, mesic Typic Hapludolls) with a pH of 6.5 and organic matter of 2%. The soil type at Urbana was a Flanagan silt loam (fine, smectitic, mesic Aquic Argiudolls) with a pH of 6.5 and 4.9% organic matter. All plots were 3 meters wide and 7.6 meters long at Kankakee and 10 meters in long at Urbana. The soil was tilled at the beginning of the season to control any existing vegetation. No soybean crop was planted at any location due to the regulated status of the dicamba-resistant trait technology. Plots were arranged in a randomized complete block design with each treatment replicated three times. All herbicides were applied with a pressurized CO_2 backpack sprayer equipped with XR8002[1] flat-fan spray tips spaced 51 cm apart on a 3 m boom. Spray volume was 187 L ha^{-1} and pressure was 276 kPa. Environmental conditions were recorded at each herbicide application.

4.3.2 Dose response of dicamba and 2,4-D for Palmer amaranth control under field conditions

Dicamba or 2,4-D was applied to 10–12 cm tall Palmer amaranth plants at increasing rates equally spaced along a base 3.16 logarithmic scale. Dicamba and 2,4-D rates ranged from 82 to 2242 g ae ha^{-1}, resulting in nine rates for each herbicide. Spray additives were included with each herbicide based on label recommendations.

Visual estimates of percent Palmer amaranth control were recorded 7, 14, and 21 DAT on a scale of 0 (no control) to 100 (complete control). In addition to visual estimates, five uniformly sized Palmer amaranth plants per plot (15 per treatment) were selected prior to treatment to quantify aboveground biomass accumulation following herbicide application. These uniformly sized Palmer amaranth plants (10–12 cm) were marked by placing a wooden stake near each plant prior to herbicide application. All other Palmer amaranth plants within a 15-cm diameter of each marked plant were carefully removed to ensure full spray interception by the marked plants. Each year, marked plants were scored dead, likely to die, or alive 21 DAT. To be scored alive, plants needed to exhibit new, unaffected tissue. The aboveground portion of all marked plants was harvested 21 DAT and placed in a dryer for seven days at 65°C, after which dried biomass was recorded.

4.3.3 Dose response of dicamba and 2,4-D for Palmer amaranth, waterhemp, and smooth pigweed under greenhouse conditions

Inflorescences of female Palmer amaranth plants were collected from Kankakee in fall 2015 when seed was mature. Seed from herbicide-sensitive biotypes of waterhemp (*Amaranthus tuberculatus*) and smooth pigweed (*Amaranthus retroflexus*) were obtained for comparison. Palmer amaranth, waterhemp, and smooth pigweed seed was cleaned and treated separately

using a 10^{-4} M dilution of ethephon. The mixture was then shaken intermittently over two hours, after which the solution was removed and the seeds were rinsed with 0.95 mL distilled water for ten minutes. This rinse was repeated and then seeds were suspended in 0.1% agarose solution. Seeds not used immediately for planting were stored at 4°C. Following treatment, seeds of each species were planted in 12-cm by 12-cm flats filled with a commercial potting medium[2] of peat and perlite and covered with sifted soil. Emerged seedlings were transplanted at the second true leaf stage into 9 by 12 cm flats also filled with potting medium. Subsequently, 4-cm tall plants were transplanted to a 950 cm^3 pot containing a 3:1:1:1 mixture of potting mix: soil: peat: sand; a slow-release fertilizer[3] was added when needed.

Dicamba or 2,4-D was applied to plants 10–12 cm tall or having 8–9 true leaves using a compressed air research sprayer[4] calibrated to deliver 185 L ha^1 at 276 kPa of pressure. Application rates were identical to those applied in the field experiment. The greenhouse experiment was conducted twice and each treatment was replicated five or six times per run. Following application, plants were placed on greenhouse benches in a randomized complete block design. Mortality of treated Palmer amaranth plants was determined 21 DAT. Plants were appraised as dead, likely to die, or alive. Plants were scored alive if they demonstrated new, actively growing tissue. At 21 DAT, the Palmer amaranth plants were harvested for aboveground biomass, samples were dried at 65°C for 7 days and biomass was recorded.

4.3.4 Statistical Analysis

The dried biomass data of all plants (field and greenhouse) within each treatment were averaged and converted to a percentage of the untreated control. Mortality ratings were calculated on the amount of plants visually assed as deceased at 21 DAT as a percentage of total plants per treatment. Data were evaluated with PROC Mixed in SAS[5] 9.4 (SAS Institute Inc,

Cary NC). Each year-location combination was considered an environment as suggested by Carmer et al. (1989). Fixed effects included herbicide treatment and rate, while random effects included environment and block within environment. All possible main effects and interactions were tested. Visual estimates mean separations were performed using Fischer's protected LSD at $P \leq 0.05$.

Data were analyzed in R software using a non-linear regression model with the dose-response curve package (R statistical software, R Foundation for Statistical Computing, Vienna, Austria) (Knezevic et al. 2007). The dose-response model was constructed using the equation

$$y = c + \frac{d - c}{1 - \exp[b(\log(x) - \log(GR_{50}))]}$$

The four parameter log-logistic is described as follows: b is the slope of the curve, c is the lower limit, d is the upper limit, and GR_{50} is a 50% reduction in biomass or 50% of plants visually estimated as deceased.

4.4 Results and Discussion

4.4.1 Response of Palmer amaranth to dicamba and 2,4-D under field conditions

Visual estimates of Palmer amaranth control, biomass, and mortality data were pooled across all environments. Both dicamba and 2,4-D caused characteristic injury (epinasty, leaf strapping, stunting, etc.) on treated plants within a few days after application. Injury tended to be greater at higher application doses. By 21 DAT, 496 g dicamba ha^{-1} and 254 g 2,4-D ha^{-1} reduced Palmer amaranth dry biomass by 50% (Table 4.1). The two lowest doses of dicamba increased biomass of treated plants to greater than the non-treated control plants (Figure 4.1), but this was not observed with the two lowest doses of 2,4-D. A 90% reduction in Palmer amaranth biomass required estimated doses of 1332 g ha^{-1} and 536 g ha^{-1} dicamba and 2,4-D, respectively. The estimated dose of dicamba to reduce Palmer amaranth biomass 90% is greater than two times the

maximum in-crop application dose of dicamba allowed by label when applied to dicamba-resistant soybean varieties, while the estimated dose of 2,4-D to reduce Palmer amaranth biomass 90% falls within the in-crop application dose labeled for application in 2,4-D resistant soybean varieties.

Control of Palmer amaranth did not exceed 86% with 560 g ha^{-1} dicamba (Table 4.2). This represents the maximum in-crop application dose of dicamba when applied to dicamba-resistant soybean varieties. Control exceeded 90% only with the three highest rates of dicamba doses 14 DAT. As described previously, 1332 g ha^{-1} dicamba was the estimated dose required to reduce Palmer amaranth biomass 90%, which closely agrees with the visual estimates of Palmer amaranth control. By 21 DAT, Palmer amaranth control exceed 80% with 2,4-D at doses of 532 g ha^{-1} or greater. Control was 87–90% with 781–1147 g 2,4-D ha^{-1}, a dose range that encompasses the maximum allowable in-crop application dose (1060 g ha^{-1}) for 2,4-D when applied to 2,4-D resistant soybean varieties (Table 4.2). A higher dose of 2,4-D was required to control Palmer amaranth 90% compared with the estimated dose required to reduce Palmer amaranth biomass 90%. While estimates of doses required for 90% biomass reduction and Palmer amaranth control were somewhat similar, no such similarity exists for plant mortality. Plant mortality of 90% or greater was only achieved at the highest (2242 g ha^{-1}) dose of each herbicide. Palmer amaranth mortality was only 57% with dicamba at the labeled in-crop application dose of 560 g ha^{-1} and between 47–80% at the maximum labeled in-crop application dose of 2,4-D. A 90% reduction in Palmer amaranth mortality required estimated doses of 885 g ha^{-1} and 1507 g ha^{-1} dicamba and 2,4-D, respectively (Table 4.3 and Figure 4.2).

4.4.2 Response of Palmer amaranth to dicamba and 2,4-D under greenhouse conditions

Estimated doses of dicamba and 2,4-D required to reduce Palmer amaranth biomass 50% under greenhouse conditions were approximately half those calculated under field conditions. Doses of dicamba and 2,4-D required to reduce Palmer amaranth biomass 50% were 217 and 129 g ae ha^{-1} for dicamba and 2,4-D, respectively, (Table 4.4) compared with doses of 496 and 254 g ae ha^{-1} under field conditions. Similar to observations by Hausman (2016) the amount of control provided by plant growth regulators was greater under greenhouse conditions when compared to evaluation under field conditions. Estimated doses to reduce waterhemp biomass 50% were similar for dicamba and 2,4-D (231 and 252 g ae ha^{-1}, respectively), and also similar for smooth pigweed (145 g ae ha^{-1} dicamba and 140 g ae ha^{-1} 2,4-D). The estimated dose of dicamba to reduce biomass 90% for each species exceeded the maximum in-crop application rate of dicamba allowed by label when applied to dicamba-resistant soybean varieties, while the estimated dose of 2,4-D to reduce biomass of each species 90% was less than the maximum in-crop application dose labeled for application in 2,4-D-resistant soybean varieties. Mortality of Palmer amaranth, waterhemp, and smooth pigweed was less than 75% with 560 g ae ha^{-1} dicamba (Table 4.5), whereas mortality of Palmer amaranth, waterhemp, and smooth pigweed was 82% or greater with 823 g ae ha^{-1} 2,4-D. The estimated dose required for 90% mortality of each species ranged from 816–1233 and 436–901 g ae ha^{-1} dicamba and 2,4-D, respectively (Table 4.6).

4.4.3 Dose response of Palmer amaranth populations in Illinois

Synthetic auxin herbicide applications in Illinois need to be at the rate of 560 g dicamba ha^{-1} and 532 g 2,4-D ha^{-1}, or as required by label in order to provide adequate control of Palmer amaranth. Using rates lower than the lethal dose can lead to increased selection pressure for dicamba and 2,4-D resistance (Ashworth et al. 2016; Tehranchian et al. 2017). The majority of

resistance cases are caused by a single gene or a couple of dominant genes when the herbicide is applied at the field rate with high plant mortality (Preston and Mallory-Smith 2001). Plant mortality is achievable in the susceptible genotype, but selection pressure can result in polygenic resistance. Plants with these select few resistances or plants that were sprayed at an improper growth stage may survive and therefore cross pollinate thus evolving resistance. To be able to combat selection pressure for resistance, synthetic auxins should not be applied below the recommended field rate.

4.5 Source of Materials

[1]TeeJet 80025EVS. TeeJet Technologies, P.O. Box 7900, Wheaton, IL 60187.

[2]LC1. Sun Gro Horticulture, 15831 N.E. 8th Street, Bellevue, WA 98008.

[3]Osmocote 13-13-13 slow release fertilizer. The Scotts Company, 14111 Scottslawn Rd., Marysville, OH 43041.

[4]Generation III Research Sprayer. DeVries Manufacturing, 28081 870th Ave., Hollandale, MN 56045.

[5]Statistical Analysis Software (SAS) 9.4. SAS Institute, Inc., 100 SAS Campus Drive, Cary, NC 27513.

4.6 Tables and Figures

Table 4.1. Estimated doses and standard errors of dicamba and 2,4-D required to reduce Palmer amaranth biomass 10, 50, or 90% under field conditions. Biomass data generated from uniformly-sized (10–12 cm) Palmer amaranth plants (15 treatment^{-1}) marked prior to herbicide application and harvested 21 DAT.

	Dicamba		2.4-D	
	Estimate	SE[b]	Estimate	SE
	(g ha^{-1})		(g ha^{-1})	
ED:10[a]	185	156	121	118
ED:50	496	169	254	85
ED:90	1332	1142	536	474

[a] ED:10, estimated dose to reduce 10% biomass; ED:50, estimated dose to reduce 50% biomass; ED:90, estimated dose to reduce 90% biomass.

[b] Standard error

Table 4.2. Visual estimates of Palmer amaranth control and mortality with dicamba and 2,4-D under field conditions. means sharing the same letter within a column are not significantly different at α=0.05

Dicamba					2,4-D				
dose[b]	DAT[a]				dose	DAT			
g ae ha^{-1}	7	14	21		g ae ha^{-1}	7	14	21	
	% control[c]			% mortality		% control			% mortality
82	63 g	63 g	57 e	0 e	82	61 g	61 f	52 f	0 g
121	68 fg	68 fg	62 de	2 e	114	67 fg	65 f	63 e	0 g
177	71 fe	72 ef	59 e	2 e	168	71 ef	73 e	66 e	0 g
260	74 de	76 de	68 cde	21 de	247	75 de	78 de	75 d	14 f
382	78 cd	83cd	74 bcd	47 cd	363	78 cd	81 cd	78 d	40 e
560	82 bc	86 bc	79 abc	57 bc	532	79 cd	86 bc	81 cd	44 d
823	83 ab	93 ab	86 ab	67 bc	781	83 bc	90 ab	87 bc	47 c
1207	88 a	97 a	89 a	85 ab	1147	88 ab	94 a	90 ab	80 b
2242	88 a	98 a	89 a	97 a	2242	93 a	97 a	95 a	94 a

[a] DAT, days after treatment.

[b] All doses included nonionic surfactant at 0.25% v/v and ammonium sulfate at 2.5% v/v

[c] Plots were rated as a whole as a percentage of total Palmer amaranth controlled

Table 4.3. Estimated doses and standard errors of dicamba and 2,4-D required to result in 10, 50, or 90% Palmer amaranth mortality under field conditions. Mortality data generated from uniformly sized (10–12 cm) Palmer amaranth plants (15 treatment^{-1}) marked prior to herbicide application and assessed 21 DAT.

	dicamba		2,4-D	
	dose	SE[b]	dose	SE
	(g ha^{-1})		(g ha^{-1})	
ED:10[a]	185	46	100	69
ED:50	404	50	388	90
ED:90	885	247	1507	1165

[a] ED:10, estimated dose to kill 10% of the population; ED:50, estimated dose to kill 50% of the population; ED:90, estimated dose to kill 90% of the population.

[b] Standard error

Table 4.4. Estimate doses and standard errors of dicamba and 2,4-D required to reduce biomass of Palmer amaranth, waterhemp, and smooth pigweed 10, 50, or 90% under greenhouse conditions. Biomass data generated from uniformly-sized (10–12 cm or 8–9 true leaves) plants (11 treatment^{-1}) harvested 21 DAT.

	Dicamba		2,4-D	
Species ED[a]	Dose	SE	Dose	SE
	g ae ha^{-1}		g ae ha-1	
PA:10	144	63	22	54
PA:50	217	28	129	136
PA:90	326	108	395	123
WH:10	67	58	86	130
WH:50	231	80	252	121
WH:90	788	299	501	246
Sm:10	24	55	37	84
Sm:50	145	143	140	127
Sm:90	889	637	327	101

[a] abbreviations; ED: estimated dose; PA:10, estimated dose to reduce 10% of Palmer amaranth biomass; PA:50, estimated dose to reduce 50% of Palmer amaranth biomass; PA:90, estimated dose to reduce 90% of Palmer amaranth biomass; WH:10, estimated dose to reduce 10% of waterhemp biomass; WH:50, estimated dose to reduce 50% of waterhemp biomass; WH:90, estimated dose to reduce 90% of waterhemp biomass; Sm:10, estimated dose to reduce 10% of smooth pigweed biomass; Sm:50, estimated dose to reduce 50% of smooth pigweed biomass; Sm:90, estimated dose to reduce 90% of smooth pigweed biomass.

Table 4.5. Mortality estimates of Palmer amaranth, waterhemp, and smooth pigweed at 21 DAT under greenhouse conditions to rates of dicamba and 2,4-D. Means sharing the same letter within a continuous treatment are not significantly different at α=0.05.

herbicide	rate	Palmer amaranth	waterhemp	smooth pigweed
	(g ae ha^{-1})		----% mortality----	
dicamba	82	9 d	0 c	0 e
	121	19 d	0 c	9 e
	177	25 cd	0 c	0 e
	260	17 d	9 bc	36 d
	382	55 bc	9 b	45 cd
	560	74 ab	27 b	64 bc
	823	90 ab	73 a	82 ab
	1207	100 a	82 a	100 a
	2242	100 a	91 a	100 a
2,4-D	82	0 d	20 c	0 c
	114	0 d	0 c	20 bc
	168	30 cd	29 bc	40 bc
	247	19 cd	20 c	54 ab
	363	40 bcd	49 abc	92 a
	532	65 abc	74 ab	100 a
	781	100 a	82 a	100 a
	1147	92 ab	82 a	100 a
	2242	100 a	100 a	100 a

Table 4.6. Estimated doses and standard errors of dicamba and 2,4-D required to result in 10, 50, or 90% mortality of Palmer amaranth, waterhemp, and smooth pigweed under greenhouse conditions. Mortality data generated from uniformly-sized (10–12 cm or 8–9 true leaves) plants (11 treatment^{-1}) evaluated 21 DAT.

	Dicamba		2,4-D	
Species ED[a]	Dose	SE[b]	Dose	SE
	g ae ha^{-1}		g ae ha^{-1}	
PA:10	225	44	199	82
PA:50	428	39	424	58
PA:90	816	184	902	295
WH:10	436	70	207	79
WH:50	648	46	407	58
WH:90	963	179	801	356
Sm:10	142	48	101	42
Sm:50	419	61	210	37
Sm:90	1233	420	436	95

[a] abbreviations; ED: estimated dose; PA:10, estimated dose to kill 10% of Palmer amaranth; PA:50, estimated dose to kill 50% of Palmer amaranth; PA:90, estimated dose to kill 90% of Palmer amaranth; WH:10, estimated dose to kill 10% of waterhemp; WH:50, estimated dose to kill 50% of waterhemp; WH:90, estimated dose to kill 90% of waterhemp; Sm:10, estimated dose to kill 10% of smooth pigweed; Sm:50, estimated dose to kill 50% of smooth pigweed; Sm:90, estimated dose to kill 90% of smooth pigweed.

[b] Standard error

Figure 4.1. Dose response curve of Palmer amaranth to dicamba and 2,4-D under field conditions. Lines are the predicted values for treated plant biomass as a percent of the non-treated control.

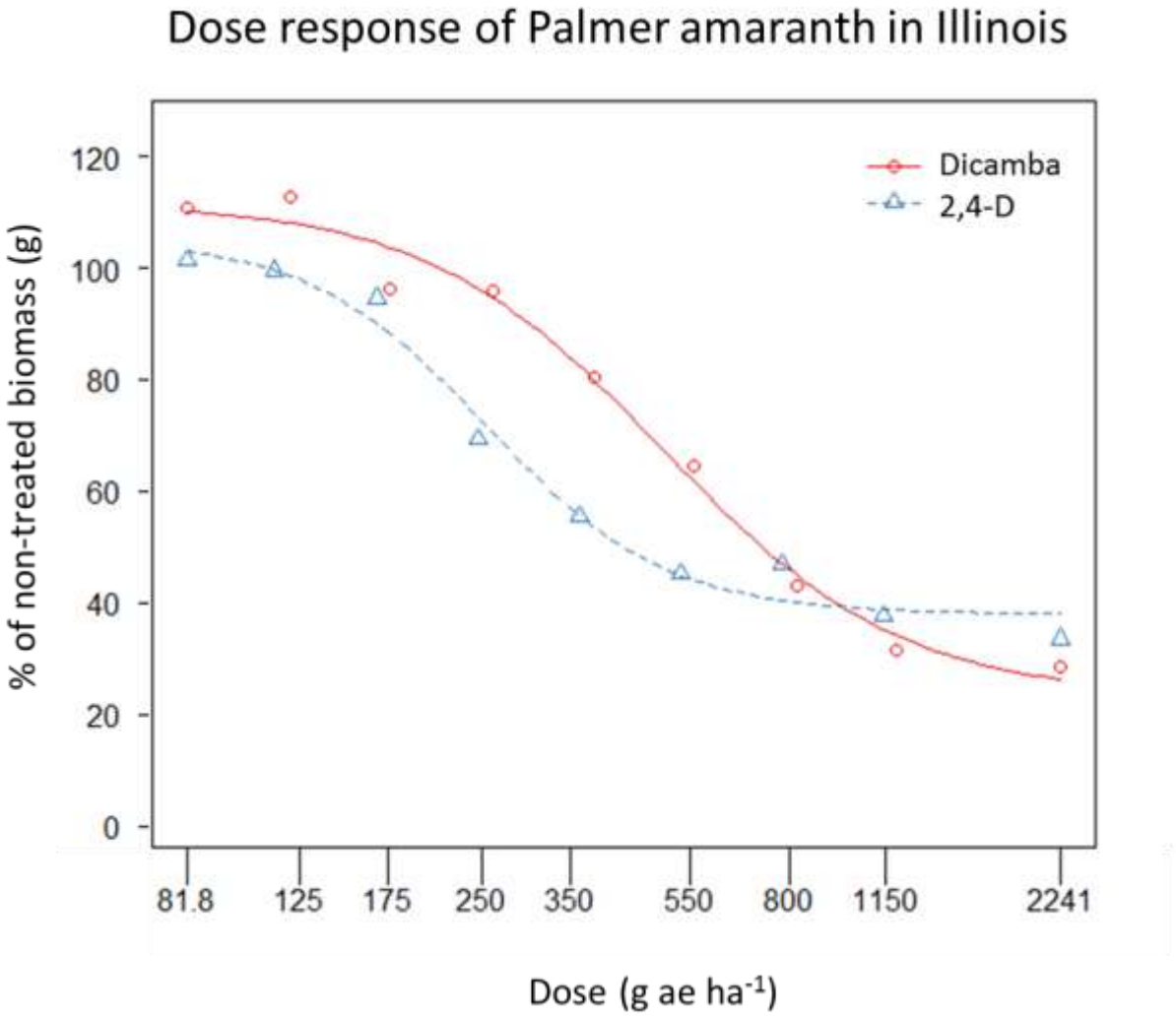


Figure 4.2. Estimated percent of Palmer amaranth survivors per treatment under field conditions. Lines are predicted values for percentage Palmer amaranth survival.

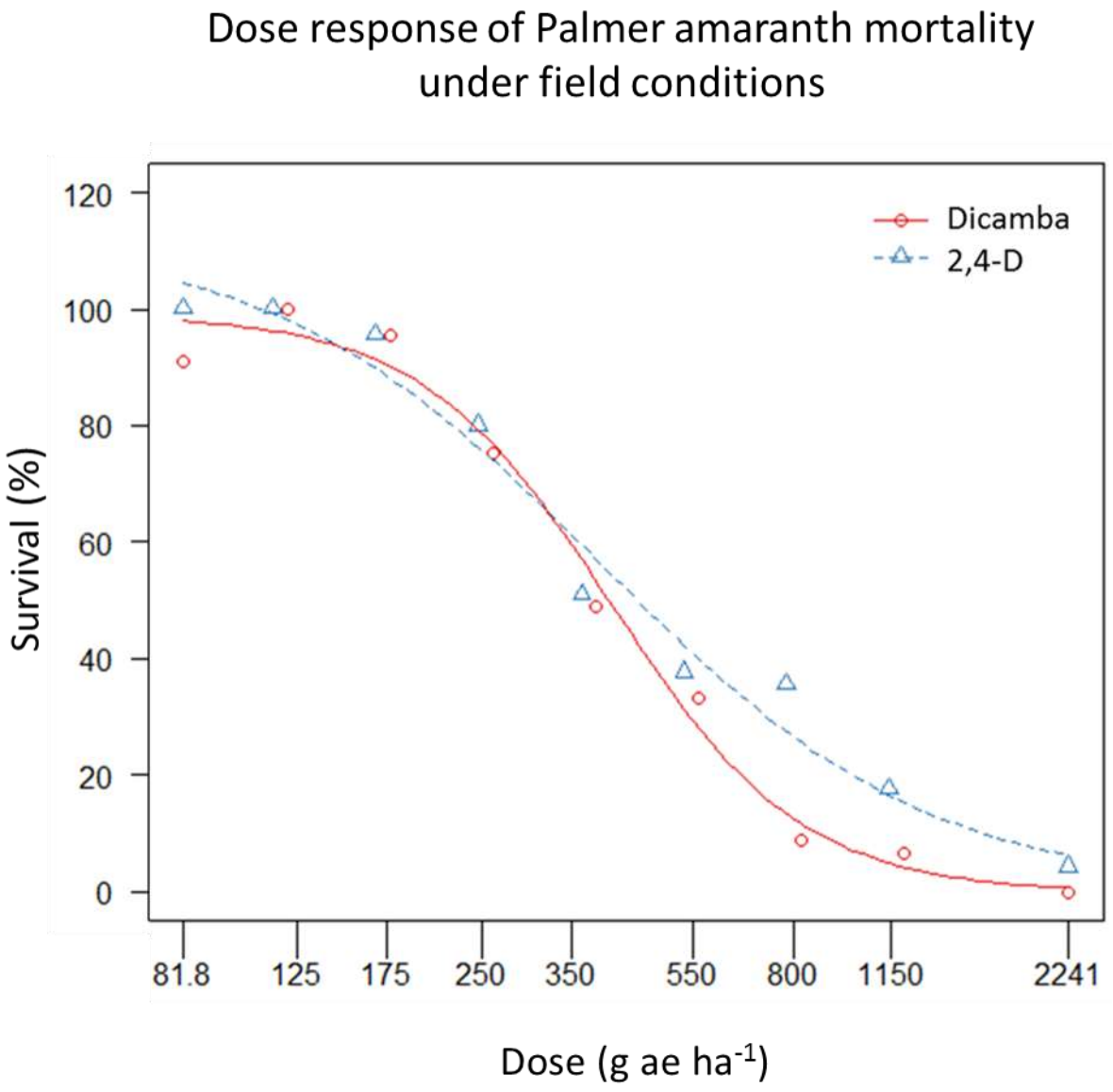


Figure 4.3. Percent of *Amaranthus* species survivors to dicamba doses under greenhouse conditions. Lines are predicted values for percentage survival.

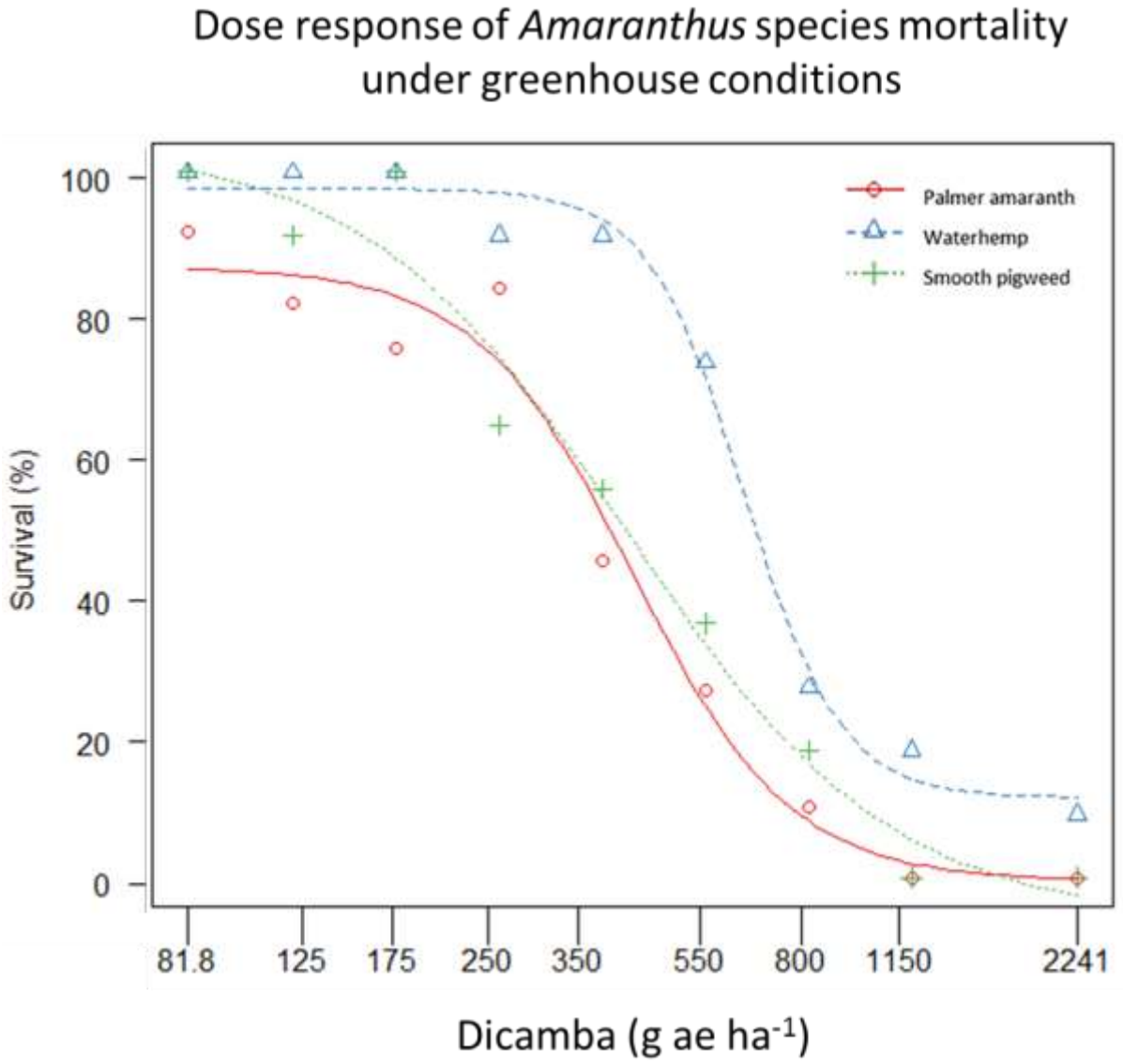
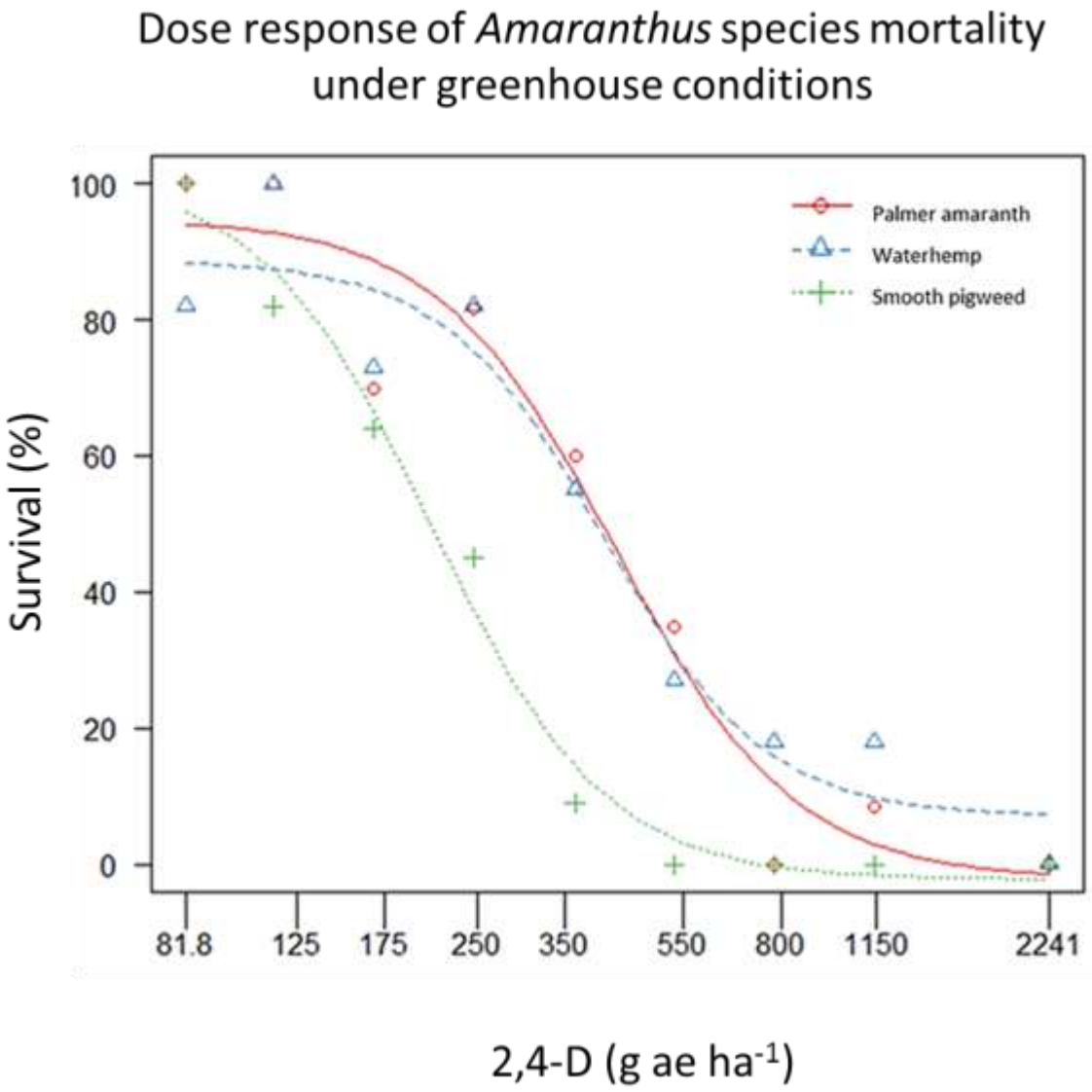


Figure 4.4. Percent of *Amaranthus* species survivors to 2,4-D doses under greenhouse conditions. Lines are predicted values for percentage survival.



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CHAPTER 5

RESIDUAL HERBICIDE COMBINATIONS FOR PALMER AMARANTH CONTROL IN GLUFOSINATE-RESISTANT SOYBEAN

5.1 Abstract

Palmer amaranth is known for rapid biomass accumulation and multiple germination events, making this weed difficult to control. Palmer amaranth resistance has been documented to herbicides from six site-of-action groups. In Illinois, however, resistance has been documented only to ALS, EPSP, and PPO inhibitors. Glufosinate-resistant soybean varieties used in combination with glufosinate is an option to combat this resistance, and therefore should be evaluated. The objectives of this study were to compare half and full rates of PRE-only applications (sulfentrazone + imazethapyr) PRE followed by (fb) early-POST (EPOST) applications (glufosinate or glufosinate + acetochlor), and PRE fb EPOST applications (glufosinate or glufosinate + acetochlor) fb POST applications (glufosinate or glufosinate + pyroxasulfone) for control of Palmer amaranth. Field experiments were conducted in 2015 and 2016 near Kankakee, Illinois. Quadrats were marked in each plot and Palmer amaranth emergence was recorded weekly. Above ground Palmer amaranth biomass was harvested from quadrats before each sequential herbicide application. End-of-season Palmer amaranth biomass from plots treated with PRE herbicides only was not significantly different than non-treated plot biomass. EPOST applications of glufosinate + acetochlor were significantly different in Palmer amaranth density than glufosinate only in 2016. EPOST fb POST applications were found to have greater control of Palmer amaranth biomass, regardless of additional residual activity herbicides.

5.2 Introduction

Palmer amaranth (*Amaranthus palmeri*) is a dioecious, summer annual, broadleaf species that originated in the southwestern United States (Sauer 1957). Palmer amaranth has a photosynthetic rate around 80 μmol m^{-2} s^{-1} and therefore can rapidly accumulate biomass (Ehleringer 1983). Research examining the influence of genetics and environmental factors on Palmer amaranth establishment determined the range of damage in the Midwest would be limited by seed dispersal rather than the environment (Davis et al 2015). McDonald et al. (2009) hypothesized that increased temperatures would expand the damage niche of southern-originating weed species northward, thus expanding the niche for favorable Palmer amaranth growth and development. The females produce a prolific amount of seed, often ranging between 200,000–600,000 seeds when the plant emerges from March to June (Ward et al. 2013). Palmer amaranth is able to overwhelm a field with multiple emergence events, predominantly after a soil disturbance or rainfall event (Sauer 1957; Ward et al. 2013). Not controlling early-emerging plants can lead to season long competition from Palmer amaranth in soybean, which caused a 17–63% yield reduction at 0.33–10 plants per m^{-1} (Klingaman and Oliver 1994), or 79% yield reduction at 8 plants per m^{-1} (Bensch 2003) when Palmer amaranth emerges shortly after soybeans. Currently in Illinois, Palmer amaranth has been documented to be resistant to herbicides from acetolactate synthethase (ALS), 5-enolpyruvyl-shikimate-3-phosphate (EPSP), and protoporphyrinogen oxidase (PPO) site of action inhibitors (Heap 2017). Options are still available to growers to control Palmer amaranth in Illinois include glutamine synthetase and very-long-chain fatty acid inhibitors since there is no documented Palmer amaranth resistance to herbicides from these site of action (Heap 2017).

One herbicide that can be used for controlling Palmer amaranth is glufosinate, which inhibits glutamine synthetase. Glufosinate is a structural analog of the amino acid glutamate, which is the substrate for the glutamine synthetase enzyme. The herbicide is a competitive inhibitor with glutamate for glutamine synthetase enzyme binding (Sauer et al. 1987). The herbicide inhibits the pathway responsible for assimilating ammonia and incorporating ammonia into a reduced, organic form such as the amino acids glutamine and glutamate. The buildup of ammonia is toxic to the plant, but the primary cause of death is due to the inhibition of transamination reactions in photorespiration (Sauer et al 1987; Wild et al. 1987). Without amino group donors, glyoxylate, glycolate, and phosphoglycolate accumulate and inhibit the enzyme RuBisCo and the carbon fixation in the Calvin cycle, which indirectly leads to inhibition of photosynthetic electron transport (Timm et al. 2016; Wild et al. 1987). Glufosinate is not translocated throughout the plant and herbicidal activity is enhanced by light.

There is no natural plant mechanism for glufosinate resistance, so crops are genetically modified for resistance using bacterial genes from *Streptomyces viridochromogenes* (Droge et al. 1992). These genes, referred to as the bar or PAT gene, codes for phosphinothricin acetyltransferase (PAT enzyme) which detoxifies glufosinate by acetylating the herbicide molecule (Devine et al. 1993). Glufosinate is a non-selective herbicide that has no residual activity in the soil (Shaner 2014). The addition of another herbicide with soil residual activity would be beneficial to control plants from multiple germination events in a growing season.

Herbicides that inhibit long-chain-fatty acid synthesis typically provide several weeks of residual control (Tanetani et al. 2009; Shaner 2014). Fatty acids are created via fatty acid synthase located in the plastid at the Acyl-carrier protein (ACP) and are then exported to the cytosol where elongation to very-long-chain fatty acids (VLCFA) occurs in the endoplasmic

reticulum (ER) (Schmalfuβ et al. 2000). The majority of VLCFA are located in the plasma membrane and when absent, the membrane loses stability and then becomes leaky, which results in plant death (Matthes and Böger 2002).

VLCFA herbicides, such as acetochlor and pyroxasulfone, inhibit VLCFA synthesis and are typically used preemergence (PRE) in corn and soybean (Fuerst 1987). Use rates are typically in low concentrations comparatively and weed resistance is rare (Götz and Böger 2004). These herbicides inhibit VLCFA elongases, which impedes cuticle synthesis and membranes, thus hindering the ability of a developing seedling shoot to emerge from the soil (Böger et al. 2000). Herbicide injury symptoms in grasses include improper unfurling of the leaf from the coleoptile (buggy-whipping) while in soybean injury is evident by slow emergence and crinkled or cupped leaves (Fuerst 1987; Yamaji et al. 2014). Acetochlor and pyroxasulfone provide control for several weeks (Shaner 2014). Palmer amaranth has a long duration of emergence and this residual control would be beneficial in controlling the new flushes of emergence.

Season-long palmer amaranth control is important for reducing yield loss and decreasing the soil seed-bank. The objective of this study was to determine the extent of Palmer amaranth control using post emergence applications of glufosinate only, or in combination with a residual herbicide.

5.3 Materials and Methods

5.3.1 Field design and implementation

Fields were located near Kankakee, Illinois in 2015 and 2016. The soil at Kankakee was a Kankakee fine sandy loam (loamy-skeletal, mixed, superactive, mesic Typic Hapludolls) with a pH of 6.5 and organic matter of 2%. Tillage was implemented each spring to remove any

existing vegetation and prepare the seedbed for planting. Glufosinate-resistant soybean variety "Credenz 3233 LL" was planted in 76-cm rows on May 19th, 2015 and May 26th, 2016 at Kankakee. Plots were 3 meters wide and 7.6 meters long and contained 4 rows of soybeans. An area between rows in the plot was marked with ½ m^2 quadrats. Experiments were designed as a randomized complete block design with four replications per treatment.

Herbicides were applied with a backpack CO_2 sprayer with Teejet[1] AI110025 and AIXR8002 spray tips for preemergence and post emergence applications, respectively. Nozzles were spaced 51 cm apart on a 3 meter boom calibrated to deliver 187 L ha^{-1} at 276 kPa. Environmental conditions were recorded at the time of each herbicide application.

Treatments consisted of combinations of preemergence and postemergence herbicides for Palmer amaranth control. Preemergence (PRE) treatments of tank-mixed sulfentrazone plus imazethapyr were applied at soybean planting at a full or half rate, based on common herbicide use rate in Illinois, to all of the plots. PRE herbicides were followed by an early postemergence (EPOST) application of glufosinate or glufosinate plus acetochlor when weeds were 5–8 cm tall and soybean were at the V2 growth stage. Additional postemergence (POST) applications consisted of glufosinate or glufosinate plus pyroxasulfone when weeds were 5–10 cm tall and soybeans were at a V6 growth stage (Table 1).

Herbicide effectiveness was visually estimated at 7, 14, and 21 days after treatment (DAT) on a scale ranging from 0 (no control) to 100 (complete weed control). The ratings were based on estimates of injury and biomass reduction when compared to non-treated plots.

Quadrats were randomly placed near the center of each plot and emerged Palmer amaranth plants within quadrats were recorded weekly to determine the number and duration of Palmer amaranth emergence after herbicide application. Above-ground Palmer amaranth

biomass was harvested within the quadrat area before each sequential herbicide application. At 21 days after final treatment, all remaining Palmer amaranth was harvested for above ground biomass from each marked quadrat. Palmer amaranth biomass was dried at 65°C for 7 days and dried biomass recorded.

5.3.2 Statistical Analysis

Data were evaluated in SAS[2] 9.4 (SAS Institute, Cary, NC 27513, USA) using the GLM procedure. Fixed effects were herbicide rate, year, and herbicide application timing. Random effects were block within year. The effect of year had a significant interaction and therefore the data from different years were not pooled. Means of significant main effects and interactions were separated using Fischer's Protected LSD test at $P \leq 0.05$.

5.4 Results and Discussion

High soybean injury ratings (Table 5.1) occurred in 2015 likely due to the excessive amount of rainfall and cool weather at the time of planting and soybean emergence. Soybean injury was low and nearly negligible in 2016 after the soybeans emerged from the soil. In both years, glufosinate in combination with glyphosate had the highest amount of soybean injury and necrosis (data not shown). Control of Palmer amaranth was greatest at 7 DAT after glufosinate only POST applications, but declined over time due to additional emerging weed seedlings and weed recovery. Glufosinate tank-mixed with acetochlor applied early postemergence (EPOST) had higher control ratings of Palmer amaranth compared to glufosinate alone at 21 DAT, however, the percent of control gradually declined as time after application increased. Similar research has also shown that a PRE herbicide followed by an EPOST application of glufosinate tank-mixed with acetochlor or pyroxasulfone provided the highest level of control for common lambsquarters (*Chenopodium album*), common waterhemp, Eastern black nightshade *(Solanum*

ptycanthum), and velvetleaf (*Abutilon theophrasti*) in glufosinate-resistant soybean compared to single or sequential glufosinate applications (Aulakh and Jhala 2015). Herbicides applied POST provided ≥90% control of Palmer amaranth in 2015 and the greatest control at 21 DAT both years, regardless if pyroxasulfone was added.

No difference in weed density or plant biomass was observed between the full and half rate of PRE-only treatments in both field years (Table 5.2). Greater Palmer amaranth biomass in the treated plots may be due to reduced interspecies competition for light and other nutrients in the marked quadrat. Single applications of glufosinate plus acetochlor had significantly lower final Palmer amaranth density than just a single application of glufosinate in 2016 (Table 5.3). This finding coincides with Coetzer et al. (2002), who reported less reduction of Palmer amaranth biomass using a single application of glufosinate rather than sequential applications. Tank mixing with glufosinate has been recommended to have greater control over species, such as common lambsquarters, that have multiple emergence events in a season (Steckel et al. 1997). Palmer amaranth control was greater when using sequential applications of glufosinate (Table 5.1) similar to studies by Hoffner et al. (2012) and Culpepper et al. (2000) reporting that a PRE followed by a POST application of glufosinate provided greater control of Palmer amaranth than a single application of glufosinate.

Biomass accumulation and Palmer amaranth density did not differ between two applications of glufosinate or two applications of glufosinate plus residual herbicides (Table 5.3). Two glufosinate applications decreased Palmer amaranth density early in the season similar to findings by Coetzer et al. (2002). Final dry biomass of Palmer amaranth harvested was lowest in plots where EPOST followed POST herbicide treatments. However, due to resistance concerns, the use of a post application of glufosinate and acetochlor or pyroxasulfone is recommended,

therefore when multiple modes of action are utilized, selection pressure is decreased (Diggle et al. 2003; Johnson et al. 2012; Norsworthy et al 2012). Acetochlor and pyroxasulfone herbicides have been reported to provide greater than 75% control of common waterhemp (Hausman 2011) and therefore could be utilized for suppression of other *Amaranthus* species.

5.5 Source of Materials

[1]TeeJet 80025EVS. TeeJet Technologies, P.O. Box 7900, Wheaton, IL 60187.

[2]Statistical Analysis Software (SAS) 9.4. SAS Institute, Inc., 100 SAS Campus Drive, Cary, NC 27513.

5.6 Tables

Table 5.1. Visual estimates of Palmer amaranth control and soybean injury at 7 and 21 DAT of latest herbicide application in Kankakee (2015 and 2016) under field conditions. Visual estimates of control sharing the same letter within a column are not significantly different at α=0.05.

treatment	herbicide[a]	rate	timing[b]	2015 control[c]		2015 injury[d]		2016 control	
				7	21	7	21	7	21
		g ai ha^{-1}		----------%----------					
0	untreated	0		0 e	0 d	0 d	0 c	0 d	0 e
1	sulfentrazone+imazethapyr	280	PRE	56 cd	45 c	2.5 c	2.3 b	26 c	14 e
2	sulfentrazone+imazethapyr glufosinate	280 594	PRE EPOST	79 b	69 b	2.8 c	2.8 ab	58 b	38 d
3	sulfentrazone+imazethapyr glufosinate+acetochlor	280 594+1260	PRE EPOST	73 b	66 b	2.5c	2.3 b	63 b	46 d
4	sulfentrazone+imazethapyr glufosinate glufosinate	280 594 594	PRE EPOST POST	95 a	93 a	3 bc	3 ab	86 a	68 bc
5	sulfentrazone+imazethapyr glufosinate+acetochlor glufosinate	280 594+1260 594	PRE EPOST POST	97 a	97 a	4.5 ab	3.5 ab	94 a	83 ab
6	sulfentrazone+imazethapyr glufosinate+acetochlor glufosinate+pyroxasulfone	280 594+1260 594+118	PRE EPOST POST	97 a	95 a	4.5 ab	3.5 ab	96 a	80 ab
7	sulfentrazone+imazethapyr	140	PRE	49 d	46 c	3.3 bc	3.3 ab	5 d	5 e

Table 5.1 (cont.)

8	sulfentrazone+imazethapyr	140	PRE						
	glufosinate	594	EPOST	79 b	70 b	2.5 c	2.3 b	59 b	35 d
9	sulfentrazone+imazethapyr	140	PRE						
	glufosinate+acetochlor	594+1260	EPOST	65 bc	61 bc	5.5 a	3 ab	68 b	50 cd
10	sulfentrazone+imazethapyr	140	PRE						
	glufosinate	594	EPOST						
	glufosinate	594	POST	96 a	97 a	5 a	4 a	91 a	79 ab
11	sulfentrazone+imazethapyr	140	PRE						
	glufosinate+acetochlor	594+1260	EPOST						
	glufosinate	594	POST	97 a	98 a	5 a	3.5 ab	97 a	88 a
12	sulfentrazone+imazethapyr	140	PRE						
	glufosinate+acetochlor	594+1260	EPOST						
	glufosinate+pyroxasulfone	594+118	POST	97 a	97 a	4.5 ab	4 a	95 a	79 ab

[a] All POST treatments included AMS, ammonium sulfate at 2.5% (v/v).

[b] PRE, pre emergence; EPOST, post emergence; POST, post emergence.

[c] average rating of Palmer amaranth control in treated plot

[d] average rating of soybean injury from herbicide treatment. Blank spaces are negligible injury

Table 5.2. Mean separation of PRE-only and the non-treated biomass of Kankakee Palmer amaranth under field conditions (2015 and 2016). Means sharing the same letter within the same column are not significantly different a α=0.05.

			biomass[a]		density[b]	
treatment	herbicide	rate	2015	2016	2015	2016
		g ha^{-1}				
0	untreated	0	34.1 a	64.9 a	24.5 a	41 a
1	sulfentrazone+imazethapyr	280	27.4 a	66.8 a	3.3 b	9.5 a
7	sulfentrazone+imazethapyr	140	11.0 a	111.7 a	5 b	24.8 a

[a] biomass of Palmer amaranth in the marked quadrat at 21 days after last experiment treatment
[c] number of Palmer amaranth in marked at 21 days after last experiment treatment quadrat

Table 5.3. Mean separation of EPOST and POST herbicide application biomass and density of Kankakee Palmer amaranth 21 days after POST application under field conditions (2015 and 2016). Means sharing the same letter within a column are not significantly different as α=0.05

	Herbicide application timing			biomass[a]		density[b]	
trt[c]	PRE	EPOST	POST	2015	2016	2015	2016
				(g)			
2	sulfentrazone+imazethapyr	glufosinate		2.83 ab	33.57 a	14.75 ab	7 a
8	sulfentrazone+imazethapyr	glufosinate		3.72 a	19.72 ab	10.75 abc	6.25 ab
3	sulfentrazone+imazethapyr	glufosinate+acetochlor		0.75 bc	5.74 b	11.25 abc	1.5 c
9	sulfentrazone+imazethapyr	glufosinate+acetochlor		2.62 abc	17.49 ab	15.25 a	1.5 c
4	sulfentrazone+imazethapyr	glufosinate	glufosinate	1.04 abc	0.03 b	1.5 bc	2.75 abc
10	sulfentrazone+imazethapyr	glufosinate	glufosinate	0 c	0.01 b	0.25 c	1.5 c
5	sulfentrazone+imazethapyr	glufosinate+acetochlor	glufosinate	0.98 bc	0 b	2 ab	0.5 c
11	sulfentrazone+imazethapyr	glufosinate+acetochlor	glufosinate	0.08 c	0 b	0.5 c	0 c
6	sulfentrazone+imazethapyr	glufosinate+acetochlor	glufosinate+pyroxasulfone	0 c	0.01 b	0 c	1 c
12	sulfentrazone+imazethapyr	glufosinate+acetochlor	glufosinate+pyroxasulfone	0 c	0 b	0.25 c	0 c

[a] biomass of Palmer amaranth in the marked quadrat at 21 days after POST treatment

[b] number of Palmer amaranth plants at 21 days after POST treatment

[c] trt, treatment

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CHAPTER 6

SOIL APPLIED HERBICIDES FOR PREEMERGENCE PALMER AMARANTH CONTROL

6.1 Abstract

Palmer amaranth is known for rapid biomass accumulation and multiple emergence events, making this weed difficult to control. Currently, Palmer amaranth populations in Illinois have only been documented for resistance to ALS, EPSP, and PPO site-of-action herbicides applied POST emergence, thus necessitating more research in PRE herbicides for Palmer amaranth suppression and control. Field experiments were conducted in 2016 near Kankakee and Urbana, IL. Herbicides from five different site-of-action groups were applied to bare ground fields, and Palmer amaranth biomass was collected at 56 days after treatment (DAT). Results indicated Palmer amaranth visual estimates of control was not different than the non-treated control 21 DAT. Only atrazine (2242 g ai ha^{-1}) and mesotrione (210 g ai ha^{-1}) had lower biomass than non-treated biomass in Kankakee. At Urbana, all treatments, excluding rimsulfuron (35 g ai ha^{-1}), had biomass lower than non-treated biomass. Under greenhouse conditions, PRE applications of rimsulfuron and imazethapyr (71 g ai ha^{-1}) were not different than the untreated control.

6.2 Introduction

Palmer Amaranth (*Amaranthus palmeri*) is a summer annual, small-seeded broadleaf species that originates from the Sonoran desert of North America (Sauer 1957; Ward et al. 2013). This plant is in the Amaranthaceae family in the order Centrospermae, a group that contains anthocyanin pigments (Steckel 2007). Palmer amaranth has been expanding into the Midwest and farmers are concerned that this weed will have the same damaging effect on their fields as Palmer amaranth has released upon the South. Studies conducted by Davis et al. (2015)

investigated the importance of genetics and environmental factors that would help determine the extent of damage Palmer amaranth would inflict upon the Midwest. McDonald et al. (2009) hypothesized that increased temperatures would expand the damage niche of southern-originating weed species northward. This temperature increase would be beneficial for Palmer amaranth growing conditions because of the favorable response of Palmer amaranth to increased temperature (Ehleringer, 1983; Guo and Al-Khatib, 2003). The damage niche for Palmer amaranth is not reliant on the genotype of this weed, but rather the growing environment in which the seed is dispersed (Davis et al. 2015).

Palmer amaranth can be identified by a glabrous stem with petioles that are longer than the ovate leaf blade. The leaves are alternate and sometimes have a chevron (Ward et al. 2013). Palmer amaranth is dioecious species with male plants producing the pollen and the female plants producing the seeds. Both inflorescences can grow up to a meter in length. The female inflorescence is distinguishable from the male inflorescence due to stiff bracts that are sharp to the touch. The females produce a prolific amount of seed, often ranging between 200,000–600,000 seeds when the plant emerges from March to June (Ward et al. 2013). The utricle in 1.5 to 2 mm long with seed 1 to 1.25 mm in diameter with dark red to black coloring (Sauer 1955; Ward et al. 2013). Location on the inflorescence influences when seed matures, and seeds that mature on the top and middle third of the inflorescence have a 67–78% greater germination rate (Jha et al. 2010). Palmer amaranth is able to overwhelm a field because of multiple emergence events that are produced within a growing season (Sauer 1957). The seeds germinate throughout the growing season, which necessitates multiple herbicides application times throughout the year if there is no lasting soil activity from the herbicide (Ward et al).

Germination is stimulated by natural or red light while far-red light inhibits germination (Jha et al. 2010) which suggests when seed is under crop canopy rather than bare ground, germination will be decreased due to less light penetration. Germination occurs over a wide range of temperatures and the increase of temperature also leads to an increase in germination, with the peak occurring when the temperature was approximately 30°C (Steckel et al. 2004). Research indicates a smaller window of opportunity to control Palmer amaranth with herbicides before the plant is too large during periods of higher temperatures (Powell 2014).

Palmer amaranth is a C4 species with a photosynthetic rate around 80 μmol m^{-2} s^{-1} and rapid biomass accumulation (Ehleringer 1983). Palmer amaranth utilizes this high photosynthetic rate via diaheliotropic movement of the leaves (Ehleringer, 1983). This weed has been recorded to be 10 centimeters tall two weeks after planting and 24 centimeters at four weeks (Sellers et al. 2003). Palmer amaranth can grow 0.18–0.21 centimeters per growing degree day (GDD), which can quickly begin to outcompete the crop in the field (Horak and Loughin, 2000). Palmer amaranth competition with soybean can cause a 17–63% reduction in yield per 0.33–10 plants per m^{-1} (Klingaman and Oliver 1994) or 79% yield reduction at 8 plants per m^{-1} (Bensch 2003) when this weed emerges shortly after soybeans. In corn, Palmer amaranth reduced yield 11–91% when this weed emerged with corn at a density of 0.5 to 8 plants m^{-1}, while emergence after corn still resulted in 7 to 35% yield reduction at the same plant densities (Massinga et al. 2001).

Due to Palmer amaranth's ability of germinate multiple times throughout the season, pre emergence herbicides are an effective option for reducing early season weed populations. Herbicide applications of a very-long-chain fatty acid (VLCFA) inhibitor has been shown to reduce sicklepod fresh biomass when compared to untreated controls (Adcock and Banks 1991).

Preemergence along with postemergence herbicides are recommended for maximum weed control and crop yield protection (Loux et al. 2016). Soil-applied herbicides have been shown to reduce weed density and dry biomass more than non-treated controls (Hager et al. 2001). Without the addition of a PRE herbicide, corn yield has been reported to decrease when POST herbicide application was delayed, while yield was unaffected by POST timing when PRE herbicides were included (Parker et al. 2006). Previous research reported flumioxazin and formesafen to control Palmer amaranth at 20 DAT ranging from 74–100% depending on environment (Whitaker et al. 2011) Palmer amaranth control in sandy soils ranged from 50–97% at 28 DAT when metolachlor (1.7 kg ha) was applied preemergence (Keeling and Abernathy 1989). Predicted control of Palmer amaranth was reported higher at 96 and 90% for pyroxasulfone (179 g ai ha^{-1}) and isoxaflutole (105 g ai ha^{-1}) and lower at 82 and 69% for S-metolachlor (1068 g ai ha^{-1}) and metribuzin (420 g ai ha^{-1}) (Meyers et al. 2016). The objective of this study was to evaluate residual control of Palmer amaranth by site-of-action herbicides commonly applied preemergence in Illinois cropping systems. Palmer amaranth has been reported to be resistant to 6 herbicide sites-of-action (Heap 2017), but only one of these site-of-action herbicides is a PRE herbicide (microtubule inhibitors) (Gosset et al. 1992).

6.3 Materials and Methods

6.3.1 Plant propagation and experimental design under greenhouse conditions

Palmer amaranth seed inflorescences were collected in fall 2015. Seeds were removed from the inflorescence and treated similar to the methods of Kępczyński, and Sznigir (2013) to improve germination. The ethylene dilution of was applied at a volume of 9 mL in each petri dishes. Petri dishes were filled with 50 Palmer amaranth seeds and three petri papers, and then sealed and stored at 4°C for four weeks.

Greenhouse experiments followed the procedure described by Hausman (2011). Plastic pots (720cm^3) were filled to the top with growth medium (1:1:1 mixture of soil, peat, and sand, with a pH of 6.8 and 3.5% organic matter), tamped to create a level planting surface, and then soaked in water for 12 hours to ensure uniform moisture. Stratified Palmer amaranth seed were placed in a 5 by 5 grid and then covered with 50 ml of the same soil mixture. The top surface was lightly tamped down to produce and even, flat surface. Pots were watered over the top with a 1.9 liter per minute (LPM) mister nozzle until the soil surface was moist to the touch. Greenhouse conditions were maintained with a 16-hour photoperiod at 28/22°C day/night fluctuation.

Preemergence herbicides representing five site-of-action groups were selected for evaluation and applied at the corresponding label-recommended rate (Table 1). Herbicides were applied using a compresses air research sprayer[3] fitted with a TeeJet[1] 80015 EVS nozzle calibrated to deliver 185 L ha^{-1} at 275 kPa. After the treatments were applied, rainfall was simulated to move the herbicide into the growth medium at a rate of 7 milliliters per pot using a 8005 E nozzle. The treated pots were then returned to the greenhouse room and arranged in a randomized complete block design with four replication per treatment and the experiment was conducted twice. The pots were watered daily with a 1.9 LPM mister until the soil was moist to the touch. Germination counts were taken and the applications were visually evaluated at 7, 14, and 21 days after treatment (DAT).

6.3.2 Field design and implementation

Fields were near Kankakee, Illinois and Urbana, Illinois in 2016. The soil at Kankakee was a Kankakee fine sandy loam (loamy-skeletal, mixed, superactive, mesic Typic Hapludolls) with a pH of 6.5 and organic matter of 2%. The Urbana soil type was a Flanagan silt loam (fine,

smectitic, mesic Aquic Argiudolls) with a pH of 6.5 and an organic content of 4.9%. Soybean planting was simulated on bare ground trials began on May 26th in 2016 for the Kankakee field location and the Champaign location was planted on June 8th in 2016. While all plots were 3 meters wide, plots in Kankakee were 7.6 meters in length and Urbana plots were 10 meters in length. Quadrats in Kankakee were ½ m^2, while a 1 x ½ m^2 was used in Urbana. Differences in quadrat size was due to a much higher Palmer amaranth density at Kankakee compared with Urbana.

Tillage was implemented each spring to remove any existing vegetation and prepare the seedbed for planting. Treatments were structured in a randomized complete block design with three replications. All herbicides were applied with a backpack CO_2 sprayer equipped with TeeJet[1] AI110025 spray tips spaced 51 cm on a 3 m spray boom. Spray volume was 187 L ha^{-1} and pressure was 276 kPa. Environmental conditions were recorded at each herbicide application. Selection of herbicides rates and additives were chosen based on label recommendations and current Illinois crop practices.

6.3.3 Statistical Analysis

Due to Palmer amaranth population variability between fields, results were analyzed separately by location. Data were analyzed in SAS[2] 9.4 (SAS Institute, Cary, NC 27513, USA) using the GLM procedure. Fixed effects were Palmer amaranth population and herbicide treatment. Random effects were year and block within year (field only), or experimental run (greenhouse only). Means of Palmer amaranth biomass were separated using Fischer's Protected LSD test at ($P \leq 0.05$).

6.4 Results and Discussion

6.4.1 Preemergence herbicide efficacy on Palmer amaranth under greenhouse conditions

Non-necrotic Palmer amaranth plants were observed in both ALS and HPPD herbicide treatments at the end of the 21 DAT. The number of Palmer amaranth that was present at 21 DAT in the untreated control was higher than all the other herbicide treatments (Table 6.1). Counts of emerged Palmer amaranth in rimsulfuron and imazethapyr treatments was lower than the amount in non-treated pots, but also greater than other herbicide treatments. When comparing 21 DAT biomass, rimsulfuron and imazethapyr treatments were similar to the non-treated (Table 6.1).

6.4.2 Preemergence herbicide efficacy on Palmer amaranth under field conditions

Lack of weed control was noted in Kankakee after 14 DAT, causing control ratings to be undiscernible by 28 DAT. Control of Palmer amaranth with PRE herbicides has been shown to be negatively correlated with weeks after treatment (Meyer et al. 2016). Only atrazine and mesotrione had biomass lower than non-treated biomass in Kankakee (Table 6.2). Palmer amaranth control has been reported as achievable in sandy soils, however, postemergence treatments will be required (Keeling and Abernathy 1989).

Urbana Palmer amaranth pressure was noted as low in the first block of the experiment. Urbana 21 DAT data showed non-treated biomass higher than all other treatments, except for rimsulfuron.

Greenhouse and field experiments in Urbana coincided with findings by Whitaker (2011) and Everman (2009) that PPO-inhibiting herbicides provide greater control of Palmer amaranth compared to other PRE treatments. PRE herbicides are important in an increasingly post emergence herbicide resistant weed species. More research is needed to validate these findings as Palmer amaranth weed pressure was inconsistent in Urbana during the summer of 2016.

6.5 Source of Materials

[1]TeeJet 80025EVS. TeeJet Technologies, P.O. Box 7900, Wheaton, IL 60187.

[2]Statistical Analysis Software (SAS) 9.4. SAS Institute, Inc., 100 SAS Campus Drive, Cary, NC 27513.

[3] Generation III Research Sprayer. DeVries Manufacturing, 28081 870th Ave., Hollandale, MN 56045.

6.6 Tables

Table 6.1. Mean separation of Palmer amaranth biomass 21 days after treatment in greenhouse experiment.

herbicide	rate	biomass[a]	plant[b]
	g ha^{-1}	(g)	
control	0	0.25 a	7.88 a
rimsulfuron	35	0.29 a	5.38 b
imazethapyr	71	0.20 a	5.13 b
isoxaflutole	105	0.03 b	1.00 c
mesotrione	210	0.33 b	0.75 c
metribuzin	420	0.001 b	0.13 c
s-metolachlor	1423	0.001 b	0.13 c
flumioxazin	108	0 b	0 c
atrazine	2242	0 b	0 c
sulfentrazone	280	0 b	0 c
pyroxasulfone	178	0 b	0 c

[a] biomass with the same letter are not significantly different

[b] Palmer amaranth counts with the same letter are not significantly different

Table 6.2. Mean separation of Palmer amaranth biomass 56 days after treatment in 2016 field locations in Kankakee and Urbana.

herbicide	rate	Kankakee biomass[a]	Urbana
	g ai ha^{-1}	(g)	
control	0	235 a	300 a
acetochlor	1715	168.3 abc	61.5 c
atrazine	2242	121.7 c	29.8 c
flumioxazin	108	176.7 abc	5.5 c
imazethapyr	71	173.3 abc	128.8 bc
isoxaflutole	105	195 abc	90.6 bc
mesotrione	210	126.7 bc	14 c
metribuzin	420	156.7 abc	2.7 c
pyroxasulfone	178	196.7 abc	2.5 c
rimsulfuron	35	148.3 abc	253.3 ab
s-metolachlor	1423	230 ab	20.5 c
sulfentrazone	108	200 abc	74.5 c

[a] biomass with the same letter are not significantly different.

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