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Postemergence Control of Palmer Amaranth with Mesotrione-Based Herbicide Mixtures and the Impact of Lactofen and Planting Date on the Growth, Development, and Yield of Indeterminate Soybean

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Postemergence control of Palmer amaranth with mesotrione-based herbicide mixtures and the impact of lactofen and planting date on the growth, development, and yield of indeterminate soybean

By

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Postemergence control of Palmer amaranth with mesotrione-based herbicide mixtures and the impact of lactofen and planting date on the growth, development, and yield of indeterminate soybean

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Research was conducted in 2013 and 2014 to evaluate the postemergence control of Palmer amaranth [*Amaranthus palmeri* (S.) Wats.] with mesotrione alone and in mixtures with fomesafen and/or glyphosate and to evaluate the impact of lactofen and planting date on growth, development, and yield of indeterminate soybean [*Glycine max* (L.) Merr.]. Studies included a greenhouse evaluation of different rates of mesotrione on the control of 5- and 10-cm Palmer amaranth and field studies evaluating the control of 5- to 10-cm Palmer amaranth with three rates of mesotrione applied alone and in mixtures with fomesafen and/or glyphosate. Lactofen studies include a planting date study evaluating one rate of lactofen applied at V2 soybean stage with planting dates of April 15, May 1, May 15, and June 1 and a lactofen timing study where one rate of lactofen was applied at soybean growth stages ranging from V1 to R5.

DEDICATION

I would like to dedicate this work to my brothers, John Finley and Alex Mangialardi, and my parents, Terri and Greg Mangialardi. Words cannot describe how thankful I am for all the support and encouragement you have provided me throughout my education. Without your support, I would not be where I am today.

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER I

INTRODUCTION

Soybean [*Glycine max* (L.) Merr.] were domesticated in China around 1500 to 1100 B.C. (Hymowitz 2004). The production of soybean spread through Japan, Indonesia, Philippines, Vietnam, Thailand, Malaysia, Myanmar, Nepal, and northern India where it was used in the development of several different foods (Hymowitz 2004). Soybean movement during this time resulted from the establishment of sea and land trade routes, migration within China, and the rapid adoption of the soybean seed as a stable food source (Hymowitz 1990; Hymowitz and Newell 1980). Soybean was introduced to North America in 1765 (Hymowitz 2004).

In the Mississippi Delta, soybean production emerged in conjunction with cotton (*Gossypium hirsutum* L.) production (Snipes et al. 2005). Traditionally, soybean production in the area consisted of planting late maturity group (MG) V, VI, and VII cultivars in May and June (Heatherly 1998). Planting at this time consistently produced poor yields due to low rainfall amounts during soybean reproduction (Snipes et al. 2005). In an effort to minimize this problem, the Early Soybean Production System (ESPS) was developed (Heatherly 1998). Early Soybean Production System focuses on planting early-maturing MG IV and V soybean cultivars in April in an attempt to avoid seasonal drought during the reproductive growth stages (Bowers et al. 1998). A systematic approach including seedbed preparation in the fall, application of preplant foliar

herbicides for control of winter/spring weeds, and planting early-maturing cultivars into undisturbed, or stale, seed beds in April are integral steps for soybean production under ESPS (Heatherly and Spurlock 1999). By 2001, adoption of ESPS was widespread in MS, and is still used extensively today (Snipes et al. 2005).

Planting an earlier MG soybean in early April increases the probability of greater yields (Heatherly 1998). Adherence to ESPS not only results in greater yields, but the same cultivars are often shorter when planted early (T.W. Eubank unpublished data). Heatherly (1998) reported that MG V soybean planted May 27 in Stoneville, MS, initiated pod set August 5 and pod fill August 19. Maturity group VI soybean planted May 12 began pod set August 11 and pod fill August 23 (Heatherly 1998). Maturity group VII soybean planted May 12 began setting pods August 16 and began pod fill August 28 (Heatherly 1998). Therefore, MG V, VI, and VII soybean cultivars planted in May or June resulted in pod set and fill during periods of low moisture and subsequently produced low yields (Heatherly 1998). However, MG IV soybean planted April 18 encountered less adverse growing conditions and initiated pod set in mid-June, pod fill July 3, and reached full pod stage on August 28 (Heatherly 1998).

To maximize soybean yield, many factors other than planting date must be considered. These include soil pH, availability of adequate soil moisture during reproductive development, achieving canopy closure for increased light interception, and maintaining adequate pest control (Cassman 1999; Heatherly and Elmore 2004; Sinclair 1993). A soil pH of 6 to 6.5 is needed to optimize soybean yield (Heatherly and Elmore 2004). The pH of acidic soils can be adjusted to the desired range, so the soybean's ability to absorb nutrients improves (Mengel et al. 1987). Applying lime to acidic soils

reduces concentrations of potentially toxic elements such as H, Al, and Mn, increases the availability of Ca, Mg, and Mo, and increases N_2 fixation by nodulation (Mengel et al. 1987).

Nodulation occurs when *Bradyrhizobia japonicum* bacteria infect the soybean plant causing nodules to form on the roots (Heatherly and Elmore 2004). The relationship between the bacteria and the soybean is symbiotic in that the soybean provides carbohydrates and mineral nutrients to the bacteria which in turn provides N to the host soybean plant (Heatherly and Elmore 2004). Approximately 75% of N in a mature soybean plant is a product of biological N fixation and the remaining comes from the soil N (Varco 1999). Both sources of N are required for maximum yield (Heatherly and Elmore 2004). Heatherly and Elmore (2004) reported that N assimilation via N_2 fixation requires more photosynthate than NO_3 uptake.

When soybean is grown under ideal conditions, such as one with adequate nutrients, water, and the absence of abiotic and biotic stress, crop mass accumulation is directly correlated to the amount of light intercepted by the crop (Cassman 1999; Sinclair 1993). Daily crop mass accumulation is a function of the amount of light interception and the radiation use efficiency, which is a measure of canopy photosynthesis (Purcell and Sinclair 2004). The use of narrow-row production systems (<50 cm) is a tool to reach canopy closure sooner and optimize the level of light interception earlier in crop development than that of a wide-row production system (Heatherly and Elmore 2004).

Like cotton, soybean were initially grown on 97- to 102-cm row spacing using PRE, POST, and post-directed herbicide applications, as well as cultivation, for weed control (Snipes et al. 2005). As the adoption of glyphosate-resistant soybean increased,

soybean row spacing decreased to 38 to 51 cm, and the use of cultivation and residual herbicides decreased (Snipes et al. 2005). However, due to weed resistance issues with glyphosate, there has been an increase in the use of soil-applied residual herbicides and tillage in an attempt to control glyphosate resistant weeds, primarily Palmer amaranth [*Amaranthus palmeri* (S.) Wats.] (Nichols et al. 2009; Prince et al. 2012).

Palmer amaranth is a member of the Amaranthaceae family, which contains approximately 75 species worldwide (Ward et al. 2013). Palmer amaranth is one of 10 dioecious *Amaranthus* spp. in North America and is native to areas from northwestern Mexico and southern California to New Mexico and Texas (Sauer 1957; Steckel 2007). Palmer amaranth grows rapidly, is drought tolerant, and adapts well to shading (Ehleringer 1983; Jha et al. 2008; Place et al. 2008; Wright et al. 1999). Palmer amaranth plants typically include a single reddish-green stem with many lateral branches (Sauer 1955). Leaves are non-pubescent and are attached to long petioles that exceed the length of the leaf blade (Sauer 1955). As plants mature, leaf blades often transform from lance-shaped to more ovate (Sauer 1955). Mature leaves of Palmer amaranth exhibit whitish veins on their underside, and often have a dark chevron shape on their upper surface (Franssen et al. 2001; Sauer 1955). Palmer amaranth is dioecious meaning the male parts, the staminate, and female parts, the pistillate, occur on separate plants (Ward et al. 2013).The male and female inflorescence can be differentiated by touch with male inflorescence soft and female inflorescence tough and prickly because of stiff bracts (Ward et al. 2013). Male Palmer amaranth plants produce large amounts of pollen, which can be dispersed up to 300 m via the wind (Sosnoskie et al. 2012; Ward et al. 2013).

Palmer amaranth seed are small, smooth, and dispersed primarily by gravity (Costea et al. 2004, 2005; Norsworthy et al. 2009; Sauer 1955). Other Palmer amaranth seed dispersal mechanisms include movement by irrigation water and mammals, or agricultural practices such as plowing, mowing, and harvesting (Costea et al. 2004, 2005; Norsworthy et al. 2009). Female Palmer amaranth can produce 200,000 to 600,000 seed plant^{-1} under ideal growing conditions such as non-shaded areas with temperatures between 36 and 46 C (Ehleringer 1983; Keeley et al. 1987). Continual, season-long emergence of the seed makes Palmer amaranth problematic (Keeley at al. 1987; Sellers et al. 2003).

After developing resistance to dinitroaniline herbicides in the 1980s, Palmer amaranth became one of the top 10 most troublesome weeds of cotton producers in South Carolina (Webster and Coble 1997). By 2009, Palmer amaranth was ranked as the most troublesome weed of cotton in nine of 10 states surveyed in the southern U.S. and among the top 10 most troublesome weeds in both soybean and corn (*Zea mays* L.) production (Webster and Nichols 2012). Palmer amaranth was ranked as the most troublesome weed of cotton in seven of eight surveyed states, and the most troublesome weed of soybean in four of seven surveyed southern U.S. states in 2013 (Webster 2013).

Continual use of herbicides with the same MOA as the only means of weed control increases the probability of herbicide resistance (Holt 1992; Owen and Zelaya 2005). Multiple resistance, such as Palmer amaranth resistant to both glyphosate and acetolactate synthase (ALS) herbicides, is common (Nandula et al. 2012). Populations of Palmer amaranth have evolved resistance to five different herbicide modes of action including glyphosate, ALS inhibitors, dinitroanilines, triazines, and 4-

hydroxyphenylpyruvate dioxygenase (HPPD) herbicides (Ward et al. 2013). Multiple resistance mechanisms such as target site mutations affecting herbicide binding kinetics and amplification of target site genes or non-target site mechanisms such as metabolism and translocation are common (Rong et al. 2013).

Mesotrione was developed by Syngenta for the control of broadleaf and grass weeds in corn (Shaner 2014). It was derived from leptospermone, which is a compound found in the roots of red bottlebrush (*Callistemon citrinus* Curtis) (Cornes 2005). Red bottlebrush is a member of the Mytraceae family, which originates from Australia (Cornes 2005). In 1977, a biologist working at the Western Research Centre of the Stauffer chemical company in California noticed a lack of plants growing under red bottlebrush bushes (Cornes 2005). After testing, he concluded the compound responsible for the herbicidal activity was leptospermone (Cornes 2005). Mesotrione was derived from leptospermone 11 years later (Cornes 2005).

Mesotrione is a triketone herbicide, which provides selective weed control in corn, asparagus (*Asparagus officinalis* L.), blueberry (*Vaccinium pallidum* Ait.), lingonberry (*Vaccinium vitis-idaea* L.), black raspberry (*Rubus occidentalis* L.), red raspberry (*Rubus idaeus* L.), blackberry (*Rubus fruticosus* L.), bluegrass (*Poa annua* L.), perennial ryegrass (*Lolium perenne* L.), tall fescue [*Lolium arundinaceum* (S.J.) Darbyshire.] grown for seed, cranberry (*Vaccinium oxycoccos* L.), flax (*Linum usitatissimum* L.), oats (*Galega officinalis* L.), okra (*Abelmoschus esculentus* L.), pearl millet (*Pennisetum americanum* L.), rhubarb (*Rheum rhabarbarum* L.), sugarcane (*Saccharum officinarum* L.), and sorghum (*Sorghum bicolor* L.) (Anonymous 2014). Mesotrione can be applied PRE or POST, depending on the crop, for control of annual

broadleaf and grass weeds (Shaner 2014). Mesotrione inhibits the enzyme HPPD, which indirectly blocks carotenoid synthesis (Shaner 2014). Carotenoids are necessary for photosynthesis and the protection of chlorophyll and plant cell membranes (Cornes 2005). The disruption of chlorophyll causes bleaching and tissue necrosis and eventual plant death (Shaner 2014). Mesotrione may be absorbed by the leaves, shoots, or roots and is moved throughout the plant via xylem and phloem (Cornes 2005).

Glyphosate is a nonselective herbicide commercialized in 1974 and used throughout the world to control a broad spectrum of weeds (Nandula et al. 2012). With the introduction of glyphosate-resistant (GR) crops in 1996, producers rapidly adopted glyphosate for in-season weed control (Sammons et al. 2007). The widespread use of glyphosate has not only caused weed species shifts in crops but has also caused some weed populations to evolve resistance (Nandula et al. 2012). Thirty-two weed species worldwide are resistant to glyphosate, and nine of these are found in Mississippi (Heap 2015).

Glyphosate inhibits 5-enolpyruvylshikimate-3-phosphate synthase (EPSPS), which leads to several metabolic disturbances, including inhibition of protein, secondary product biosynthesis, and deregulation of the shikimate pathway (Franz et al. 1997). Phosphoenolpyruvate (PEP) and erythrose 4-phosphate are converted to chorismate via the shikimate pathway (Herrmann and Weaver 1999; Kishore and Shah 1988). Chorismate is the precursor to aromatic amino acids and many aromatic secondary metabolites (Herrmann and Weaver 1999; Kishore and Shah 1988). This process prevents the biosynthesis of aromatic amino acids (Jones and Smith 2010). In turn, glyphosate competes with PEP by occupying its binding site (Vencill et al. 2012). Because

glyphosate is a non-selective herbicide, it was first used strictly for preplant weed control (Nandula et al. 2012). In 2012, 83% of the total soybean acres in MS were treated with glyphosate (USDA 2014).

Glyphosate resistant Palmer amaranth was first reported in Georgia in 2004 when glyphosate was applied at 12X field rates and failed to control the biotype (Culpepper et al. 2006). Currently, GR Palmer amaranth can be found in 24 states throughout the U.S. (Heap 2015). Gaines et al. (2011) reported glyphosate resistance in Palmer amaranth was due to increased amounts of 5-enolpyruvylshikimate-3-phosphate synthase (EPSPS). Genomic copies of EPSPS enzymes appeared to act like a molecular sponge to absorb glyphosate and enabled resistant plants to continue functioning after application of glyphosate (Ward et al. 2013). Mohseni-Moghadam et al. (2013) further supports these findings in that the level of resistance in GR Palmer amaranth populations in New Mexico was due to the negative correlation between EPSPS copy numbers and shikimate accumulations. Studies in Kansas also documented resistance in Palmer amaranth to ALS where 14 different ALS-inhibiting herbicides, applied at 8X field rates, failed to provide adequate control (Gaeddert et al. 1997). Palmer amaranth populations resistant to both glyphosate and ALS herbicides have also been reported (Bond et al. 2010; Sosnoskie et al. 2011).

Prior to the development of PPO resistance in tall waterhemp [*Amaranthus tuberculatus* Moq.], PPO herbicides, such as aciflurofen, lactofen, and fomesafen, were applied in mixtures with ALS herbicides to control ALS-resistant biotypes (Gaedert et al. 1997). In 2012, 16% of the total soybean hectares in Mississippi received an application of fomesafen (USDA 2014). In the U.S., tall waterhemp and common ragweed (*Ambrosia*

artemisiifolia L.), were identified resistant to PPO herbicides in 2001 and 2005, respectively (Heap 2015).

Protoporphyrinogen oxidase (PPO) herbicides inhibit the protox enzyme, the last enzyme of the common branch of the heme and Chl biosynthetic pathways, and prevent protoporphyrinogen IX (protogen) from converting to protoporphryin IX (proto) within the plastid of the cell (Matringe et al. 1989). As a result, protogen accumulates in the plastid (Jacobs et al. 1991; Witkowski and Halling 1989). Protogen is then exported from the plastid into the cytoplasm and surrounding cellular membranes where it is converted to proto (Jacobs et al. 1991; Jacobs and Jacobs 1993). Proto will react with light and molecular oxygen to form radical singlet oxygen causing lipid peroxidation of the cellular membranes (Becerril and Duke 1989). After membrane disruption has occurred, cell contents are released, causing electrolyte leakage and cells to dry up, resulting in plant death (Becerril and Duke 1989; Duke and Kenyon 1993).

Fomesafen is a PPO herbicide that can be applied PRE or POST-directed in cotton and PRE and POST in soybean, potato (*Solanum tuberosum* L.), dry bean (*Phaseolus vulgaris* L.), and snap bean (*Phaseolus vulgaris* L.) to control broadleaf weeds, including *Ipomoea* spp., *Amaranthus* spp., jimsonweed (*Datura stramonium* L.), wild mustard (*Sinapis arvensis* L.), black nightshade [*Solanum americanum* (P.) Mill], and *Ambrosia* ssp. (Shaner 2014). Fomesafen is absorbed through the roots when soil-applied and leaf tissue when foliar-applied, but translocation is limited within the plant (Fadayomi and Warren 1977; Ritter and Coble 1981; Unland et al. 1999). Fomesafen is xylem mobile (Shaner 2014). When foliar-applied, fomesafen causes leaves of susceptible plants to become chlorotic and eventually necrotic (Shaner 2014).

Lactofen is a PPO herbicide used for control of broadleaf weeds in cotton, peanut, and soybean **(**Hart and Roskamp 1998; Kapusta et al.1986; Wichert and Talbert 1993). Lactofen's primary target site is the chlorophyll synthesis pathway, where it inhibits the enzyme protoporphyrinogen oxidase **(**Fennimore and Hembree 2006**).** Tolerant crops exhibit bronzing on young, expanded leaves following treatment with lactofen (Shaner 2014). Lactofen often causes chlorosis, necrosis, or stunting of soybean, and soybean canopy closure can be decreased by 6% following an application (Edwards and Purcell 2005; Hart and Roskamp 1998; Kapusta et al. 1986; Wichert and Talbert 1993). Injury from PPO herbicides may persist up to 21 d after treatment (Kapusta et al. 1986). Lactofen is currently labeled for white mold suppression in soybean (Anonymous 2013). A delay in canopy closure resulting from POST lactofen application reduced incidence of white mold due to increased air movement in both irrigated and nonirrigated soybean (Dann et al. 1999; Levene et al. 1998).

Maximized and sustainable soybean yields are the ultimate goal of producers. After a popular press article reported a Missouri producer set a record soybean yield of 10,348 kg ha^{-1} in 2007 and partially credited routine lactofen applications with enhancing yield due to altering plant height and branching, it was questioned if this could be done on large scale production under growing conditions in the midsouthern U.S. (Anonymous 2008). Application of plant growth regulators (PGR) is common in multiple crops, including the use of mepiquat chloride to control plant growth and maximize yield and quality of cotton (Ren et al. 2013). Mepiquat chloride use is advantageous for controlling vegetative growth, which can be detrimental to fiber yield and quality if left uncontrolled (Constable 1995; Kerby 1985; Oosterhuis and Egilla 1996). Increased fruit abscission and

reduced yield can be attributed to shade within the canopy as a result of excessive vegetative growth (Guinn 1974). Mepiquat chloride application results in shorter and more compact plants, lower leaf area index as a result of smaller leaf size, and early maturity (Kerby 1985; Reddy et al. 1990; York 1983). Some herbicides are used as PGRs. Glyphosate is applied at 0.04 to 0.18 kg ae ha^{-1} to stimulate sucrose accumulation and suppress flowering in sugarcane (Bennet and Montes 2003; Velini et al. 2010).

Lactofen is recommended at 0.22 kg ha^{-1} for POST control of hemp sesbania [*Sesbania herbacia* (P. Mill.) McVaugh], *Ipomoea* ssp, common ragweed (*Ambrosia artemisiifolia* L.), and *Amaranthus* ssp. in Mississippi soybean (Byrd 2015). Lactofen could alter soybean growth in a positive way; however, soybean injury can be severe when treated with lactofen (J. A. Bond, personal communication).

Mesotrione and lactofen have the potential to benefit soybean producers. This research will be beneficial in production scenarios by advancing weed control and determining the effect of POST applications of lactofen on the growth, development, and yield of indeterminate soybean. Specific objectives of this research are to (1) evaluate POST control of Palmer amaranth with mesotrione-based herbicide mixtures and (2) determine the impact of planting date and lactofen application on growth, development, and yield of indeterminate soybean. We determined the effect of different application rates of mesotrione on the POST control of Palmer amaranth, the efficacy of mesotrione when applied alone and in mixtures with fomesafen and/or glyphosate, the effect of lactofen and planting date on growth, development, and yield of indeterminate soybean, and the effect of lactofen application timing on the growth, development, and yield of indeterminate soybean.

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CHAPTER II

POSTEMERGENCE CONTROL OF PALMER AMARANTH WITH MESOTRIONE BASED HERBICIDE MIXTURES

Abstract

A two-year study was conducted at the Mississippi State University Delta Research and Extension Center in Stoneville, MS, to determine the optimum use rate of mesotrione for control of 5- to 10-cm Palmer amaranth when applied alone and in mixtures with glyphosate and fomesafen. A greenhouse study was also conducted to identify the optimum rate of mesotrione alone for control of 5- and 10-cm Palmer amaranth. In the greenhouse, control of 5-cm Palmer amaranth 14 and 21 d after treatment (DAT) was comparable with mesotrione at 0.11 and 0.17 kg ai ha^{-1}. No differences in control of 10-cm Palmer amaranth were observed 21 DAT. When mesotrione at 0, 0.05, 0.11, and 0.16 kg ai ha^{-1} was applied alone in the field, control was ≤49% 28 DAT. Glyphosate at 0.86 kg ae ha^{-1} and fomesafen at 0.26 kg ai ha^{-1} applied alone in the field provided ≤ 35 and 93% control, respectively, 28 DAT. Control with fomesafen applied alone was not improved with the addition of mesotrione and/or glyphosate.

Nomenclature: Fomesafen; Glyphosate; Mesotrione; Palmer amaranth, *Amaranthus palmeri* S. Wats.

Key words: Glyphosate resistant, Application timing, Treatments

Introduction

Palmer amaranth, is a member of the Amaranthaceae family, which contains approximately 75 species worldwide (Ward et al. 2013). Palmer amaranth is one of 10 dioecious *Amaranthus* spp. in North America and is native to areas from northwestern Mexico and southern California to New Mexico and Texas (Sauer 1957; Steckel 2007). Palmer amaranth grows rapidly, is drought tolerant, and adapts well to shading (Ehleringer 1983; Jha et al. 2008; Place et al. 2008; Wright et al. 1999). Palmer amaranth plants typically include a single reddish-green stem with many lateral branches (Sauer 1955). Leaves are non-pubescent and are attached to long petioles that exceed the length of the leaf blade (Sauer 1955). As plants mature, leaf blades often transform from lance-shaped to more ovate (Sauer 1955). Mature leaves of Palmer amaranth exhibit whitish veins on their underside, and often have a dark chevron shape on their upper surface (Franssen et al. 2001; Sauer 1955). Palmer amaranth is dioecious meaning the male parts, the staminate, and female parts, the pistillate, occur on separate plants (Ward et al. 2013).The male and female inflorescence can be differentiated by touch with male inflorescence soft, and female inflorescence tough and prickly because of stiff bracts (Ward et al. 2013). Male Palmer amaranth plants produce large amounts of pollen, which can be dispersed up to 300 m via the wind (Sosnoskie et al. 2012; Ward et al. 2013).

Palmer amaranth seed are small, smooth, and dispersed primarily by gravity (Costea et al. 2004, 2005; Norsworthy et al. 2009; Sauer 1955). Other Palmer amaranth seed dispersal mechanisms include movement by irrigation water and mammals, or agricultural practices such as plowing, mowing, and harvesting (Costea et al. 2004, 2005; Norsworthy et al. 2009). Female Palmer amaranth can produce 200,000 to 600,000 seed

plant^{-1} under ideal growing conditions such as non-shaded areas with temperatures between 36 and 46 C (Ehleringer 1983; Keeley et al. 1987). Continual, season-long emergence of the seed makes Palmer amaranth problematic (Keeley at al. 1987; Sellers et al. 2003).

After developing resistance to dinitroaniline herbicides in the 1980s, Palmer amaranth became one of the top 10 most troublesome weeds of cotton [*Gossypium hirsutum* (L.)] in South Carolina (Webster and Coble 1997). By 2009, Palmer amaranth was ranked as the most troublesome weed of cotton in nine of 10 states surveyed in the southern U.S. and among the top 10 most troublesome weeds in both soybean [*Glycine max* (L.) Merr.] and corn (*Zea mays* L.) production (Webster and Nichols 2012). Palmer amaranth was ranked as the most troublesome weed of cotton in seven of eight surveyed states, and the most troublesome weed of soybean in four of seven surveyed states surveyed in the southern U.S. in 2013 (Webster 2013).

Continual use of herbicides with the same MOA as the only means of weed control increases the probability of herbicide resistance (Holt 1992; Owen and Zelaya 2005). Multiple resistance, such as Palmer amaranth resistant to both glyphosate and acetolactate synthase (ALS) herbicides, is common (Nandula et al. 2012). Populations of Palmer amaranth have evolved resistance to five different herbicide modes of action including glyphosate, ALS inhibitors, dinitroanilines, triazines, and 4-hydroxyphenylpyruvate dioxygenase (HPPD) herbicides (Ward et al. 2013). Multiple resistance mechanisms, such as target site mutations affecting herbicide binding kinetics and amplification of target site genes or non-target site mechanisms such as metabolism and translocation are common (Rong et al. 2013).

Mesotrione was developed by Syngenta for the control of broadleaf and grass weeds in corn (Shaner 2014). It was derived from leptospermone, which is a compound found in the roots of red bottlebrush (*Callistemon citrinus* Curtis) (Cornes 2005). Red bottlebrush is a member of the Mytraceae family, which originates from Australia (Cornes 2005). In 1977, a biologist working at the Western Research Centre of the Stauffer chemical company in California noticed a lack of plants growing under red bottlebrush bushes (Cornes 2005). After testing, he concluded the compound responsible for the herbicidal activity was leptospermone (Cornes 2005). Mesotrione was derived from leptospermone 11 years later (Cornes 2005).

Mesotrione is a triketone herbicide, which provides selective weed control in corn, asparagus (*Asparagus officinalis* L.), blueberry (*Vaccinium pallidum* Ait.), lingonberry (*Vaccinium vitis-idaea* L.), black raspberry (*Rubus occidentalis* L.), red raspberry (*Rubus idaeus* L.), blackberry (*Rubus fruticosus* L.), bluegrass (*Poa annua* L.), perennial ryegrass (*Lolium perenne* L.), tall fescue [*Lolium arundinaceum* (S.J.) Darbyshire.] grown for seed, cranberry (*Vaccinium oxycoccos* L.), flax (*Linum usitatissimum* L.), oats (*Galega officinalis* L.), okra (*Abelmoschus esculentus* L.), pearl millet (*Pennisetum americanum* L.), rhubarb (*Rheum rhabarbarum* L.), sugarcane (*Saccharum officinarum* L.), and sorghum (*Sorghum bicolor* L.) (Anonymous 2014). Mesotrione can be applied PRE or POST, depending on the crop, for control of annual broadleaf and grass weeds (Shaner 2014). Mesotrione inhibits the enzyme HPPD, which indirectly blocks carotenoid synthesis (Shaner 2014). Carotenoids are necessary for photosynthesis and the protection of chlorophyll and plant cell membranes (Cornes 2005). The disruption of chlorophyll causes bleaching and tissue necrosis and eventual

plant death (Shaner 2014). Mesotrione may be absorbed by the leaves, shoots, or roots and is moved throughout the plant via xylem and phloem (Cornes 2005).

Fomesafen is a protoporphyrinogen oxidase (PPO) herbicide that can be applied PRE or POST-directed in cotton and PRE and POST in soybean, potato (*Solanum tuberosum* L.), dry bean (*Phaseolus vulgaris* L.), and snap bean (*Phaseolus vulgaris* L.) to control broadleaf weeds, including *Ipomoea* spp., *Amaranthus* spp., jimsonweed (*Datura stramonium* L.), wild mustard (*Sinapis arvensis* L.), black nightshade [*Solanum americanum* (P.) Mill], and *Ambrosia* ssp. (Shaner 2014). Fomesafen is absorbed through the roots when soil-applied and leaf tissue when foliar-applied, but translocation is limited within the plant (Fadayomi and Warren 1977; Ritter and Coble 1981; Unland et al. 1999). Fomesafen is xylem mobile (Shaner 2014). When foliar-applied, fomesafen causes leaves of susceptible plants to become chlorotic and eventually necrotic (Shaner 2014).

Prior to the development of PPO resistance in tall waterhemp [*Amaranthus tuberculatus* Moq.], PPO herbicides, such as aciflurofen, lactofen, and fomesafen, were applied in mixtures with ALS herbicides to control ALS-resistant biotypes (Gaeddert et al. 1997). In 2012, 16% of the total soybean hectares in Mississippi received an application of fomesafen (USDA 2014). In the U.S., tall waterhemp and common ragweed (*Ambrosia artemisiifolia* L.), were identified resistant to PPO herbicides in 2001 and 2005, respectively (Heap 2015).

Glyphosate is a nonselective herbicide commercialized in 1974 and used throughout the world to control a broad spectrum of weeds (Nandula et al. 2012). With the introduction of glyphosate-resistant (GR) crops in 1996, producers rapidly adopted

glyphosate for in-season weed control (Sammons et al. 2007). The widespread use of glyphosate has not only caused weed species shifts in crops but has also caused some weed populations to evolve resistance (Nandula et al. 2012). Thirty-two weed species worldwide are resistant to glyphosate, and nine of these are found in Mississippi (Heap 2015).

Glyphosate resistant Palmer amaranth was first reported in Georgia in 2004 when glyphosate was applied at 12X field rates and failed to control the biotype (Culpepper et al. 2006). Currently, GR Palmer amaranth can be found in 24 states throughout the U.S. (Heap 2015). Gaines et al. (2011) reported glyphosate resistance in Palmer amaranth was due to increased amounts of 5-enolpyruvylshikimate-3-phosphate synthase (EPSPS). Genomic copies of EPSPS enzymes appeared to act like a molecular sponge to absorb glyphosate and enabled resistant plants to continue functioning after application of glyphosate (Ward et al. 2013). Mohseni-Moghadam et al. (2013) further supports these findings in that the level of resistance in GR Palmer amaranth populations in New Mexico was due to the negative correlation between EPSPS copy numbers and shikimate accumulations. Studies in Kansas also documented resistance in Palmer amaranth to ALS where 14 different ALS-inhibiting herbicides, applied at 8X field rates, failed to provide adequate control (Gaeddert et al. 1997). Palmer amaranth populations resistant to both glyphosate and ALS herbicides have also been reported (Bond et al. 2010; Sosnoskie et al. 2011).

Infestations of Palmer amaranth hinder crop production in the southern U.S. (Bond et al. 2006). Furthermore, Palmer amaranth is the most troublesome weed of soybean in Mississippi (Webster 2013). Mesotrione was developed by Syngenta for the

control of broadleaf and grass weeds in corn (Shaner 2014). With the development of Syngenta's MGI (Mesotrione, Glufosinate, and Isoxaflutole) resistant soybean (Syngenta Crop Protection Wilmington, DE), research is needed to determine how to utilize mesotrione with current soybean herbicides. The objectives of this research were to (1) identify the most effective POST rate of mesotrione for Palmer amaranth control and (2) evaluate mesotrione alone and in mixtures with glyphosate, and/or fomesafen for control of GR Palmer amaranth.

Materials and Methods

Mesotrione Rate Evaluation

A greenhouse study was conducted in 2013 (33.42°N, -90.91°W) at the Mississippi State University Delta Research and Extension Center in Stoneville, MS, to identify the most effective POST rate of mesotrione for control of 5- and 10-cm Palmer amaranth. Palmer amaranth seed were surface-planted into 53- by 28-cm trays containing Metro-Mix (Sun Gro Horticulture 770 Silver Street Agawam, MA, U.S. 01001) potting media. Trays were sub-irrigated and placed in a greenhouse with 25/15 C (± 3 C) day/night temperatures and supplemented with light from sodium vapor lamps set to a 14-h photoperiod. Once emerged plants reached 1 cm in height, they were transplanted into separate 10-cm wide and 13-cm deep pots. Pots were sub-irrigated as needed throughout the study.

Treatments were arranged as a factorial of application timing and mesotrione rate within a randomized complete block design with four replications, and the study was repeated three times. Factor A was application timing and included Palmer amaranth at 5 cm with two to three leaves plant^{-1} and 10 cm with four to six leaves plant^{-1}. Factor B was

mesotrione (Callisto herbicide, Syngenta Crop Protection, 3411 Silverside Rd., Suite 100, Shipley building, Concord Plaza, Wilmington, DE, 19810) applied at 0 (nontreated control), 0.04, 0.07, 0.11, 0.14, and 0.17 kg ai ha^{-1}. All treatments containing mesotrione included COC (Agri-Dex, a 99% crop-oil concentrate, Helena Chemical Co., 5100 Poplar Ave., Memphis, TN 38137) at 1% (v/v). A treatment containing only COC at 1% (v/v) was included for comparison at both application timings. Treatments were applied when plants uniformly reached 5 and 10 cm. Treatments were applied in a spray chamber with an extended range even-flat fan spray nozzle (XR8002E TeeJet nozzles, Spraying Systems Co., P.O. Box 7900, Wheaton, IL, 60189) calibrated to deliver 140 L ha^{-1} at 220 kPa. After treatments were applied, plants were returned to the greenhouse.

Visual estimates of Palmer amaranth control were recorded at 7, 14, and 21 DAT on a scale of 0 to 100%, with 0% representing no control and 100% representing complete plant death. After the final visual evaluation, plant heights were measured to calculate height reduction for each treatment compared with the nontreated control in each application timing and replication using the formula:

$$\text{Height reduction (\%)} = \frac{\text{Height of nontreated control (cm)} - \text{Height of treated (cm)}}{\text{Height of nontreated control (cm)}} \times 100 \quad \{2.1\}$$

The nontreated control used in calculating height reduction was treated with mesotrione at 0 kg ha^{-1}. All aboveground portions of the plants were harvested by cutting at the soil level, oven-dried at 60 C for 7 d, and weighed to calculate biomass reduction for each treatment compared with the nontreated control in each application timing and replication using the formula:

$$\text{Biomass reduction (\%)} = \frac{\text{Dry weight of nontreated control (g)} - \text{Dry weight of treated plant (g)}}{\text{Dry weight of nontreated control (g)}} \times 100 \quad \{2.2\}$$

The nontreated control used in calculating biomass reduction was treated with mesotrione at 0 kg ha^{-1}.

The square roots of visual control estimates were arcsine transformed. Transforming the data did not improve homogeneity of variance based on visual inspection of plotted residuals; therefore, nontransformed data were used in analysis. Nontransformed data were subjected to ANOVA using the Mixed Procedure in SAS (Statistical software Release 9.3, SAS Institute Inc., 100 SAS Campus Drive, Cary, NC 27513-2414) with experiment as a random effect parameter (Blouin et al. 2011). No control of Palmer amaranth was observed with COC alone, so data from plots treated with COC alone and the nontreated control were excluded from analysis of visual control estimates. Type III Statistics were used to test the fixed effects of herbicide and application timing for control data 7, 14, and 21 DAT. Least square means were calculated and mean separation ($p \leq 0.05$) was produced using PDMIX800 in SAS, which is a macro for converting mean separation output to letter groupings (Saxton 1998). A regression trend was not detected for visual control and Palmer amaranth height data, so visual control data and height reduction were only subjected to ANOVA using the Mixed Procedure in SAS. Biomass reduction was regressed on mesotrione rate allowing for both linear and quadratic terms with coefficients depending on application timing (Golden et al. 2006). Nonsignificant ($P > 0.05$) terms were removed sequentially and the model was refit until a satisfactory model was obtained (Golden et al. 2006). The first derivative of the biomass reduction regression model was calculated in order to determine the mesotrione rate providing greatest biomass reduction of 5- and 10-cm Palmer amaranth.

Mesotrione Mixtures

A study to evaluate mesotrione alone or in mixture with glyphosate and/or fomesafen for GR Palmer amaranth control was conducted once in 2013 (33°24'29.1"N 90°55'31.6"W) and twice in 2014 (33°24'29.2"N 90°55'36.0"W) at the Mississippi State University Delta Research and Extension Center in Stoneville, MS. Soil was a Newellton silty clay (clayey over loamy, smectitic over mixed, superactive, nonacid, thermic Fluvaquentic Epiaquepts) with a pH of 6.85 and soil organic matter content of 1.6%. The experimental site was surface-seeded with GR Palmer amaranth each year to ensure uniform infestations. In site year three, the study area was surface-irrigated to stimulate Palmer amaranth germination and emergence.

The experiment was designed as a randomized complete block with a three factor factorial treatment arrangement and four replications. Factor A was mesotrione rates of 0, 0.05, 0.11, and 0.16 kg ha^{-1}. Factor B was fomesafen (Flexstar herbicide, Syngenta Crop Protection, 3411 Silverside Rd., Suite 100, Shipley building, Concord Plaza, Wilmington, DE, 19810) rates of 0 and 0.26 kg ai ha^{-1}. Factor C was glyphosate (Touchdown Total herbicide, Syngenta Crop Protection, 3411 Silverside Rd., Suite 100, Shipley building, Concord Plaza, Wilmington, DE, 19810) rates of 0 and 0.86 kg ae ha^{-1}. The study was initiated on June 5, 2013, and on May 22 and June 25, 2014. Each plot was 3 m wide and 9 m long, and treatments were applied once Palmer amaranth plants uniformly averaged 5 to 10 cm in height with a tractor-mounted sprayer calibrated to deliver 140 L ha^{-1} using extended range flat-fan spray nozzles at 248 kPa.

Visual estimates of Palmer amaranth control were recorded 7, 14, 21, and 28 DAT on the previously described scale. Following the final visual evaluation, Palmer amaranth

densities in two 1 m^2 quadrats in each plot were recorded. All aboveground plant parts in these quadrats were harvested by clipping at the soil surface. Plants from each quadrat were bagged separately, oven dried for 7 days at 60 C, and weight was recorded.

The square roots of visual control estimates were arcsine transformed. Transforming the data did not improve homogeneity of variance based on visual inspection of plotted residuals; therefore, nontransformed data were used in analysis. Nontransformed data were subjected to the Mixed Procedure in SAS with experiment as a random effect parameter (Blouin et al. 2011). Type III Statistics were used to test the three fixed effects of herbicide. Least square means were calculated and mean separation ($p \leq 0.05$) was produced using PDMIX800 in SAS, which is a macro for converting mean separation output to letter groupings (Saxton 1998).

Results and Discussion

Mesotrione Rate Evaluation

A main effect of mesotrione rate was detected for Palmer amaranth control 7 DAT; therefore, data were pooled across application timings (Table 2.1). At 7 DAT, Palmer amaranth control was greater with mesotrione at 0.14 and 0.17 kg ha^{-1} compared with mesotrione at 0.04 and 0.07 kg ha^{-1}. However, control was ≤ 66% 7 DAT with all rates. Schuster et al. (2008) reported mesotrione at 0.105 kg ha^{-1} controlled Palmer amaranth 64% 7 DAT.

An interaction of mesotrione rate and application timing was detected for Palmer amaranth control 14 and 21 DAT (Table 2.2). Control of 5-cm Palmer amaranth 14 and 21 DAT was comparable with mesotrione at 0.11 and 0.17 kg ha^{-1} (Table 2.2). At 14 DAT, mesotrione at 0.11 and 0.07 kg ha^{-1} provided similar control of 5-cm Palmer

amaranth; however, control was greater with mesotrione at 0.11 kg ha^{-1} compared with mesotrione at 0.07 kg ha^{-1} 21 DAT. Mesotrione at 0.14 kg ha^{-1} controlled 5-cm Palmer amaranth 95% 21 DAT. Jhala et al. (2014) reported 99% Palmer amaranth control 21 DAT with mesotrione at 0.105 kg ha^{-1} in greenhouse research. Mesotrione at 0.07 and 0.04 kg ha^{-1} controlled 5-cm Palmer amaranth ≤ 41% 21 DAT.

Control of 10-cm Palmer amaranth 14 DAT was greater with mesotrione at 0.17 kg ha^{-1} compared with mesotrione at 0.04 kg ha^{-1} (Table 2.2). The greatest control of 10-cm Palmer amaranth 14 DAT was only 57% with mesotrione at 0.17 kg ha^{-1}. Increasing mesotrione rate did not result in improved control of 10-cm Palmer amaranth 21 DAT. Mesotrione at 0.07 kg ha^{-1} only provided 53% control of 5-cm Palmer amaranth 14 DAT. Control of 10-cm Palmer amaranth 14 DAT with all rates of mesotrione was comparable to control of 5-cm Palmer with mesotrione at 0.07 kg ha^{-1}. By 21 DAT, control of 10-cm Palmer amaranth with any rate of mesotrione was comparable to control of 5-cm Palmer amaranth with mesotrione at 0.04 or 0.07 kg ha^{-1}. Herbicide efficacy decreases with increasing weed size (Tharp et al. 1999). Therefore, application timing was critical for optimizing control of Palmer amaranth with mesotrione alone.

An interaction of mesotrione rate and application timing was detected for Palmer amaranth height reduction 21 DAT (Table 2.2). Height reduction with treatments applied to 5-cm Palmer amaranth was greatest with mesotrione at 0.11, 0.14, and 0.17 kg ha^{-1}. No differences in height reduction were observed with mesotrione applied at the later timing. Similar to control data, height reduction with any rate of mesotrione applied to 10-cm Palmer amaranth was comparable to that with mesotrione at 0.04 kg ha^{-1} applied to 5-cm Palmer amaranth.

An interaction between intercepts for the 5- and 10-cm Palmer amaranth application timings was detected for biomass reduction 21 DAT. Mesotrione at 0.134 kg ha^{-1} provided the maximum biomass reduction at both the 5- and 10-cm application timings (Figure 2.1). At this rate, biomass reduction of 5- and 10-cm Palmer amaranth was 89 and 60%, respectively. Exceeding 0.134 kg ha^{-1} of mesotrione provided no added biomass reduction of 5- and 10-cm Palmer amaranth. This corresponds with control data in that exceeding the rate of 0.14 kg ha^{-1} of mesotrione did not provide added control of 5-cm Palmer amaranth 21 DAT (Table 2.2).

Mesotrione Mixtures

A three-way interaction of mesotrione, fomesafen, and glyphosate rates was detected for GR Palmer amaranth control at all four evaluations. Glyphosate-resistant Palmer amaranth control was ≤ 55% with all rates of mesotrione alone 7 DAT (Table 2.3). Control increased to ≥ 94% with all treatments containing fomesafen. Glyphosate alone provided 61% control 7 DAT, and the addition of any rate of mesotrione did not improve control 7 DAT.

Glyphosate-resistant Palmer amaranth control 14 DAT was ≤ 58% with mesotrione alone (Table 2.4). McMullan and Green (2011) reported 59% control of Palmer amaranth 15 DAT with mesotrione at 0.21 kg ha^{-1}. Mesotrione at 0.11 and 0.16 kg ha^{-1} provided similar control of GR Palmer amaranth at each evaluation after 7 DAT (Table 2.4, 2.5, 2.6). Mesotrione at 0.16 kg ha^{-1} controlled GR Palmer amaranth better than mesotrione at 0.05 kg ha^{-1} 14, 21, and 28 DAT. Fomesafen applied alone or in mixture with mesotrione and glyphosate controlled GR Palmer amaranth ≥ 93, 92, and 90% 14, 21, and 28 DAT, respectively (Tables 2.4, 2.5, 2.6) Bond et al. (2006) observed

fomesafen at 0.42 kg ha^{-1} control 47 populations of Palmer amaranth ≥ 96% 21 DAT. Sweat et al. (1998) reported Palmer amaranth control of 74 and 76% 21 DAT with fomesafen at 0.28 kg ha^{-1}. Increased GR Palmer amaranth control with fomesafen in this study was attributed to weed size at time of application and the abundance of rainfall during all three site years allowing for active growth of Palmer amaranth before and after application. Prostko (2011) and Steckel et al. (2012) reported that application timing was crucial in obtaining effective control of Palmer amaranth with diphenylether herbicides.

Glyphosate alone controlled GR Palmer amaranth 35% 28 DAT (Table 2.6). Whitaker et al. (2010) reported 10 to 23% control of GR Palmer amaranth 30 DAT with glyphosate at 1 kg ha^{-1}. Sosnoskie et al. (2011) reported 5% GR Palmer amaranth control 21 DAT when glyphosate was applied at 0.87 kg ha^{-1}. Applications of glyphosate alone and in mixtures with mesotrione at 0.05 kg ha^{-1} provided similar GR Palmer amaranth control 14, 21, and 28 DAT (Table 2.4, 2.5, 2.6). Palmer amaranth control was greater when glyphosate was combined with mesotrione at 0.11 and 0.16 kg ha^{-1} compared with glyphosate alone or in mixture with mesotrione at 0.05 kg ha^{-1} 14, 21, and 28 DAT. Control 21 and 28 DAT was similar with mesotrione at 0.16 kg ha^{-1} alone and glyphosate in mixture with mesotrione at 0.11 and 0.16 kg ha^{-1} (Table 2.5, 2.6).

A main effect of mesotrione rate was detected for plant density and dry weight 28 DAT (Table 2.7). No differences in plant density or dry weight were observed in plots treated with mesotrione at 0 and 0.05 kg ha^{-1}. However, plant densities and dry weight were lower in plots treated with mesotrione at 0.11 and 0.16 kg ha^{-1} compared to plots without mesotrione.

Results indicate that GR Palmer amaranth control with mesotrione alone was not commercially acceptable. Control of 5-cm Palmer amaranth was 95% 21 DAT with mesotrione at 0.14 kg ha^{-1} in the greenhouse (Table 2.2); however, when mesotrione was applied alone in the field, control of GR Palmer amaranth was ≤ 52% 21 DAT with all rates of mesotrione. Increased levels of control in the greenhouse was likely due to the controlled environment (Edwards 2013). To maximize control of GR Palmer amaranth when using mesotrione-based herbicide systems, mesotrione should not be used individually. No differences in control were observed when fomesafen, glyphosate, and mesotrione were applied in mixtures compared with fomesafen alone. However, Whaley et al. (2009) reported 98% control of smooth pigweed (*Amaranthus hybridus* L.) 6 weeks after application with mesotrione applied PRE at 0.15 kg ha^{-1}. Therefore, the ability to use mesotrione PRE in soybean will likely aid in maintaining GR Palmer amaranth control. Although this data indicates control was optimized with fomesafen alone, adding mesotrione to POST applications of fomesafen may provide increase residual control of GR Palmer amaranth and slow the incidence of resistance from occurring by applying more than one herbicide MOA.

Table 2.1 Palmer amaranth control 7 d after treatment (DAT) with different rates of mesotrione in a greenhouse study at Stoneville, MS, in 2013[a].

Treatment	Rate	Palmer amaranth control
	kg ai ha^{-1}	%
Mesotrione	0.04	45 c
Mesotrione	0.07	51 bc
Mesotrione	0.11	59 ab
Mesotrione	0.14	66 a
Mesotrione	0.17	66 a

[a] Data are pooled over three experiments and two application timings. Means followed by the same letter are not different at $p \leq 0.05$.

Table 2.2 Palmer amaranth control 14 and 21 d after treatment (DAT) and height reduction with different rates of mesotrione applied at two application timings in a greenhouse study at Stoneville, MS, in 2013[a].

		Palmer amaranth control					
		14 DAT		21 DAT		Height reduction	
Treatment	Rate	5-cm	10-cm	5-cm	10-cm	5-cm	10-cm
	kg ai ha^{-1}	%					
Mesotrione	0.04	48 de	36 e	40 d	31 d	45 bc	40 bc
Mesotrione	0.07	53 cde	40 de	41 d	42 d	50 b	26 c
Mesotrione	0.11	70 bc	50 de	65 bc	50 cd	70 a	37 bc
Mesotrione	0.14	90 a	43 de	95 a	38 d	89 a	36 bc
Mesotrione	0.17	81 ab	57 cd	77 ab	46 cd	82 a	39 bc

[a] Data are pooled over three experiments. Means followed by the same letter for each parameter are not different at $p < 0.05$.

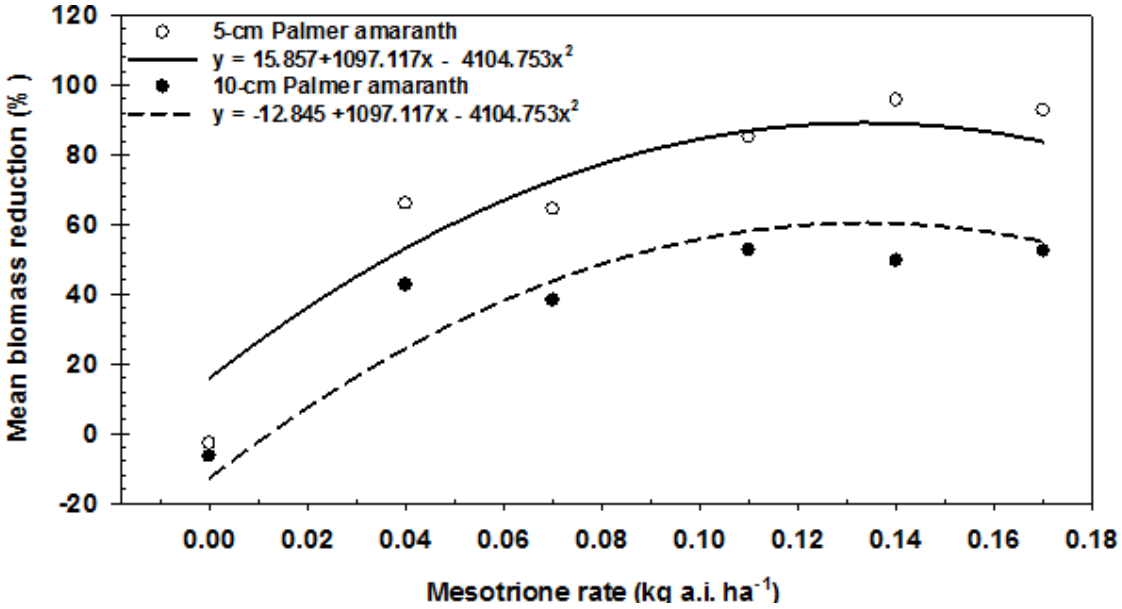

Figure 2.1 Palmer amaranth biomass reduction 21 d after treatment (DAT) with different rates of mesotrione applied at two application timings in a greenhouse study at Stoneville, MS, in 2013.

Table 2.3 Glyphosate-resistant Palmer amaranth control 7 d after treatment (DAT) with POST applications of mesotrione applied alone and in mixtures with fomesafen and glyphosate at Stoneville, MS, in 2013 and 2014[a,b].

		No glyphosate		Glyphosate	
Treatments	Rate	No fomesafen	Fomesafen	No fomesafen	Fomesafen
	kg ai ha^{-1}	%			
Mesotrione	0	0 f	94 a	61 bc	94 a
Mesotrione	0.05	40 e	96 a	57 c	96 a
Mesotrione	0.11	53 d	97 a	72 b	98 a
Mesotrione	0.16	55 c	97 a	70 bc	98 a

[a] Data are pooled over three experiments. Means followed by the same letter are not different at $p \leq 0.05$.

[b] No glyphosate and glyphosate represent applications at 0 and 0.86 kg ae ha^{-1}, respectively; No fomesafen and fomesafen represent applications at 0 and 0.26 kg ai ha^{-1}, respectively.

Table 2.4 Glyphosate-resistant Palmer amaranth control 14 d after treatment (DAT) with POST applications of mesotrione applied alone and in mixtures with fomesafen and glyphosate at Stoneville, MS, in 2013 and 2014[a,b].

		No glyphosate		Glyphosate	
Treatments	Rate	No fomesafen	Fomesafen	No fomesafen	Fomesafen
	kg ai ha^{-1}	%			
Mesotrione	0	0 f	94 a	50 d	93 a
Mesotrione	0.05	34 e	94 a	55 d	96 a
Mesotrione	0.11	48 d	98 a	75 bc	98 a
Mesotrione	0.16	58 cd	99 a	78 b	98 a

[a] Data are pooled over three experiments. Means followed by the same letter are not different at $p \leq 0.05$.

[b] No glyphosate and glyphosate represent applications at 0 and 0.86 kg ae ha^{-1}, respectively; No fomesafen and fomesafen represent applications at 0 and 0.26 kg ai ha^{-1}, respectively.

Table 2.5 Glyphosate-resistant Palmer amaranth control 21days after treatment (DAT) with POST applications of mesotrione applied alone and in mixtures with fomesafen and glyphosate at Stoneville, MS, in 2013 and 2014[a,b].

		No glyphosate		Glyphosate	
Treatments	Rate	No fomesafen	Fomesafen	No fomesafen	Fomesafen
	kg ai ha^{-1}	%			
Mesotrione	0	0 e	94 a	39 cd	92 a
Mesotrione	0.05	30 d	93 a	42 cd	95 a
Mesotrione	0.11	40 cd	97 a	66 b	97 a
Mesotrione	0.16	52 bc	98 a	71 b	97 a

[a] Data are pooled over three experiments. Means followed by the same letter are not different at $p \leq 0.05$.

[b] No glyphosate and glyphosate represent applications at 0 and 0.86 kg ae ha^{-1}, respectively; No fomesafen and fomesafen represent applications at 0 and 0.26 kg ai ha^{-1}, respectively

Table 2.6 Glyphosate-resistant Palmer amaranth control 28 days after treatment (DAT) with POST applications of mesotrione alone and in mixtures with fomesafen and glyphosate at Stoneville, MS, in 2013 and 2014[a,b].

		No glyphosate		Glyphosate	
Treatments	Rate	No fomesafen	Fomesafen	No fomesafen	Fomesafen
	kg ai ha^{-1}	%			
Mesotrione	0	0 e	93 a	35 cd	90 a
Mesotrione	0.05	29 d	93 a	38 cd	93 a
Mesotrione	0.11	37 cd	97 a	63 b	97 a
Mesotrione	0.16	49 bc	98 a	68 b	97 a

[a] Data are pooled over three experiments. Means followed by the same letter are not different at $p \leq 0.05$.

[b] No glyphosate and glyphosate represent applications at 0 and 0.86 kg ae ha^{-1}, respectively; No fomesafen and fomesafen represent applications at 0 and 0.26 kg ai ha^{-1}, respectively.

Table 2.7 Impact of mesotrione rate on density and dry weight of glyphosate-resistant Palmer amaranth 28 d after treatment (DAT) at Stoneville, MS, in 2013 and 2014[a].

Treatments		Plant density	Dry weight
	kg ai ha^{-1}	no.	g
Mesotrione	0	29 a	54 a
Mesotrione	0.05	20 ab	42 ab
Mesotrione	0.11	11 b	30 b
Mesotrione	0.16	10 b	26 b

[a] Data are pooled over three experiments, two glyphosate rates, and two fomesafen rates. Means within a column followed by the same letter are not different at $p \leq 0.05$.

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CHAPTER III

IMPACT OF LACTOFEN AND PLANTING DATE ON GROWTH, DEVELOPMENT, AND YIELD OF INDETERMINATE SOYBEAN

Abstract

A two-year study was conducted at the Mississippi State University Delta Research and Extension Center in Stoneville, MS, to determine the effect of lactofen and planting date on the growth, development, and yield of indeterminate soybean. In addition, an application timing study was conducted to determine soybean response to lactofen applied across multiple growth stages. Pooled across planting dates, soybean necrosis 14 d after treatment (DAT) with lactofen was similar to that in nontreated plots and those treated with only COC; however, soybean height 28 DAT was 11% lower in plots treated with lactofen compared with control plots. Pooled across herbicide treatments, soybean height was 17 and 32% greater 28 DAT with June 1 planting date compared with earlier plantings. The number of nodes at maturity was similar across soybean planting dates in plots treated with lactofen, but soybean plants in control plots produced more nodes with May 15 and June 1 plantings than with April 15 or May 1 plantings. In the lactofen timing study, soybean necrosis with lactofen was 5 to 28% 7 DAT and 5 to 23% 14 DAT. Soybean necrosis was greater 7 DAT with lactofen applied at V2 and R1 compared with applications > R2. At 14 DAT necrosis was greater with V2 applications than those > R2. Soybean height 14 d after last lactofen application, number

of nodes, and soybean yield were not affected by lactofen applied from V1 to R5. Early-season soybean growth can be altered with lactofen; however, it has little utility as a plant growth regulator to improve yields.

Nomenclature: Lactofen; Soybean, *Glycine max* L. Merr.

Key words: Planting date, Growth stage, Treatments

Introduction

Soybean were domesticated in China around 1500 to 1100 B.C. (Hymowitz 2004). The production of soybean spread through Japan, Indonesia, Philippines, Vietnam, Thailand, Malaysia, Myanmar, Nepal, and northern India where it was used in the development of several different foods (Hymowitz 2004). Soybean movement during this time resulted from establishment of sea and land trade routes, migration within China, and the rapid adoption of the soybean seed as a stable food source (Hymowitz 1990; Hymowitz and Newell 1980). Soybean was introduced to North America in 1765 (Hymowitz 2004).

In the Mississippi Delta, soybean production emerged in conjunction with cotton (*Gossypium hirsutum* L.) production (Snipes et al. 2005). Traditionally, soybean production in the area consisted of planting late maturity group (MG) V, VI, and VII cultivars in May and June (Heatherly 1998). Planting at this time consistently produced poor yields due to low rainfall amounts during soybean reproduction (Snipes et al. 2005). In an effort to minimize this problem, the Early Soybean Production System (ESPS) was developed (Heatherly 1998). Early Soybean Production System focuses on planting early-maturing MG IV and V soybean cultivars in April in an attempt to avoid seasonal drought during the reproductive growth stages (Bowers et al. 1998). A systematic

approach including seedbed preparation in the fall, application of preplant foliar herbicides for control of winter/spring weeds, and planting early-maturing cultivars into undisturbed, or stale, seed beds in April are integral steps for successful soybean production under ESPS (Heatherly and Spurlock 1999). By 2001, adoption of ESPS was widespread in MS, and is still used extensively today (Snipes et al. 2005).

Planting an earlier MG soybean in early April increases the probability of greater yields (Heatherly 1998). Adherence to ESPS not only results in greater yields, but the same cultivars are often shorter when planted early (T.W. Eubank unpublished data). Heatherly (1998) reported MG V, VI, and VII soybean cultivars planted in May or June resulted in pod set and fill during periods of low moisture and subsequently produced low yields. However, MG IV soybean planted mid-April encountered less adverse growing conditions and initiated pod set and pod fill earlier in the calendar year when weather conditions were more conducive for plant growth and development (Heatherly 1998).

Lactofen is a protoporphyrinogen oxidase (PPO) herbicide used for control of broadleaf weeds in cotton, peanut (*Arachis hypogaea* L.), and soybean **(**Hart and Roskamp 1998; Kapusta et al.1986; Wichert and Talbert 1993). Lactofen's primary target site is the chlorophyll synthesis pathway, where it inhibits the enzyme protoporphyrinogen oxidase **(**Fennimore and Hembree 2006**).** When applied POST, PPO herbicides are absorbed into leaf tissue resulting in the breakdown of cell membranes causing loss of turgor pressure and death (Fennimore and Hembree 2006).Tolerant crops exhibit bronzing on young, expanded leaves following treatment with lactofen (Shaner 2014). Injury from PPO herbicides may persist up to 21 d after treatment (Kapusta et al. 1986).

Lactofen often causes chlorosis, necrosis, or stunting of soybean, and soybean canopy closure can be decreased by 6% following an application (Edwards and Purcell 2005; Hart and Roskamp 1998; Kapusta et al. 1986; Wichert and Talbert 1993). Lactofen is currently labeled for white mold suppression in soybean (Anonymous 2013). A delay in canopy closure resulting from POST lactofen application reduced incidence of white mold due to increased air movement in both irrigated and nonirrigated soybean (Dann et al. 1999; Levene et al.1998).

Maximized and sustainable soybean yields are the ultimate goal of producers. After a popular press article reported a Missouri producer set a record soybean yield of 10,348 kg ha^{-1} in 2007 and partially credited routine lactofen applications with enhancing yield due to altering plant height and branching, it was questioned if this could be done on large scale production under growing conditions in the midsouthern U.S. (Anonymous 2008). Application of plant growth regulators (PGR) is common in multiple crops, including the use of mepiquat chloride to control plant growth and maximize yield and quality of cotton (Ren et al. 2013). Mepiquat chloride use is advantageous for controlling vegetative growth, which can be detrimental to fiber yield and quality if left uncontrolled (Constable 1995; Kerby 1985; Oosterhuis and Egilla 1996). Increased fruit abscission and reduced yield can be attributed to shade within the canopy as a result of excessive vegetative growth (Guinn 1974). Mepiquat chloride application results in shorter and more compact plants, lower leaf area index as a result of smaller leaf size, and early maturity (Kerby 1985; Reddy et al. 1990; York 1983). Some herbicides are used as PGRs. Glyphosate is applied at 0.04 to 0.18 kg ae ha^{-1} to stimulate sucrose accumulation

and suppress flowering in sugarcane (*Saccharum officinarum* L.) (Bennet and Montes 2003; Velini et al. 2010).

Lactofen is recommended at 0.22 kg ai ha^{-1} for POST control of hemp sesbania [*Sesbania herbacia* (P. Mill.) McVaugh], *Ipomoea* ssp, common ragweed (*Ambrosia artemisiifolia* L.), and *Amaranthus* ssp. in Mississippi soybean (Byrd 2015). Lactofen could alter soybean growth in a positive way; however, soybean injury can be severe when treated with lactofen (J. A. Bond, personal communication). Lactofen is currently labeled as a form of PGR in soybean (Anonymous 2013; Dann et al.1999; Levene et al. 1998) and soybean canopy can be altered using lactofen (Edwards and Purcell 2005; Hart and Roskamp 1998; Kapusta et al. 1986; Wichert and Talbert 1993). An altered plant canopy could potentially alter light interception and subsequently growth and development. Owen (2010) reported no soybean yield loss until 67% whole plant defoliation occurred at R3 and R5 stages. The objectives of this research were to (1) characterize the influence of lactofen and planting date on the growth, development, and yield of indeterminate soybean and (2) determine the soybean response to lactofen applied over a range of growth stages.

Materials and Methods

Planting Date Study

A field study was conducted in 2013 and 2014 (33°25'26.3"N 90°54'03.6"W) at the Mississippi State University Delta Research and Extension Center in Stoneville, MS, to evaluate the impact of planting date and lactofen application on the growth, development, and yield of indeterminate soybean. Soil was a Bosket silt loam (fine-loamy, mixed, active, thermic Mollic Hapludalfs) with a pH of 6.9 and soil organic

matter content of 0.89%. A MG IV soybean cultivar 'Progeny 4819LL' was planted at 370,000 seed ha^{-1} with a small-plot vacuum planter (John Deere 1730, Deer and Company, One John Deere Place, Moline, IL) at a depth of 4 cm. Individual plots consisted of four rows spaced 76 cm apart and measuring 60 m in length. The experimental sites were prepared by fall-disking, field cultivation, disk-hipping, and rolling. Flumioxazin (Valor SX, herbicide, Valent Corporation, 1600 Riviera Avenue, Suite 200, Walnut Creek, CA) at 0.07 kg ai ha^{-1} plus glyphosate (Roundup Powermax , herbicide, Monsanto Company, 800 North Lindbergh Blvd. St. Louis, MO) at 1.26 kg ae ha^{-1} was applied PRE to control winter/spring weeds. All plots were maintained weed free throughout the growing season and furrow-irrigated as needed (Byrd 2015).

The experimental design was a split-plot with four replications. Whole plots were planting dates and included targeted dates of April 15, May 1, May 15, and June 1. The April 15 date represented the Early Soybean Production System (Heatherly 1998). Soybean were planted within 2 d of the targeted planting date each site year. Subplots were herbicide treatments and consisted of a control that received no broadcast POST herbicide throughout the growing season, COC (Agri-Dex, a 99% crop-oil concentrate, Helena Chemical Co., 5100 Poplar Ave., Memphis, TN) at 1% (v/v), and lactofen (Cobra, herbicide, Valent Corporation, 1600 Riviera Avenue, Suite 200, Walnut Creek, CA) at 0.22 kg ha^{-1} plus COC at 1% (v/v). Herbicide treatments were applied with a tractor-mounted sprayer calibrated to deliver 140 L ha^{-1} at 248 kPa with extended range flat-fan spray nozzles (XR11002 TeeJet nozzles, Spraying Systems Co., P.O. Box 7900, Wheaton, IL) once soybean plants uniformly reached the V2 growth stage. The V2

growth stage was denoted by the emergence and full expansion of the second trifoliate leaf (Pedersen 2015).

Visual estimates of soybean necrosis 7 and 14 d after treatment (DAT) and biomass reduction 21 and 28 DAT were recorded on a scale from 0 to 100%, with 0% representing no injury or biomass reduction and 100% representing complete plant death. Photosynthetically active radiation (PAR) was determined with a PAR sensor (AccuPar model LP-80 PAR/LAI Ceptometer, Decagon Devices, 2365 NE Hopkins, Court Pullman, WA) 21 and 28 DAT to compare the level of available light above the plant canopy to that reaching the soil surface. Plant heights and number of nodes were recorded from 10 randomly selected plants in each plot 28 DAT and at soybean maturity. Plant heights were measured from the soil surface to the terminal bud. A soybean node is a lateral branch from the main stem which contains a fully emerged trifoliate leaf. Lodging was recorded prior to harvest on a scale from 1 to 10, where 1 represented a completely flattened plant and 10 represented a completely erect plant. Plots were harvested with a small-plot combine (Kincaid Equipment Manufacturing, 210 West First St., P.O. Box 400, Haven, KS), and soybean yield was adjusted to 13% moisture content.

The square roots of soybean necrosis and visual biomass reduction estimates were arcsine transformed. Transforming the data did not improve homogeneity of variance based on visual inspection of plotted residuals; therefore, nontransformed data were used in analysis. Nontransformed data were subjected to ANOVA using the Mixed Procedure in SAS (Statistical Analysis software Release 9.3, SAS Institute Inc., 100 SAS Campus Drive, Cary, NC) with year and replication (nested within year) as random effect parameter (Blouin et al. 2011). Type III Statistics were used to test the fixed effects of

herbicide and planting date. Least square means were calculated and mean separation ($p \leq 0.05$) was produced using PDMIX800 in SAS, which is a macro for converting mean separation output to letter groupings (Saxton 1998).

Application Timing Study

A field study was conducted at two sites (33°26'12.7"N 90°54'33.3"W) (33°25'26.3"N 90°54'03.6"W) in 2014 at the Mississippi State University Delta Research and Extension Center in Stoneville, MS, to determine the soybean response to lactofen applied over a range of soybean growth stages. Soil at site one was same as in the Planting Date Study. Soil at site two was a Commerce sandy loam (fine-silty, mixed, superactive, nonacid, thermic Fluvaquentic Endoaquepts) with a pH of 6.8 and soil organic content of 0.55%.

Site preparation and maintenance, soybean cultivar, and seeding information were as previously described in the Planting Date Study. Individual plots were 9 m in length and consisted of four rows spaced 76 cm apart at site one and 102 cm apart at site two. Both sites were planted April 15, 2014.

The experimental design was a randomized complete block with four replications. Lactofen at 0.22 kg ha^{-1} plus COC at 1% (v/v) was applied weekly at soybean growth stages ranging from V1 to R5. Treatments were applied with a CO_2-pressurized backpack sprayer and hand-held boom equipped with extended range flat-fan spray nozzles set to deliver 140 L ha^{-1} at 172 kPa.

Visual estimates of soybean necrosis were recorded 7, 14, 21, and 28 DAT using the previously described scale. Plant heights were measured 14 d after the last lactofen application and at soybean maturity as previously described in the Planting Date Study.

Number of soybean nodes was recorded at soybean maturity. A lodging score was recorded prior to harvest using the previously described scale. Plots were harvested with a small-plot combine and soybean yield was adjusted as previously described. All data was regressed; however, a regression trend was not detected, so analysis was conducted as previously discussed in the Planting Date Study.

Results and Discussion

Planting Date Study

No main effect or interaction was detected for soybean necrosis 14 DAT, height at maturity, number of nodes 28 DAT and at maturity, lodging, PAR values 21 and 28 DAT, or soybean yield (Table 3.1). However, a main effect or interaction was detected for soybean necrosis 7 DAT, biomass reduction at 21 and 28 DAT, soybean height 28 DAT, and soybean node count at maturity (Table 3.1). Soybean necrosis in plots treated with lactofen plus COC was 35% 7 DAT (Table 3.2). However, this was transient, and by 14 DAT, soybean necrosis with lactofen plus COC was similar to that in control plots or those treated with COC alone (Data not presented). Krausz and Young (2001) reported 35% necrosis 7 DAT when lactofen at 0.14 kg ha^{-1} was applied to soybean at the V4 stage, but visual necrosis was not present 28 DAT. A similar trend was observed with biomass reduction 21 DAT (Table 3.2). Plots treated with lactofen plus COC exhibited 19% biomass reduction compared with 0% reduction in those treated with COC alone (Table 3.2)

An interaction of herbicide treatment and planting date was detected for biomass reduction 28 DAT and number of nodes at maturity (Table 3.1 and 3.3). At 28 DAT, biomass reduction following lactofen plus COC was similar with April 15 and June 1

planting dates (Table 3.3). No difference in biomass reduction with lactofen plus COC was observed with planting dates of May 1, May 15, or June 1 (Table 3.3). Regardless of planting date, biomass reduction with lactofen plus COC was ≤ 19% 28 DAT (Table 3.3).

Main effects of herbicide treatment and planting date were significant for soybean plant height 28 DAT (Tables 3.1, 3.2, and 3.4). Heights in control plots and those treated with COC alone were similar 28 DAT; however, plant heights were reduced 10 to 11% in plots treated with lactofen plus COC (Table 3.2). Krausz and Young (2001) reported 20% reduction in soybean height 28 d following an application of lactofen at 0.14 kg ha^{-1} applied at V4. Soybean planted April 15, May 1, and May 15 exhibited comparable height 28 DAT (Table 3.4). Soybean heights in plots planted June 1 were 32, 26, and 17% greater compared with those planted April 15, May 1, and May 15, respectively (Table 3.4).

For each planting date, the number of nodes at maturity was similar following all herbicide treatments (Table 3.3). Plots treated with COC alone produced similar nodes $plant^{-1}$ with April 15, May 15, and June 1 planting dates; however, number of nodes was lower with May 1 compared with May 15 and June 1 planting dates (Table 3.3). Number of nodes was similar in plots treated with lactofen plus COC, across all planting dates (Table 3.3). The number of nodes in control plots was greatest with planting dates of May 15 and June 1 (Table 3.3). Therefore, the differences observed in nodes $plant^{-1}$ resulted more from planting date than herbicide treatment. Similar results were observed with multiple cultivars at Stoneville, MS, producing more nodes $plant^{-1}$ with May 15 compared with April 15 planting dates (T.W. Eubank, unpublished data).

No main effects or interactions of planting date or herbicide treatment were detected for soybean yield (Table 3.1). Heatherly (1998) reported that early-planted soybean produce greater yields compared with late-planted soybean due to periods of drought that are common later in the calendar year coinciding with reproductive development of late-planted soybean. Similar soybean yield across planting dates in this study can be attributed to the level of rainfall received and moderate air temperature during soybean reproductive development both years of the study (Tables 3.5 and 3.6). Wichert and Talbert (1993) also reported no reduction in yield with lactofen at 0.22 kg ha^{-1} applied at V2 soybean growth.

Application Timing Study

No effect of lactofen application timing was detected for soybean necrosis 21 and 28 DAT, plant height 14 d after last application, number of nodes at maturity, or soybean yield. Soybean necrosis 7 and 14 DAT and plant height at maturity varied with lactofen application timing. Soybean necrosis 7 DAT was 5 to 28% with greater necrosis from applications early in vegetative growth (Table 3.7). Kapusta et al. (1986) reported soybean was more sensitive to acifluorfen at V3 compared with the V5 soybean growth stage. Greater necrosis from applications early in vegetative growth could be attributed to less surface area of the plant at time of evaluation due to a smaller plant size and less leaf area (Jason A. Bond, personal communication). Reproductive development did not begin until week three of this study. Soybean necrosis with lactofen 7 DAT applied at V2 (week 2) and R1 was greater than when applied > R2 (week 4) (Table 3.7). The same trend was less apparent 14 DAT; however, necrosis was greater with V2 (Week 2) applications than those applied > R2 (Week 5) (Table 3.7). Mature soybean height was less following

lactofen applied at soybean growth stages of R2 and R3 (weeks 4 to 7) than those applied at V1 or ≥ R4 (Week 8) (Table 3.7).

Edwards and Purcell (2005) reported a reduction in yield of ultra-short-season maturity group 0 and II soybean cultivars when lactofen at 0.22 kg ha^{-1} was applied during reproductive stages from R1 to R5, but they attributed the decrease in yield to inadequate irrigation during pod fill. No effect of lactofen on soybean yield in this study can be attributed to the large amounts of rainfall throughout the growing season (Tables 3.5 and 3.6). Adequate soil moisture allowed for soybean to be actively growing before and after application; therefore, herbicidal effects were transient (Tables 3.5 and 3.6).

In conclusion, results indicate that lactofen applied across multiple application timings does not affect soybean yield. Lactofen applied at V2 in the Planting Date Study reduced soybean height early in the season; therefore, plant growth was altered by lactofen application. However, there was no difference in mature soybean height in plots treated with lactofen compared with control plots; therefore, soybean was able to recover from lactofen applied at V2. Within each planting date, there was no difference in the number of nodes following all herbicide treatments. Later planting dates of May 15 and June 1 produced greater numbers of nodes $plant^{-1}$ in control plots compared with April 15 and May 1 planted plots; therefore, differences in number of nodes at maturity resulted more from planting date than lactofen applications. Ultimately, early-season growth can be altered with lactofen; however, it has little utility as a plant growth regulator to improve soybean yields in midsouthern U.S. environments.

Table 3.1 Significance of main effects of herbicide treatment and planting date and interaction among the main effects for soybean necrosis, biomass reduction, soybean height, node count, lodging, photosynthetically active radiation (PAR) values, and soybean yield in planting date study at Stoneville, MS, in 2013 and 2014[a].

Effects	Soybean necrosis		Soybean biomass reduction		Soybean height		Soybean node count		Soybean lodging	PAR values		Soybean yield
	7 DAT	14 DAT	21 DAT	28 DAT	28 DAT	Maturity	28 DAT	Maturity	Maturity	21 DAT	28 DAT	Maturity
	p-value											
Herbicide	0.001	0.097	0.001	0.001	0.0001	0.878	0.686	0.283	0.930	0.260	0.055	0.896
Planting date	0.335	0.681	0.244	0.067	0.025	0.110	0.472	0.0001	0.357	0.594	0.082	0.067
Herbicide* planting date	0.301	0.774	0.237	0.031	0.073	0.595	0.569	0.035	0.886	0.450	0.276	0.912

[a] Column headings 7, 14, 21, and 28 designate evaluation intervals of 7, 14, 21, and 28 d after herbicide treatment.

Table 3.2 Soybean necrosis 7 d after treatment (DAT), biomass reduction 21 DAT, and soybean height 28 DAT with POST herbicide treatments at Stoneville, MS, in 2013 and 2014[a].

Herbicide treatments	Necrosis at 7 DAT	Biomass reduction at 21 DAT	Soybean height at 28 DAT
	%		cm
Control	0 b	0 b	44 a
COC	1 b	0 b	45 a
Lactofen + COC	35 a	19 a	40 b

[a] Data are pooled over four planting dates and two experiments. Means within a column followed by the same letter are not different at $p \leq 0.05$.

Table 3.3 Impact of herbicide and planting date on biomass reduction 28 d after treatment (DAT) and number of nodes at maturity at Stoneville, MS, in 2013 and 2014[a].

	Biomass reduction at 28 DAT			Number of nodes at maturity		
Planting date	Control	COC	Lactofen + COC	Control	COC	Lactofen + COC
	——————————%——————————			——————————no.——————————		
April 15	0 c	0 c	19 a	18 b	19 ab	19 ab
May 1	0 c	0 c	13 b	18 b	18 b	19 ab
May 15	0 c	0 c	13 b	20 a	20 a	20 a
June 1	0 c	0 c	15 ab	20 a	20 a	19 ab

[a] Data are pooled over two experiments. Means followed by the same letter for each parameter are not different at $p \leq 0.05$.

Table 3.4 Effect of planting date on soybean height 28 d after treatment (DAT) at Stoneville, MS, in 2013 and 2014[a].

Planting date	Soybean height at 28 DAT
	cm
April 15	36 b
May 1	39 b
May 15	44 b
June 1	53 a

[a] Data are pooled over three herbicide treatments and two experiments. Means followed by the same letter are not different at $p \leq 0.05$.

Table 3.5 Average weekly maximum and minimum air temperature, relative humidity weekly averages, and weekly total precipitation from mid-April to mid–October at Stoneville, MS, in 2013.

Dates[a]	Maximum air temperature	Minimum air temperature.	Maximum relative humidity	Minimum relative humidity	Precipitation
	C	C	%		cm
April 15-21	25	12	97	46	4.62
April 22-28	22	9	93	46	2.34
April 29-May 5	22	12	99	58	4.29
May 6-12	24	13	98	52	4.62
May 13-19	28	17	91	43	2.31
May 20-26	29	19	95	45	2.59
May 27-June 2	27	19	93	46	4.16
June 3-9	24	18	99	54	5.03
June 10-16	32	21	97	47	0.61
June 17-23	33	22	97	49	0.05
June 24-30	32	23	96	49	0.08
July 1-7	31	18	98	41	0.25
July 8-14	31	21	96	45	1.24
July 15-21	33	22	99	51	1.85
July 22-28	31	20	97	55	1.49
July 29-August 4	34	22	98	45	0.00
August 5-11	36	24	97	44	0.00
August 12-18	31	19	99	48	0.79
August 19-25	34	21	100	45	4.27
August 26-September 1	35	21	96	37	0.00
September 2-8	34	20	96	37	3.48
September 9-15	34	19	94	32	0.00
September 16-22	33	18	94	38	3.91
September 23-29	29	17	98	44	3.78
September 30-October 6	29	19	100	59	15.16
October 7-13	24	13	99	53	0.79

[a]Dates in weekly intervals from first planting date to end of harvest.

Table 3.6 Average weekly maximum and minimum air temperature, maximum and minimum relative humidity weekly average, and weekly total precipitation from mid-April to mid-October at Stoneville, MS, in 2014.

Dates[a]	Maximum air temperature	Minimum air temperature	Maximum relative humidity	Minimum relative humidity	Precipitation
	C	C	%		cm
April 14-20	20	8	96	44	7.34
April 21-27	27	12	99	35	6.83
April 28-May 4	25	12	93	36	1.68
May 5-11	29	17	92	42	1.65
May 12-18	25	14	99	46	3.40
May 19-25	31	18	92	37	0.51
May 26-June 1	26	18	94	57	11.05
June 2-8	31	22	97	54	0.64
June 9-15	30	20	96	50	6.22
June 16-22	33	23	92	43	0.00
June 23-29	32	23	96	53	6.91
June 30-July 6	32	20	95	49	0.03
July 7-13	3	22	96	49	3.07
July 14-20	29	19	98	57	9.04
July 21-27	32	20	98	55	0.03
July 28-August 3	30	21	96	52	0.61
August 4-10	33	22	98	49	0.76
August 11-17	31	19	98	48	3.07
August 18-24	34	21	97	46	0.53
August 25-31	33	22	97	45	2.77
September 1-7	33	22	99	54	0.97
September 8-14	31	19	97	54	1.55
September 15-21	32	19	97	41	0.10
September 22-28	30	16	96	30	0.00
September 29-October 5	29	17	95	39	2.46
October 6-12	29	17	97	51	6.86

[a]Dates in weekly intervals from first planting date to end of harvest.

Table 3.7 Impact of lactofen application timing on soybean necrosis 7 and 14 d after treatment (DAT) and heights at maturity at Stoneville, MS, in 2014[a].

Application timing in weeks	Growth stage at application	Necrosis		
		7 DAT	14 DAT	Mature height
		——————%——————		cm
1	V1	20 bc	14 abc	83 ab
2	V2	28 a	23 a	79 bcd
3	R1	24 ab	18 ab	79 bcd
4	R2	20 bc	13 bcd	77 d
5	R2	16 cd	14 a-d	76 d
6	R3	15 d	11 b-e	76 d
7	R3	15 d	10 cde	77 d
8	R4	15 d	10 cde	81 abc
9	R5	8 e	8 de	84 a
10	R5	5 e	5 e	84 a

[a] Data are pooled over two experiments. Means within a column followed by the same letter are not different at $p \leq 0.05$.

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