PYRASULFOTOLE & BROMOXYNIL RESPONSE IN GRAIN SORGHUM

by

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B.S., Northwest Missouri State University, 2008

A THESIS

submitted in partial fulfillment of the requirements for the degree

MASTER OF SCIENCE

Department of Agronomy
College of Agriculture

KANSAS STATE UNIVERSITY
Manhattan, Kansas

2011

Approved by:

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Abstract

Postemergent herbicide options for grain sorghum are limited and increasingly challenged by the development of herbicide resistant weeds. The herbicide pyrasulfotole & bromoxynil (P&B) was evaluated for potential use in grain sorghum and for control of a suspected HPPD-resistant Palmer amaranth population. Field experiments were conducted near Manhattan and Rossville, KS, to evaluate grain sorghum response to P&B with and without 2,4-D applied to growth stages from 1-leaf through the flag leaf stage and tankmixed with 2,4-D ester, amine, or dicamba applied to 3- and 6-leaf sorghum. The addition of 2,4-D ester did not reduce sorghum injury from P&B alone. Increasing the rate of P&B increased injury. Treatments applied to 1- and 4-leaf sorghum were injured the most. All P&B treated sorghum, regardless of timing, yielded 8 to 20% less than the untreated check. Pyrasulfotole & bromoxynil applied alone or with dicamba injured sorghum less than 2,4-D applied at 3- or 6-leaf. Increasing the rate from 140 to 280 g ha^{-1} 2,4-D amine or ester increased injury by 6 to 11%. Yields were lowest when P&B was applied with 2,4-D amine at 140 g ha^{-1} and 2,4-D amine or ester at 280 g ha^{-1} compared to all other treatments. Increasing the rate of growth regulator herbicides decreased yields by 8% and did not reduce crop injury from P&B alone. Greenhouse and field experiments were conducted to evaluate the response of two suspected P&B-resistant (R1 & R2) and one susceptible (S) Palmer amaranth population to P&B, atrazine, and tembotrione. Herbicides were applied when plants were 7 to 19 cm tall. The S population was controlled with less than field use rates. A resistance index (RI) of 4.8 to 11.0 was determined for R1 and R2 in greenhouse and field experiments. Tembotrione controlled 100% of S in all experiments, while providing 63 to 86% injury to R1 and R2 populations. Atrazine did not control the resistant

populations. Pyrasofotole & bromoxynil will be an valuable tool for weed control in sorghum, however, Palmer amaranth populations exist that will not be controlled.

Table of Contents

List of Figures

List of Tables

Acknowledgements

I would like to thank Dr. Curtis Thompson for all of his help, guidance, and continuous support over the past two years. He was always there to answer questions, help with field work, and provided direction for this project. I would also like to extend my appreciation to Dr. Anita Dille and Dr. Kraig Roozeboom for their help and serving on my graduate committee.

I would like to thank Cathy Minihan for helping me with all my research projects. I would also like to thank the Department of Agronomy, Bayer Crop Science, and the Kansas Grain Sorghum Commission for providing me with research materials and funding for the project.

Thank you to my parents and my sisters for all their understanding and support as I worked toward this degree.

Chapter 1 - Review of Literature

Grain sorghum (*Sorghum bicolor* L. Moench) is an important crop grown on 2.2 million ha in the United States, producing 8.8 million Mg of grain nationwide (Anonymous 2011a). Kansas ranks number one in the country for production at 4.3 million Mg. Grain sorghum production ranks third overall in the state behind corn (14.8 million Mg) and winter wheat (9.8 million Mg) (Anonymous 2011a). Grain sorghum is a warm season grass that can tolerate hot weather and drier climates, making it an ideal match for the environment found in Kansas and the central Great Plains region (Stahlman and Wicks 2000).

Weed control in sorghum has always been an issue from a production standpoint because summer annual weeds emerge at the same time sorghum is planted (Stahlman and Wicks 2000). Sorghum seed is smaller than the seed of other major spring planted crops, which results in slow early growth, preventing it from competing effectively with weed species, especially in cool, wet conditions (Vanderlip 1998). Summer annual weeds favor the same growing conditions as sorghum and compete for light, nutrients, and water. This weed competition can reduce grain yields up to 62% if not controlled (Burnside and Wicks 1967). Burnside and Wicks (1967) also observed that if weeds were controlled within one to three weeks after planting, no reductions in grain yield, stover accumulation, or heads per plant occurred. This indicates that adequate weed control is important during the first month of sorghum growth because yield potential is determined at this time (Vanderlip 1993).

Weeds reduce yields by intercepting light, nutrients, and water, but can also cause yield loss by decreasing grain quality, increasing insect and disease pressures, and increase harvesting difficulty through several factors such as reduced cylinder speed or clogging, and cause

gathering and threshing losses (Burnside et al. 1969; Moore et al. 2004; Zimdahl 1999). Studies conducted by Burnside et al. (1969) indicated large weed species can cause gathering losses of 7% and threshing loss of 1%. Moore et al. (2004) evaluated sorghum harvest efficiency with Palmer amaranth (*Amaranthus palmeri* (S) Wats) densities ranging from 0.07 to 1.2 plants m^{-1} row in Oklahoma. They observed an increase in grain moisture of 0.7 and 0.2% at two locations, and threshing loss from the combine of 11 kg ha^{-1} for each increase of 0.07 Palmer amaranth plants m^{-1} row. The weeds may delay harvest, cause grain moisture variability across a field, increase drying costs for storage, and reduce yields.

Integrated weed management strategies utilize mechanical, cultural, and chemical practices. Mechanical weed control is used in conventional and conservation tillage, but is irrelevant to producers who use no-till. Cultural weed control practices include crop rotation, narrower row spacing, increased plant density, delayed planting and hybrid selection (Stahlman & Wicks 2000). Many of these practices are dependent upon the environment in which sorghum is grown. Chemical control methods are a major component in sorghum weed control, making conservation and no-till cropping systems possible (Brown et al. 2004; Regehr 1998). A 2003 survey indicated 90% of the sorghum hectares in Kansas and 85% nationally were treated with herbicides (Anonymous 2011b). Fewer herbicides are registered for use in sorghum than corn or soybeans, which limit options for growers and current herbicides can result in undesirable crop injury or poor weed control (Stahlman and Wicks 2000).

Current herbicides used in sorghum production are applied early pre-plant (EPP), pre-emergence (PRE), or post-emergence (POST). Herbicides used in EPP or PRE applications include acetochlor, alachlor, atrazine, propazine, dimethenamid-P, carfentrazone, glyphosate, mesotrione, prosulfuron, and s-metolachlor (Brown et al. 2004, Thompson et al. 2011). POST

treatments include atrazine, bromoxynil, bentazon, carfentrazone, dicamba, fluroxypyr, halosulfuron, 2,4-D, metsulfuron, prosulfuron, and quinclorac (Brown et al. 2004; Thompson et al. 2011). Frequently, sorghum producers use an EPP or PRE residual herbicide + burndown herbicides followed by a POST herbicide if needed. Sorghum is typically grown in moisture-limited environments, and the lack of rainfall can lead to inadequate incorporation of residual PRE herbicides, providing poor weed control and necessitating earlier POST applications (Tapia et al. 1997). Atrazine is the most common herbicide used in Kansas, covering 79% of the sorghum hectares, followed by glyphosate at 38%, and s-metolachlor at 32%. The same trend occurs nationally with 70% of sorghum hectares treated with atrazine, followed by glyphosate at 27%, and s-metolachlor at 22% (Anonymous 2011b).

The following weed species have developed resistance to triazine herbicides in Kansas from repeated use of atrazine for weed control in both sorghum and corn downy brome (*Bromus tectorum* L.), kochia (*Kochia scoparia* (L.) Schrad.), common waterhemp (*Amaranthus rudis* Sauer), Palmer amaranth, and redroot pigweed (*Amaranthus retroflexus* L.) (Peterson 1999; Heap 2011). In addition to triazine resistance, resistance to acetolactate synthase (ALS)-inhibitor herbicides has also been documented for: Palmer amaranth, common waterhemp, Russian thistle (*Salsola tragus* L.), kochia, common cocklebur (*Xanthium strumarium* L.), shattercane (*Sorghum bicolor* L. Moench), and common sunflower (*Helianthus annuus* L.) in Kansas (Peterson 1999; Heap 2011). Glyphosate-resistant giant ragweed (*Ambrosia trifida* L.), common ragweed (*Ambrosia artemisiifolia* L.), marestail (*Conyza Canadensis* (L.) Cronq.), common waterhemp, and kochia has been reported in Kansas (Heap 2011). The occurrence of herbicide-resistant weeds has led to the need for integrated weed management strategies which include crop rotation, tillage, the use of directed herbicides, and tank mixing herbicides with different modes

of action (Peterson 1999; Abit et al. 2009). There is a demand for development of new herbicides to help manage herbicide-resistant weed populations. These herbicides must provide similar or greater weed control at a comparable price with herbicides currently used (Peterson 1999).

One possible alternative to improve weed control in sorghum would be to evaluate a POST herbicide with a mode of action that currently isn't registered for sorghum. The first 4-hydroxphenylpyruvate dioxygenase (HPPD)-inhibiting herbicide was discovered in the early 1980's, and the first product to become commercially available was sulcotrione in 1993 (Lee et al. 1997; Liebl and Seitz 2008). The HPPD enzyme is essential for the biosynthesis of plastoquinone (PQ) and tocopherols in plants (Freigang et al 2008). Plastoquinone is an important co-factor in electron transport during photosynthesis which shuttles electrons from photosystem II to photosystem I, where sunlight is converted into chemical energy (Freigang et al. 2008). Plastoquinone is also an important cofactor for desaturase enzymes such as phytoene, which are needed for carotenoid biosynthesis. Carotenoids protect the chloroplasts from excessive sunlight by quenching excess triplet energy, allowing chlorophyll to return to a ground energy state (Freigang et al. 2008). When the HPPD enzyme is blocked, PQ levels go down, increasing sensitivity to photosystem II-inhibitors, and stops carotenoid synthesis, leaving the plant with bleached foliage due to excess energy destroying chlorophyll, ultimately resulting in plant death (Freigang et al. 2008; Hess 2000).

Pyrasulfotole is a HPPD-inhibiting herbicide labeled in 2008 for use on the cereal crops of wheat (*Triticum aestivum* L), barley (*Hordeum vulgare* L), and triticale (*X Triticosecale* Wittmack) (Schmitt et al. 2008). Pyrasulfotole is premixed in a commercial formulation with bromoxynil at a concentration of 29 g L^{-1} of pyrasulfotole and 230 g L^{-1} of bromoxynil. Pyrasulfotole & bromoxynil provides good control of many of the annual broadleaf weeds that

commonly occur in wheat fields. The inclusion of bromoxynil or other photosystem II (PSII)-inhibitors with pyrasulfotole has a synergistic interaction (Abendroth 2006). Bromoxynil binds to the D1 protein at the same site where Q_B, a plastoquinone (PQ) molecule, binds. This displacement of Q_B from its binding site stops the transport of electrons to photosystem I (Hess 2000; Woodyard et al. 2009; Freigang et al. 2008). The necrosis exhibited by PSII-inhibitors is due to membrane damage caused by the inability of chlorophyll to transfer energy through the blocked Q_B site in PSII reaction centers. The singlet energy chlorophyll molecules start accumulating and are transformed into a triplet energy state (Hess 2000). Carotenoids usually dissipate small amounts of triplet chlorophyll, but because there is an elevated level, the system becomes overloaded and triplet chlorophyll forms lipid radicals that destroy the cell membrane via lipid peroxidation (Hess 2000).

Since pyrasulfotole indirectly reduces the amount of plastoquinone by inhibiting HPPD, PSII-inhibiting herbicides can easily bind to the D1 protein structures at lower rates. Carotenoid and tocopherol synthesis are stopped by pyrasulfotole, preventing the quenching of triplet chlorophyll molecules, which ultimately destroy membranes (Freigang et al. 2008; Hess 2000).

Most HPPD-inhibiting herbicides were developed for use in corn production systems and only mesotrione is registered for use in sorghum as a soil-applied herbicide. Mesotrione has been studied for use in grain sorghum as a POST treatment (Abit et al. 2009, 2010; Abit and Al-Khatib 2009). Abit et al. (2009) examined 85 sorghum hybrids to determine differences in susceptibility to mesotrione. Pioneer 84G62, Pioneer 85G01, and Triumph TR 438 were the most susceptible hybrids, whereas Dekalb DKS35-70, Frontier F222E, and Asgrow Seneca were the most tolerant. Abit and Al-Khatib (2009) also investigated sorghum absorption, translocation, and crop tolerance of mesotrione applied POST in a sensitive and a more tolerant sorghum

hybrid and detected little difference in absorption and translocation between the two hybrids. Abit also reported that differences in metabolism rate resulted in the differential injury response between the two hybrids.

Because mesotrione causes injury to sorghum applied POST, one can expect other HPPD inhibiting herbicides like pyrasulfotole, may also cause injury. It has been shown that herbicide injury symptoms may be mitigated with the addition of growth regulator herbicides. A study conducted by Brown et al. (2004) observed a safening response in sorghum when 2,4-D or dicamba were applied with metsulfuron to 3- or 4-leaf sorghum by reducing visual injury and reducing yield loss. Other growth regulators such as fluroxypyr or clopyralid did not reduce injury or provide adequate weed control. The results indicated that 2,4-D or dicamba can safen metsulfuron applied to sorghum without reducing efficacy, suggesting auxinic herbicides may reduce injury symptoms in sorghum from other herbicides.

Olson et al. (2011) evaluated sorghum response to pyrasulfotole & bromoxynil with and without the addition of growth regulators 2,4-D ester and dicamba at multiple locations throughout Kansas. The addition of 2,4-D ester or dicamba to pyrasulfotole & bromoxynil applied early POST reduced sorghum injury by 12 and 16% in Colby and 6 and 9% in Wichita compared to pyrasulfotole & bromoxynil applied alone. At the other five locations growth regulators did not reduce sorghum injury from pyrasulfotole & bromoxynil. When pyrasulfotole & bromoxynil with and without growth regulators were applied to late POST sorghum, little to no safening resulted from the addition of 2,4-D ester or dicamba (Olson et al. 2011).

Any safening effect that growth regulators could potentially have on herbicides such as pyrasulfotole & bromoxynil could be contradicted by the injury caused by the growth regulator itself (Phillips 1958). Growth regulator herbicides can cause sorghum injury through root growth

reductions, brace root malformations, leaf rolling, plant leaning, and green snap injury (Peterson et al. 2010). Besides causing visual injury to sorghum, flowering delays were observed when 2,4-D was applied before 7- to 9- leaves have developed (Wiese and Rea 1958). Phillips (1958) observed malformed brace root development with 2,4-D rates ranging from 280 to 1120 g ha^{-1}. The same study also reported different responses from formulations of 2,4-D and yield reductions from 2,4-D ester when applied to 9- to 11-leaf sorghum, however, the results were not consistent year to year. Additionally, yield reductions were observed with rates as low as 280 g ha^{-1} if applied during pollination. Penetration of leaf surfaces is faster with 2,4-D ester than amine formulations because ester formulations pass through lipid membranes more readily (Nice et al. 2004). This faster leaf absorption of 2,4-D ester provides better weed control than amine formulations, however crop injury responses may also increase.

Olsen et al. (2011) observed the best weed control from pyrasulfotole & bromoxynil + atrazine when applied to smaller broadleaved weeds. Kochia, common sunflower, tumble pigweed (*Amaranthus albus* L.), and redroot pigweed were controlled > 95% when applied at 4- to 6-leaf sorghum. Puncturevine control ranged from 89 to 96% with early POST applications, but declined significantly to 58 to 78% when applied to 30 to 40 cm tall sorghum. Control of Palmer amaranth was > 92% with early POST applications and with late POST applications declined to 90% at Wichita and 95% at Topeka locations, 89% at Manhattan and Tribune, and 66% at Colby locations.

Palmer amaranth belongs to the pigweed (*Amaranthus*) family. Several species of this family such as redroot pigweed, smooth pigweed (*A. hybridus* L.), Powell amaranth (*A. powellii* (S.) Wats.), spiny amaranth (*A. spinosus* L.), tumble pigweed, prostrate pigweed (*A. blitoides* (S.) Wats.), common waterhemp, and tall waterhemp (*A. tuberculatus* Sauer) commonly infest crop

acres across the Great Plains region (Horak et al. 1994). Palmer amaranth is distributed across the Southern half of the US and competes with crops like cotton, soybeans, corn, and grain sorghum for light, nutrients, water, and space.

Palmer amaranth can be difficult to distinguish from other pigweed species at the seedling stage, however, once the plant starts flowering, morphological differences between each species become evident and identification can be easier (Horak et al. 1994). The morphological characteristics of Palmer amaranth can be described as having ovate or egg-shaped leaves which are hairless and in an alternate leaf arrangement with petioles longer than the leaf blade, resembling a poinsettia-like appearance (Horak et al. 1994; Mayo et al.1995; Whitson et al. 1992). Palmer amaranth is only one of three weedy *Amaranthus* species that are dioecious and its inflorescence consists of leafless terminal spikes reaching up to 0.5 m in length (Horak and Peterson 1995; Barkley 1986). Female inflorescences have stiff bracts which are prickly to the touch and in contrast to the mature male inflorescences that do not have stiff bracts. These differences make it simple to distinguish a flowering plant's gender (Horak and Peterson 1995).

Under favorable conditions, Palmer amaranth has a long germination window, is fast growing, and a prolific seed producer which contribute to its weedy nature. Keeley et al. (1987) investigated the growth and seed production of Palmer amaranth and reported plants produced 62,000 to 600,000 seeds when grown without competition. Other studies examining crop-Palmer amaranth competition reported seed production of 140,000 to 514,000 seeds m^{-2} at 0.5 and 8 plants m^{-1} row, respectively, when Palmer amaranth emerged at the same time as corn. However, seed production declined to 1,800 and 91,000 seeds m^{-2} at the same densities when the weeds emerged at 3- to 6-leaf corn (Massinga et al. 2001). Interspecific and intraspecific competition

may explain the observed lower seed production in the crop-Palmer amaranth competition study, but seed production was still generous for one plant.

Palmer amaranth also has a long germination window that stretches from late spring to late summer in Kansas. Seed germination is regulated by the interaction of environmental conditions and the state of physiological readiness (Baskin and Baskin 1989). Each plant species has a specific range of environmental requirements necessary for germination (Gallagher and Cardina 1998). Steckel et al. (2004) reported Powell amaranth and Palmer amaranth consistently had 15 to 35% greater germination than tumble pigweed, prostrate pigweed, common waterhemp, redroot pigweed, smooth pigweed, spiny amaranth, and tall waterhemp. The pigweeds examined had a general increase in germination when exposed to alternating temperatures ± 40% of a mean constant temperature ranging from 5 to 35 C. Palmer amaranth germinated 83 and 73% when exposed to ± 40% alternating temperatures of 30 and 35 C, respectively. Other germination studies had similar trends but Palmer amaranth did not germinate when day/night temperatures were at or below 15/10 C, and that 35/30 C had the greatest germination with 43% (Guo and Al-Khatib 2003). Keeley et al. (1987) observed germination < 3% when day/night temperatures were 16/10 C, and germination gradually increased with increasing temperatures, reaching the highest germination at 38/32 C. These reports suggest that Palmer amaranth germination stops at low temperatures, which is why it is one of the last weeds to emerge in the spring.

Palmer amaranth is a fast growing plant capable of reaching heights of 2.7 m (Keeley et al. 1987). Palmer amaranth is capable of producing biomass in excess of 7.1 kg plant^{-1} when allowed to grow in a field setting from March to November (Keeley et al. 1987). During peak

growing conditions, Palmer amaranth grew 0.21 and 0.18 cm GDD^{-1}, when planted in June or July (Horak and Loughin 2000).

The rapid growth of Palmer amaranth can be attributed to its physiology (Ehleringer 1983). Diaheliotropic movement allows the plant's leaves to move throughout the day to remain perpendicular to the sun, which maximizes the light interception on the leaf surfaces to achieve high photosynthetic rates. This C_4 plant is capable of reaching photosynthetic rates of 81 µmol m^{-2} s^{-1} at a temperature of 42 C, which is one of the highest of all C_4 plants (Ehleringer 1983). This rate is nearly twice that of C_4 crops corn and grain sorghum and nearly four times the rate of C_3 crops cotton and soybeans (Prasad et al 2009; Kim et al. 2006; Steckel 2007).

Palmer amaranth competes with crops for light, nutrients, and water. Light is essential for all plant life and its energy is the driving force of photosynthesis, and regulates plant processes such as seed dormancy, germination, and flowering (Radosevich et al. 1997). Leaf area index (LAI) is the ratio of a plant's canopy leaf area to the area covered on the ground, and LAI indicates a plants potential to intercept light (Asner et al. 2003). Massinga et al. (2001) reported Palmer amaranth densities from 0 to 8 plants m^{-1} row decreases corn LAI by 2.9 m^2 m^{-2} when the weeds emerged at the same time as corn, however when weeds emerged in 6- and 7-leaf corn LAI, was reduced only slightly. Additionally, 1, 4, and 12 pigweeds m^{-2} caused reductions in sorghum LAI of 19, 35, and 63%, respectively (Graham et al. 1988). The reduction in corn LAI indicates that light absorption also will decrease, resulting in less photosynthesis and growth and ultimately decreasing yield.

There is limited research investigating the effects of Palmer amaranth-crop water use competition across different soils and environments. However, Massinga et al. (2003) investigated water use in irrigated corn and water use efficiency when competing with varying

densities of Palmer amaranth. Total seasonal water use increased with increasing Palmer amaranth densities with 2 plants m^{-1} row producing the greatest increase in water use of 4.1 cm. As Palmer amaranth densities increased from 2 to 8 plants m^{-1} row, a slower, gradual increase of 0.34 cm was observed. Volumetric soil water content was lowest in the upper 30 cm of soil; indicating Palmer amaranth and corn compete for available soil water in this area. Because this study was irrigated, soil water was maintained above 80% of total field capacity. Massinga et al. (2003) indicated that future work needs to investigate corn and Palmer amaranth dynamics at different of soil water levels.

Palmer amaranth is a problematic weed in corn. Massinga et al. (2001) observed 0.5 and 8 plants m^{-1} row reduced corn yield from 11 to 91% when Palmer amaranth emerged the same time as corn. When Palmer amaranth emerged in 4- to 7-leaf corn, yield loss was 7 to 35%. Later emerging Palmer amaranth plants have less effect on yield, which can be explained by the competitive height advantage corn had before Palmer amaranth starting growing.

Bensch et al. (2003) reported similar effect on yield from pigweed interference in soybeans. Palmer amaranth, common waterhemp, and redroot pigweed at 8 plants m^{-1} row reduced soybean yield 78, 56, and 39%, respectively when weeds emerged with soybeans, however, if the pigweeds emerged later, yield was not reduced. Klingaman and Oliver (1994) observed that when Palmer amaranth emerged at the same time as soybeans, weed densities of 0.33 to 10 plants m^{-1} row reduced soybean yield 17 to 68%.

Palmer amaranth is a major weed in cotton in the Southern US. Palmer amaranth reduces cotton biomass, yield, and fiber properties (Rowland et al. 1999; Morgan et al. 2001). A Texas study conducted by Morgan et al. (2001) examined the effect of Palmer amaranth density on cotton biomass and yield. Palmer amaranth at 0.1 to 1.1 plants m^{-1} row reduced cotton biomass

44 and 56% and reduced cotton lint yields linearly from 11 to 59%. Rowland et al. (1999) reported similar trends with each additional 0.1 Palmer amaranth m^{-1} row decreasing cotton yields 11.5%.

Palmer amaranth competition in sorghum also has been shown to affect yield, grain moisture, and grain seed loss through a machine harvester (Moore et al. 2004). Their results showed the effect of Palmer amaranth densities at 0 to 1.2 plants m^{-1} row, on sorghum yield, yield components, and harvest efficiency. Increasing Palmer amaranth density by 0.07 plant m^{-1} row increased grain moisture by 0.7 and 0.2% at two locations. Foreign matter increased by 67 and 3 kg ha^{-1} at two locations. Sorghum yield decreased 1.8 to 3.5% with each additional 0.07 Palmer amaranth plant m^{-1} row (Moore et al. 2004).

Because Palmer amaranth has been repeatedly reported to cause significant yield losses in major crops, the importance of controlling this weed in cropping systems is vital to maximize profitability of crop production (Massinga et al. 2003; Bensch et al. 2003; Morgan et al. 2001; Moore et al 2004). Many producers are shifting from a conventional tillage practice to conservation or no till systems to increase soil moisture, reduce erosion, and to improve soil physical properties (Wicks et al. 1988). Reduced or no tillage systems extensively rely on chemical control methods to control Palmer amaranth as well as other weeds in the field (Burnside et al. 1980). Monocropping and herbicide resistant crops, such as glyphosate tolerance, has led to repeated use of herbicides with the same modes of action on fields for many years (Regehr and Morishita 1989; Culpepper et al. 2006). Due to the obligate out-crossing of dioecious Palmer amaranth and the repeated selection pressure by using the same herbicide in cropping systems, herbicide-resistant accessions are expected to develop (Martin et al. 2000).

Currently, Palmer amaranth has been reported to be resistant to acetolactate synthase (ALS)-inhibitors, dinitroanilines, glycines, and photosystem II-inhibitors in thirteen states (Heap 2011).

Palmer amaranth resistance to glyphosate was first reported in Georgia cotton fields in 2006 and resistant populations are currently found in nine states in 2.4 million ha^{-1} (Heap 2011; Culpepper et al. 2006). Gaines et al. (2011) reported glyphosate resistance is caused by increases in the plant's genomic copy number of the enzyme 5-enlopyruvylshikimate-2-phosphate synthase (EPSPS) and that 30 to 50 EPSPS copies are needed to survive glyphosate rates ranging from 0.5 to 1.0 kg ha^{-1}. A study in Arkansas reported 12.5 kg ha^{-1} of glyphosate was needed to gain 95% control of a resistant biotype (Norsworthy et al. 2008). Glyphosate use is no longer economically feasible for weed control in many cotton fields, prompting implementation of new weed management strategies.

ALS-inhibiting herbicides have been used since 1982 and within nine years of herbicide commercialization, resistant Palmer amaranth populations were reported in Kansas (Heap 2011; Horak and Peterson 1995). Currently, there are three chemical groups of ALS inhibiting herbicides (sulfonylureas, imidazolinones, and sulfonamides) that have documented weed resistance in approximately 2.2 million ha^{-1} (Heap 2011). Repeated use of ALS-inhibiting herbicides has led to biotypes surviving eight times the normal use rate of imazethapyr and thifensulfuron (Horak and Peterson 1995). Sprague et al. (1997) reported biotypes cross-resistant to sulfonylurea and imidazolinone herbicides having > 3,700-fold resistance to thifensulfuron, > 1,900-fold resistance to chlorimuron, and > 2,800 fold resistance to imazethapyr compared to the susceptible biotype. Resistance to ALS-inhibiting herbicides is generally due to a single amino acid change in the active binding site in most species including Palmer amaranth (Woodworth et al. 1996).

Dinitroaniline resistant Palmer amaranth was confirmed in South Carolina in 1989, and currently infests 40,500 ha^{-1} (Heap 2011). Resistance was first suspected when poor control was observed with trifluralin, which had been used 24 consecutive years in the same cotton field (Gossett et al. 1992). Gossett et al. (1992) reported that trifluralin at 3.4 kg ha^{-1} was required to obtain satisfactory Palmer amaranth control, which is six times the normal use rate. Other dinitroaniline herbicides provided poor control of the resistant biotype with four times the recommended use rates.

Atrazine is PS II inhibitor and is one of the oldest herbicides currently used on many hectares in the United States. Atrazine-resistant Palmer amaranth was confirmed in Kansas in 1995 and resistant biotypes are estimated to have spread to 400,000 ha^{-1} in Georgia, Kansas, and Texas (Heap 2011). Although resistant biotypes have been confirmed, producers continue to use triazine herbicides because of the low cost and the high level of weed control it still provides (Peterson 1999). Resistance to triazine herbicides is reported to be an altered binding site where the herbicide attaches to Q_b in the D1 protein to prevent the transfer of electrons within the PS II reaction center (Foes et al. 1998; Hess 2000). This altered binding site is shown to reduce overall electron transfer within the PS II reaction center. The resistant trait is passed through the female plant only because the gene that encodes the altered amino acid occurs in the chloroplasts (Foes et al. 1998).

Interspecific hybridization between Palmer amaranth and other *Amaranthus* species can occur, resulting in the spread of nuclear-inherited herbicide resistant traits (Wetzel et al. 1999; Franssen 2001). Wetzel et al. (1999) reported that a resistant-Palmer amaranth male and a susceptible common waterhemp female or the reciprocal, can cross-pollinate and transfer the single dominant ALS-inhibitor resistant trait. The hybrid offspring can back-cross to either

species, spreading ALS-inhibitor resistance to a susceptible population. Although Palmer amaranth and common waterhemp overlap in geographic distribution, Franssen et al. (2001) speculated that hybridization occurs at low frequencies but can contribute to the spread of herbicide resistance. Rayburn et al. (2005) analyzed the genome size of several key *Amaranthus* species and observed Palmer amaranth to have the smallest genome size of 0.95 picograms (pg) and tall waterhemp has the largest genome size of 1.4 pg. Additionally both species have 32 mitotic chromosomes. Hybrid plants from Palmer amaranth and tall waterhemp would have an intermediate genome size. This information can be useful to identify hybrid populations where these two species grow together.

Currently, there are no reported cases of Palmer amaranth resistance to HPPD-inhibitors, however, tall waterhemp was confirmed resistant to HPPD-inhibitors in Illinois and Iowa recently (Hausman et al. 2011; McMullan and Green 2011). In Illinois, repeated use of mesotrione, topramezone, or tembotrione for seven years in continuous seed corn production resulted in a population developing 10-fold resistance to mesotrione based on greenhouse experiments (Hausman et al. 2011). Additionally, tembotrione and topramezone provided inadequate control and tank-mixing atrazine with mesotrione, tembotrione, or topramezone increased efficacy. Preliminary research on the same waterhemp population by Tranel et al. (2011) indicated a non-target resistance mechanism inherited by a single-dominant gene and has shown to be resistant to triazines and ALS-inhibiting herbicides.

A field in Iowa that has been in a seed corn-soybean crop rotation for the last 10 years started to display inadequate tall waterhemp control with a mesotrione + atrazine tank-mix when treated POST in seed corn. HPPD-inhibitors were used every year seed corn was grown. Greenhouse experiments conducted by McMullan and Green (2011) calculated resistance index

(RI) values of 7.7, 10.5, and 28.2 when treated with mesotrione, atrazine, or thifensulfuron, respectively, to achieve 50% control compared to a susceptible biotype. Similar trends were observed with that of Hausman et al. (2011) where atrazine, mesotrione, tembotrione, and topramezone provided poor weed control in the suspected resistant population.

Because pyrasulfotole & bromoxynil is a newer herbicide, little information is known about the response it can cause in grain sorghum or its overall fit in sorghum production. The objectives of this research were to (1) determine grain sorghum response to applications of pyrasulfotole & bromoxynil when applied to 1-leaf through flag leaf sorghum, (2) determine if the addition of growth regulator herbicides to pyrasulfotole & bromoxynil provides safening to grain sorghum injury and yield components when applied to two growth stages, and (3) determine if a population of Palmer amaranth is resistant to pyrasulfotole & bromoxynil, tembotrione, or atrazine.

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Chapter 2 - Grain Sorghum Response to Pyrasulfotole & Bromoxynil Applied to Different Growth Stages

ABSTRACT

Weed control in grain sorghum can be challenging for Kansas producers because many competitive weeds emerge at the same time as grain sorghum. The limited moisture conditions grain sorghum is grown in can cause poor incorporation of residual PRE herbicides which leads to poor weed control. Additionally, sorghum POST herbicide program options are fewer than options for other spring planted crops and herbicide resistant weeds further reduce herbicide options. Pyrasulfotole & bromoxynil was evaluated as a potential new herbicide in grain sorghum. Field experiments were conducted during 2009 and 2010 near Manhattan and Rossville, KS, to evaluate grain sorghum response to postemergence pyrasulfotole & bromoxynil, a 1:8 ratio, at 246 and 492 g ha^{-1} with and without 2,4-D ester at 140 g ha^{-1}. Pyrasulfotole & bromoxynil was applied to sorghum at six timings from 1-leaf to the flag leaf growth stages. Treatments included atrazine + ammonium sulfate at 0.56 + 2.8 kg ha^{-1}. All treatments injured sorghum at all application timings in all experiments. Sorghum injury increased as rate of pyrasulfotole & bromoxynil increased. Injury diminished 28 days after treatment (DAT) to 9% or less for all timings except at Rossville where sorghum treated at the 1-leaf in 2010 and 4-leaf sorghum in both years with 492 g exhibited injury of 20, 18 and 24%, respectively. 2,4-D did not reduce injury or increase grain yield compared to the pyrasulfotole & bromoxynil applied alone. The addition of 2,4-D reduced plant heights 0 to 6 cm when applied to 1- to 7-leaf sorghum, and increased plant height 0 to 7 cm when applied to 10- to flag-leaf sorghum compared to pyrasulfotole & bromoxynil alone. Pyrasulfotole & bromoxynil delayed flowering 1.3 to 5 d when applied to 1- and 4-leaf sorghum in all experiments. Sorghum treated

at the flag leaf had 23 to 34% fewer seeds panicle^{-1} than the untreated check. Kernel weights were 6 to 30% heavier from sorghum treated at 10- to flag leaf, while sorghum treated at the 7-leaf stage provided the lowest kernel weights in all experiments. Yields were reduced at all application timings from 7 to 19% in 2009 compared to the untreated check. At Rossville in 2010, flag leaf treated sorghum yielded 30% less than the untreated check Although sorghum injury occurred at all applications and grain yield reductions were observed in 2009 in a weed-free environment, the potential remains for pyrasulfotole & bromoxynil to be used in grain sorghum, to manage annual broadleaf weed pressure.

Nomenclature: Pyrasulfotole & bromoxynil; sorghum, *Sorghum bicolor* (L.) Moench. SORBI

Key words: growth stages, herbicide timing, HPPD-inhibiting herbicides, injury, yield components.

INTRODUCTION

Kansas is the number one grain sorghum (*Sorghum bicolor* (L.) Moench) producing state in the United States, with 4.3 million Mg of grain harvested in 2010, accounting for 49.5% of the total national production (Anonymous 2011a). Annual precipitation in Kansas ranges from 38 cm in Western Kansas to 117 cm in Eastern Kansas (Anonymous 2011b). This limited moisture together with high temperatures create challenges for crop producers. Grain sorghum is a C_4 plant and is considered to be relatively drought tolerant, able to perform better than crops such as corn or soybeans when temperatures are high and water is limited, making it a suitable choice in arid regions (Stahlman and Wicks 2000; Unger 1994).

Weed control in sorghum is challenging because many summer annual weeds favor similar growing conditions as sorghum. Sorghum seed is smaller than the seed of other major spring planted crops, which results in slow early growth, preventing it from competing effectively with weed species, especially in cooler conditions (Vanderlip 1998). Weeds compete with sorghum for light, nutrients, water, and space (Grichar et al. 2005; Stahlman and Wicks 2000; Graham et al. 1988). Sorghum-weed competition reduces grain yield and quality, increases harvest difficulty, insect pressure, and disease pressure (Moore et al. 2004; Zimdahl 1999; Burnside et al. 1969). Burnside and Wicks (1967) indicate that mixed weed populations left uncontrolled reduced yield 62%, and if sorghum was maintained weed-free for four weeks after planting, no yield reductions occurred. Grain sorghum leaf area index (LAI) was decreased by 19, 35, and 65% with densities of 1, 4, and 12 pigweeds (*Amaranthus spp.*) m^{-2}, respectively (Graham et al. 1988).

Weed control is achieved through integrated weed management which utilizes cultural, mechanical, and chemical practices; however chemical control is especially important in reduced

or no-tillage systems (Brown et al. 2004; Regehr 1998). A 2003 survey indicated 90% of the sorghum hectares in Kansas and 85% nationally were treated with herbicides (Anonymous 2011c). Sorghum has fewer herbicides registered for use than corn or soybeanss which limits options for growers and increase the risk of crop injury or poor weed control (Stahlman & Wicks 2000).

Current herbicides used in sorghum production are applied early pre-plant (EPP), pre-emergence (PRE) or post-emergence (POST). Herbicides used in EPP or PRE applications include acetochlor, alachlor, atrazine, propazine, dimethenamid-P, carfentrazone, glyphosate, mesotrione, prosulfuron, and s-metolachlor (Brown et al. 2004, Thompson et al. 2011). POST treatments include atrazine, bromoxynil, bentazon, carfentrazone, dicamba, fluroxypyr, halosulfuron, 2,4-D, metsulfuron, prosulfuron, and quinclorac (Brown et al. 2004; Thompson et al. 2011). Frequently, sorghum producers use EPP or PRE residual herbicides and or combined with burndown herbicides followed by a POST herbicide if needed. Sorghum is typically grown in moisture-limited environments and the lack of rainfall can lead to inadequate incorporation of residual PRE herbicides resulting in poor weed control and necessitating earlier POST applications (Tapia et al. 1997). This stresses the importance of an effective POST herbicide program if the PRE herbicides fail.

Growth regulator herbicides such as 2,4-D can cause crop injury through root growth reductions, brace root malformations, leaf rolling, plant leaning, and green snap injury (Peterson et al. 2010). Besides causing visual injury to sorghum, flowering delays can occur if 2,4-D is applied before 7- to 9- leaves have developed (Wiese and Rea 1958). Phillips (1958) observed malformed brace root development when 2,4-D was applied at 280 to 1120 g ha^{-1}. Phillips (1958) also reported that 2,4-D ester caused more yield reduction than 2,4-D amine when applied

to 9- to 11-leaf sorghum. The results were not consistent year to year. Additionally, yield reductions were observed with rates as low as 280 g ha^{-1} if applied during pollination. The ester formulation penetrates leaf surfaces faster than the amine formulation since ester can pass through lipid membranes more readily (Nice et al. 2004). The fast leaf absorption of 2,4-D ester provides better weed control than amine, however crop injury responses may also increase (Stahlman and Wicks 2000).

Atrazine is the most common herbicide used in Kansas and is applied to 79% of the sorghum hectares (Anonymous 2011c). Heavy reliance and repeated use of triazine herbicides in corn and sorghum cropping systems have resulted in the development of triazine-resistant populations of Palmer amaranth (*Amaranthus palmeri* (S.) Wats.), common waterhemp (*A. rudis* Sauer), redroot pigweed (*A. retroflexus* L.), downy brome (*Bromus tectorum* L.), and kochia (*Kochia scoparia* (L.) Schrad.) in Kansas (Heap 2011; Peterson 1999). Additionally, acetolactate synthase (ALS)-inhibitor resistant biotypes of Palmer amaranth, common waterhemp, Russian thistle (*Salsola tragus* L.), kochia, common cocklebur (*Xanthium strumarium* L.), shattercane (*Sorghum bicolor* (L.) Moench), and common sunflower (*Helianthus annuus* L.) have been reported (Heap 2011; Peterson 1999). Herbicide-resistant weeds have led to changes in production practices such as increased tillage, crop rotation, tank-mixing herbicides with different modes of action, and the use of directed herbicides (Peterson et al. 1999; Abit and Al-Khatib 2009).

Because many weed species are developing resistance to herbicides commonly used in sorghum, a solution is needed to gain control of these resistant weeds. One possible solution to control the resistant weeds is to use a hydroxyphenylpyruvate dioxgenase (HPPD)-inhibitor herbicide called pyrasulfotole. HPPD is an important enzyme for the production of

plastioquinone (PQ), which is involved in transporting electrons in the electron transport chain of photosynthesis, and is a cofactor for the synthesis of carotenoids (Freigang et al. 2008). Carotenoids are needed to quench triplet chlorophyll and singlet oxygen otherwise they can form lipid radicals and destroy chloroplast membranes (Freigang et al. 2008; Hess 2000). A prepackaged mix of pyrasulfotole & bromoxynil in a 1:8 ratio, controls many summer annual broadleaved weed species such as pigweeds, kochia, and common sunflower (Olson et al. 2011). Pyrasulfotole & bromoxynil became commercially available in 2008 and is currently labeled for use in wheat (*Triticum aestivum* L), barley (*Hordeum vulgare* L), and triticale (*X Triticosecale* Wittmack).

A synergistic weed control relationship occurs between HPPD-inhibitors and photosystem II (PSII)-inhibitors like atrazine or bromoxynil (Abendroth et al. 2006). Photosystem II-inhibitors bind to the D1 protein at the same site where Q_B, a PQ molecule binds. This displacement of Q_B from its binding site stops the transport of electrons within the photosynthetic electron transport chain (Hess 2000; Woodyard et al. 2009; Freigang et al. 2008). This displacement causes an accumulation of singlet chlorophyll, triplet chlorophyll, and singlet oxygen (Hess 2000; Abendroth et al. 2006). Carotenoids usually dissipate small amounts of triplet chlorophyll but due to the greater amount of triplet chlorophyll present and the reduced amount of carotenoids resulting from the application of a HPPD-inhibitor, the system becomes overloaded and the triplet chlorophyll forms lipid radicals, destroying membranes by lipid peroxidation (Abendroth et al. 2006; Hess 2000).

Most HPPD-inhibiting herbicides are currently registered for use in corn production systems and only mesotrione is labeled for use in sorghum as a PRE herbicide. Abit et al. (2009) examined 85 sorghum hybrids to determine differences in susceptibility to mesotrione. Pioneer

84G62, Pioneer 85G01, and Triumph TR 438 were the most susceptible hybrids, whereas Dekalb DKS35-70, Frontier F222E, and Asgrow Seneca were the most tolerant. Abit and Al-Khatib (2009) also investigated sorghum absorption, translocation, and crop tolerance of mesotrione applied POST to sensitive and more tolerant sorghum hybrids and concluded that there were few differences in absorption and translocation between the two hybrids. Abit concluded that differences in the metabolism rate resulted in the differential injury between the two hybrids.

Because mesotrione applied POST to sorghum causes injury, other HPPD-inhibiting herbicides like pyrasulfotole, may also cause injury. Adding other herbicides with HPPD-inhibitors my reduce injury symptoms in sorghum. It has been shown that injury symptoms from certain herbicides may be mitigated with the addition of growth regulator herbicides. A study conducted by Brown et al. (2004) observed a safening response in sorghum when 2,4-D or dicamba was applied with metsulfuron to 3- or 4-leaf sorghum by reducing visual injury and reducing yield loss. Other growth regulators such as fluroxypyr or clopyralid did not reduce injury or provide adequate weed control. The results of this study indicate that 2,4-D or dicamba can safen metsulfuron applied to sorghum without reducing efficacy, suggesting auxinic herbicides may reduce injury symptoms in sorghum from other herbicides.

Olson et al. (2011) evaluated sorghum response to pyrasulfotole & bromoxynil with and without the addition of the growth regulators 2,4-D ester and dicamba at multiple locations throughout Kansas. The addition of growth regulators with pyrasulfotole & bromoxynil applied early POST reduced sorghum injury by 12 and 16%, respectively, in Colby. Injury was also reduced by 6 and 9%, respectively, in Wichita compared to pyrasulfotole & bromoxynil applied alone. At the remaining locations, growth regulators did not affect sorghum injury from pyrasulfotole & bromoxynil. When pyrasulfotole & bromoxynil were applied with dicamba to

late POST sorghum injury was reduced by 7% at Garden City compared to pyrasulfotole & bromoxynil applied alone. No safening resulted from the addition of 2,4-D ester or dicamba at the remaining locations.

The objectives of this study were to evaluate the response of grain sorghum to pyrasulfotole & bromoxynil applied to six vegetative growth stages, to determine if timing of application affects crop injury, grain yield, and yield components, and to determine if the addition of 2,4-D ester increases crop safety to pyrasulfotole & bromoxynil.

MATERIALS AND METHODS

Field experiments were conducted at Kansas State University Agronomy Department fields at Ashland Bottoms 8 km south of Manhattan, KS and at the Kansas River Valley Experiment Field 2 km east of Rossville, KS in 2009 and 2010. At Manhattan, the soil was a Reading Silt loam (Fine-silty, mixed, superactive, mesic, Pachic Argiudolls) with a pH of 6.3 and 2.3% organic matter in 2009, and in 2010 the soil was a Wymore silty clay loam (fine, montmorillonitic, mesic, Aquic Argiudolls) with a pH of 6.0 and 2.5% organic matter. The soil at Rossville was a Eudora silt loam (Coarse-silty, mixed, superactive, mesic, Fluventic Hapludolls) with a pH of 6.5 and 1.3% organic matter in 2009 and 2010. Nitrogen at 247 kg ha^{-1} was applied and incorporated into the soil prior to planting both years at Manhattan. Nitrogen at 150 kg ha^{-1}, phosphorus at 58 kg ha^{-1}, and potassium at 67 kg ha^{-1} were applied and incorporated into the soil prior to planting both years at Rossville. In 2010 at Rossville, the experiment was irrigated July 27, August 4, and August 9 with 1.9, 2.5, and 2.5 cm of water, respectively. No irrigation was applied in 2009.

Pioneer grain sorghum hybrid ‘84G62’ was planted in 76 cm rows at 154,000 seeds ha^{-1} on June 4, 2009 and May 24, 2010 at Rossville, and 143,000 seeds ha^{-1} on June 18, 2009 and

June 3, 2010 at Manhattan. Abit et al. (2009) reported that '84G62' was among the most susceptible sorghum hybrids to the HPPD-inhibitor, mesotrione and may also be more susceptible to pyrasulfotole & bromoxynil. Plots were 3.1 m wide by 8.2 m long and were maintained weed-free with a pre-emergence application of s-metolachlor 1.41 kg ha^{-1} + atrazine 1.12 kg ha^{-1}, followed by hand weeding throughout the growing season when needed. Herbicide treatments were applied with a CO_2 pressurized backpack sprayer equipped with TurboTee[1] 11002 flat fan nozzles calibrated to deliver 140 L ha^{-1} at 234 kPa. Weather data at each application time are shown in Tables 2-1, 2-2, and 2-3.

Herbicide treatments were applied to 1-, 4-, 7-, 10-, 13-leaf collar, and flag leaf sorghum. The four herbicide treatments applied at each timing were pyrasulfotole & bromoxynil 246 g ha^{-1}; pyrasulfotole & bromoxynil 492 g ha^{-1}; pyrasulfotole & bromoxynil 246 g ha^{-1} + 2,4-D ester 140 g ha^{-1}; and pyrasulfotole & bromoxynil 492 g ha^{-1} + 2,4-D ester 140 g ha^{-1}. Pyrasulfotole & bromoxynil applied at 246 g ha^{-1} is considered the use rate in grain sorghum and the inclusion of 2,4-D was to determine if any crop safening occurred. All treatments included atrazine + ammonium sulfate at 0.56 + 2.8 kg ha^{-1}. The inclusion of ammonium sulfate was to condition the carrier as required by the pyrasulfotole & bromoxynil label. Atrazine was included with each treatment due to the known synergistic weed efficacy that exists between HPPD inhibitors and PS II inhibitors (Abendroth et al. 2006). A non-treated control plot was included for comparison.

Sorghum injury was rated visually 7, 14, 21, and 28 days after treatment (DAT) based on a scale of 0 to 100%, where 0% = no injury and 100% = dead. Plant population was determined by counting plants in 8.2 m of row 10 d after sorghum emergence. Flowering date was recorded when 50% of the main stems reached half-bloom and is presented as days to flowering from crop

emergence. Grain sorghum height was determined during grain fill from three measurements in each plot. Heads were counted during grain fill from 8.2 m of row. Sorghum grain yield was determined by mechanical harvesting the middle two rows in each plot. Moisture content was determined with a grain analyzer[2], and grain yield was adjusted to 14% moisture. Seed samples were collected from each plot and 1000 kernel weight was determined. The number of seeds per panicle was also calculated.

The experimental design for each location and year was a randomized complete block with a factorial arrangement of six application timings and four herbicide treatments, replicated four times. Data were subjected to analysis of variance using PROC MIXED in SAS[3], and means were separated using Fisher's protected LSD at $P \leq 0.05$. Days to flowering, plant height, grain yield, 1000 kernel weight, seeds per head, and heads per plant, were then compared to the untreated check using contrasts to determine if significant differences were observed.

RESULTS AND DISCUSSION

Injury

Location by year interactions occurred for sorghum injury, so the data were analyzed and presented separately for each location and year. All treatments injured grain sorghum at all application timings when observed at 7 and 14 DAT (Table 2-4). Injury symptoms from pyrasulfotole & bromoxynil consisted of general leaf chlorosis, necrotic leaf margins and leaf tips, necrotic spotting on treated leaves, red-brownish banding on treated leaves, and stunting. Rosales-Robles (2005) reported that bromoxynil caused leaf burn injury in sorghum. Growth regulator injury was evident in treatments applied with 2,4-D ester and was characterized by leaf rolling, plant leaning, and some minor lodging as described by Brown et al. (2004) and Phillips (1958).

Data were pooled over the addition of 2,4-D because no 2,4-D by application timing by herbicide rate interactions occurred 7 and 14 DAT, which is contrary to previous reports by Brown et al. (2005) (Table 2-4). Generally, the higher rate of pyrasulfotole & bromoxynil at 492 g ha^{-1} caused greater injury than the lower rate of 246 g ha^{-1}. Treatments applied to 4-, 7-, and 10-leaf sorghum caused the greatest injury 7 DAT. Pyrasulfotole & bromoxynil applied at either rate caused the least injury to flag leaf sorghum at Manhattan 2009 and 2010, and Rossville in 2009. Injury was consistently the greatest when 492 g ha^{-1} pyrasulfotole & bromoxynil was applied to 4- and 10-leaf sorghum at both locations and years. Observed injury at 7 DAT ranged from necrotic leaf margins and stunting with 1- and 4-leaf application timings, to necrotic leaf tips, necrotic leaf margins, and red-brownish banding across the leaf blade of treated leaves in 7-, 10-, and 13-leaf timings, to necrotic spotting and necrotic leaf margins on flag leaf treatments.

At Manhattan in 2009, 14 DAT no rate by application timing interactions were observed for sorghum at 14 DAT (Table 2-4). At Manhattan 2010, injury was greatest on 4-leaf treated sorghum and increased with the higher rate of pyrasulfotole & bromoxynil. Flag leaf treated sorghum was injured the least compared to all other application timings at Manhattan 14 DAT. At Rossville in both 2009 and 2010, pyrasulfotole & bromoxynil applied to 1-, 4-, and 10-leaf sorghum had more injury as rate increased. The injury observed with the 1-leaf application timing in 2010, and 4-leaf application timing in both 2009 and 2010 was stunting. Stunting was most severe when sorghum was treated with the higher rate of pyrasulfotole & bromoxynil. Treatments applied to 1-, 4-, and 7-leaf sorghum produced new leaves by 14 DAT minimizing the appearance of necrosis, leaf burn, and the 2,4-D injury symptoms diminished. Treatments applied to 10- and 13-leaf sorghum still had evidence of necrotic spotting on leaves, necrotic leaf margins, and red-brownish banding on leaves, because treated leaves on the plants were not

covered up by new foliage. In general, injury decreased from 7 to 14 DAT and new growth appeared to be unaffected, except at Rossville in 2010 where stunting and injury ratings from the 1- and 4-leaf application timings increased from 7 to 14 DAT.

A rate by application timing interaction for injury occurred 21 and 28 DAT across all locations and years (Table 2-5). At Manhattan in 2009 no differences were observed among treatments 21 DAT. Injury from stunting remained evident 21 DAT in 1- and 4-leaf sorghum at Manhattan 2010 and Rossville 2009 and 2010. The most injury observed 21 DAT ranged from 20 to 30% which occurred when 492 g ha^{-1} pyrasulfotole & bromoxynil was applied to 4-leaf sorghum at Manhattan 2010 and at Rossville 2009 and 2010. The 246 g ha^{-1} rate of pyrasulfotole & bromoxynil at 7-, 10-, and 13-leaf sorghum had 8% or less injury 21 DAT for all years and locations. Pyrasulfotole & bromoxynil applied at 492 g ha^{-1} to flag leaf sorghum provided 12 to 15% injury and caused necrotic foliage. Injury for 13- and flag leaf sorghum remained unchanged as time progressed because no new foliage emerged.

Injury continued to decline by 28 DAT and all treatments had 6% or less injury at Manhattan 2009 (Tables 2-4 and 2-5). At Manhattan in 2010, injury was greatest when 13-leaf sorghum was treated with 492 g ha^{-1} pyrasulfotole & bromoxynil 28 DAT (Table 2-5). All other treatments had 7% injury or less. Injury declined by 28 DAT both years at Rossville. At Rossville in 2009 all treatments except 492 g ha^{-1} pyrasulfotole & bromoxynil applied to 4-leaf sorghum had 6% or less injury. This suggests that growers could expect minimal injury from 246 g ha^{-1} pyrasulfotole & bromoxynil applied to all vegetative growth stages by 28 DAT. At Rossville 2010, evidence of stunting was still observed in sorghum treated at the 1- and 4-leaf timings, and 492 g ha^{-1} of pyrasulfotole & bromoxynil injured sorghum 20 to 24%. By the time anthesis was initiated, stunting was negligible but red-brownish banding on leaves, necrotic leaf

margins, and necrotic leaf tips were still noticeable from the flag leaf sorghum treatments. Broadcast POST applications to flag leaf sorghum are generally discouraged especially with treatments containing 2,4-D due to the risk of head blasting and yield reduction (Wiese and Rea 1958; Peterson et al. 2010). In addition, atrazine is only labeled for use in sorghum up to 31 cm tall (Thompson et al. 2011).

At Manhattan 2009, 2010, and Rossville 2010, an application timing by 2,4-D interaction occurred for observation of injury at 14 DAT (Table 2-6). Few differences in injury were observed with the addition of 2,4-D ester to pyrasulfotole & bromoxynil compared to pyrasulfotole & bromoxynil applied alone, however, 2,4-D ester did reduce injury symptoms when applied to 10-leaf sorghum in Manhattan 2009, 7-leaf sorghum Manhattan 2010 and Rossville 2009, and 4- and 10-leaf sorghum at Rossville in 2010. This agrees with Olson et al. (2011) who observed inconsistent safening with 2,4-D ester or dicamba applied with pyrasulfotole & bromoxynil compared to pyrasulfotole & bromoxynil alone. These differences were generally 4% or less, suggesting little response to injury occurred with the addition of 2,4-D. At Manhattan in 2009, injury was reduced by 2% with the addition of 2,4-D applied to 10-leaf sorghum 21 DAT (Table 2-6). All other application timings indicate a slight injury increase with the addition of 2,4-D to pyrasulfotole & bromoxynil, however, all injury was less than 7%, indicating no large differences were observed with the inclusion of 2,4-D ester.

A three way interaction of rate by timing by 2,4-D addition occurred for injury observations 21 and 28 DAT at Manhattan in 2010 (Table 2-7). The 4-leaf treated sorghum was injured the most 21 DAT with pyrasulfotole & bromoxynil, especially when treated with the 492 g ha^{-1} rate. Sorghum treated at the 7-leaf stage had no necrosis or stunting visible by 21 DAT. Treatments applied to 1- or 4-leaf sorghum were stunted, while 10-leaf treatments had visible

red-brownish banding and some necrosis on the lower leaves right below the upper canopy by 21 DAT. The 1- and 4-leaf treated sorghum injury declined from 21 to 28 DAT and all sorghum had 8% or less injury by 28 DAT except for pyrasulfotole & bromoxynil 492 g ha^{-1} with and without 2,4-D applied to 13-leaf sorghum which had 12% injury.

Plant Height

Plant height reductions were affected by timing and the addition of 2,4-D thus data were pooled over pyrasulfotole & bromoxynil rates and means were compared to each untreated check (Table 2-8). At Manhattan in 2009, the addition of 2,4-D applied to 7-leaf sorghum and all herbicide treatments applied to 13- and flag leaf sorghum reduced final plant height by 6 to 7 cm relative to the untreated check. At Manhattan in 2010, all herbicide treatments applied to 4- through 10-leaf sorghum had reduced plant height relative to the untreated check. Pyrasulfotole & bromoxynil applied to 13- and flag leaf sorghum reduced plant height, however, the addition of 2,4-D ester resulted in plants 3 to 6 cm taller. This was consistent with that observed at Rossville in 2009 such that pyrasulfotole & bromoxynil applied to 13-leaf sorghum produced the shortest plants at 129 cm, which was 5 cm shorter than the untreated check. The addition of 2,4-D ester applied to the 13-leaf sorghum resulted in 7 cm taller plants than pyrasulfotole & bromoxynil applied alone. At the earlier applications, the addition of 2,4-D ester to pyrasulfotole & bromoxynil caused similar or reduced plant height, especially when applied to 4- or 7-leaf sorghum. These results were inconsistent with Brown et al. (2004) who observed height increases with growth regulator herbicides when applied with metsulfuron to 3- to 4-leaf sorghum. When 2,4-D ester was applied with pyrasulfotole & bromoxynil to 10-, 13-, and flag leaf sorghum, a similar or increased height occurred, especially in 10-leaf sorghum at Rossville

in 2009 and both years at Manhattan. These results are variable but suggest that 2,4-D may safen pyrasulfotole & bromoxynil when sorghum is treated at later stages.

Pyrasulfotole & bromoxynil rate by timing interactions occurred for plant height at Manhattan in 2009 (Table 2-9). Both rates of pyrasulfotole & bromoxynil applied to 7-, 13-, and flag leaf sorghum were shorter than the untreated check. Additionally, pyrasulfotole & bromoxynil at 492 g ha^{-1} applied to 13- and flag leaf sorghum reduced plant height compared to pyrasulfotole & bromoxynil at 246 g ha^{-1} applied at the same stage.

Days to Flowering

Days to flowering from sorghum emergence had no observed interactions but differences between application timings were significant, thus data was averaged over herbicide treatments (Table 2-10). A trend across both years and locations indicate that pyrasulfotole & bromoxynil applied to 1- or 4-leaf sorghum delayed flowering 1 to 5 d compared to the untreated check. Flowering delays of 0.7 to 1.7 d were also observed with 10- to flag leaf treated sorghum at Manhattan in 2010. This delay in flowering with 10-leaf treated sorghum was not observed at Manhattan in 2009 or Rossville in either year. Delays in flowering by 1 or 2 d may not seem significant and generally is not a concern, however, a stress event such as high temperatures, limited moisture, or early frost could cause additional problems with pollination, grain fill, or attaining physiological maturity.

Yield Components

In August 2010, a storm event occurred at Manhattan producing strong winds which caused lodging to sorghum plants in the experiment. The storm damage was so severe that yield components could not be measured accurately thus yield components results from only Manhattan 2009, Rossville 2009 and 2010 were determined.

Heads per plant was only affected by timing of application thus data were averaged over herbicide treatments (Table 2-10). Heads per plant were reduced when treatments were applied to 4-leaf sorghum at the 2009 Rossville experiment compared to the untreated check. Herbicides applied at the other locations and application timings did not affect heads per plant when compared to the untreated check. A study conducted by Gerik and Neely (1987) suggests that tillers provide little contribution to grain yield when plant populations were above 12.3 m^{-2}. In these experiments plant populations ranged from 13.1 to 14.2 plants m^{-2}, thus the number of heads per plant observed may not directly reflect grain yield.

Number of seeds per panicle was only affected by application timing thus data were pooled over herbicide treatments for all experiments (Table 2-10). Applications made to 7-leaf or later sorghum at Manhattan and Rossville 2009 or to 10-leaf or later sorghum at Rossville in 2010 reduced seeds per panicle compared to the untreated check. Additionally, applications to 4-leaf sorghum at Rossville in 2009 had reduced seeds per panicle compared to the untreated check. At all locations and years, 1- and 4-leaf treatments had the greatest number of seeds per panicle. The exception was Rossville in 2010 where treatments applied to 7-leaf sorghum produced 168 more seeds per panicle than the untreated check. No other increase was observed with any treatment at any location or year.

Sorghum 1000 kernel weight was only affected by timing of application (Table 2-11). Sorghum treated at the 10-leaf or later timings at Manhattan 2009 and both years at Rossville had greater kernel weights than the untreated check. In addition, 1- to 7-leaf treated sorghum had greater kernel weights than the untreated check at Rossville in 2010. In 2009 at both locations, treatments applied to 10- to flag leaf sorghum had greater kernel weights than sorghum treated at the earlier stages. Sorghum treated at the 7-leaf timing had the lowest kernel weight in all

locations and years. These low kernel weights were not less than the kernel weight of the untreated check. The increase in kernel weight reflects the decreased seeds per panicle observed at these application timings and demonstrates the elasticity of sorghum yield components to try to maintain stable grain yields (Table 2-10).

Yields were reduced by all application timings at Manhattan and Rossville in 2009 compared to the untreated check (Table 2-11). Pyrasulfotole & bromoxynil applied to 7- and 13-leaf sorghum had the lowest yield while 1- and 10-leaf treated sorghum produced the greatest yields at Manhattan in 2009. At Rossville in 2009, pyrasulfotole & bromoxynil had the lowest yields when applied to 4- and 7-leaf sorghum and were highest yielding when treated at 1-, 10-, and 13-leaf stages. In Rossville 2010, the 7-leaf application was the highest yielding with 6471 kg ha^{-1} which is contrary to that observed in 2009 at both locations. This yield was 40% greater than that from flag leaf timing and was more than the untreated check. In Rossville 2010, only the flag leaf treated sorghum yielded less than the untreated check.

At Manhattan in 2009, a timing by 2,4-D interaction indicates that the treatment of pyrasulfotole & bromoxynil + 140 g ha^{-1} 2,4-D ester applied to 4-leaf sorghum reduced heads per plant by 0.07 compared to pyrasulfotole & bromoxynil applied alone at the same stage or the untreated check (Table 2-12). Seeds per panicle was reduced when pyrasulfotole & bromoxynil was applied with and without 2,4-D ester to 7-, 10-, 13-, and flag leaf sorghum compared to the untreated check (Table 2-12). The flag leaf treatments produced the fewest seeds per panicle. The addition of 2,4-D to pyrasulfotole & bromoxynil did not affect seeds per panicle compared to pyrasulfotole & bromoxynil applied alone regardless of the application timing.

At Manhattan 2009, a timing by 2,4-D interaction showed that the addition of 2,4-D provided similar or decreased 1000 kernel weight when applied to 1-, 4-, and 7-leaf sorghum

compared to pyrasulfotole and bromoxynil applied alone (Table 2-12). For 10-, 13-, and flag leaf timings, the addition of 2,4-D caused similar or increased 1000 kernel weights. The increased 1000 kernel weights to 13- and flag-leaf sorghum was likely due to reduced seeds per panicle caused by tank-mixing 2,4-D with pyrasulfotole & bromoxynil and demonstrates the yield compensation ability of sorghum.

At Manhattan in 2009, the addition of 2,4-D ester applied to 7-leaf sorghum reduced yield by 16% compared to pyrasulfotole & bromoxynil applied alone at the same stage (Table 2-12). All treatments at all application timings reduced yields compared to the untreated check except for pyrasulfotole & bromoxynil applied alone to 1-leaf sorghum.

The addition of 2,4-D ester to pyrasulfotole & bromoxynil applied to 10-, 13-, and flag leaf sorghum increased plant height and 1000 kernel weight, yet final grain yield was not affected compared to pyrasulfotole & bromoxynil applied alone at these stages (Tables 2-8 and 2-12). In general, treatments applied to 13- and flag leaf sorghum should be avoided due to the increased risk of sterile spikelets, called head blasting, even though it was not observed in these experiments (Peterson et al. 2010). Growth regulator herbicides are commonly associated with this phenomena, and the late applications in this study demonstrate pyrasulfotole & bromoxynil applied with or without 2,4-D ester caused the number of seeds per panicle to decline. Sorghum partially compensated the reduced seeds per panicle by increasing the kernel weight which also was reported to occur by Phillips in 1958. The increased kernel weights were not enough to compensate the reduced seeds per panicle and consequently, grain yields were reduced. Note that all treatments contained atrazine at 560 g ha^{-1} and the atrazine label states that sorghum over 31 cm should not be treated.

Pyrasulfotole & bromoxynil caused injury to all application timings but had more injury when applied to 1- and 4-leaf sorghum. The addition of 2,4-D ester did not reduce sorghum injury from pyrasulfotole & bromoxynil. The addition of 2,4-D ester to pyrasulfotole & bromoxynil did not influence yield and yield components. Flowering delays of 1 to 5 d were observed with 1- and 4-leaf treated sorghum. Few differences in heads per plant occurred between application timings of pyrasulfotole & bromoxynil. Pyrasulfotole & bromoxynil applied to 10- to flag leaf sorghum reduced seeds per panicle and caused an increase in 1000 kernel weight at these application timings. Grain yield reductions of 7 to 19% were observed when pyrasulfotole & bromoxynil was applied to the six growth stages in 2009. There still remains potential for pyrasulfotole & bromoxynil to be used in sorghum production systems to control infestations of broadleaf annual weeds because yield loss from weed infestations will be greater than that observed from pyrasulfotole & bromoxynil.

SOURCES OF MATERIALS

[1]TeeJet Spraying Systems, Wheaton, IL 60189-7900.

[2]Dickey-John GAC II grain analysis computer, Dickey-John Corporation, P.O. Box 10, Auburn, IL 62165.

[3]SAS version 8.2, SAS Institute Inc., 100 SAS Campus Drive, Cary, NC 27513.

ACKNOWLEDGEMENTS

The authors thank Bayer Crop Science and the Kansas Grain Sorghum Commission for funding the project.

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Table 2-1. Weather data at time of applications to 1- and 4-leaf sorghum at Manhattan and Rossville in 2009 and 2010.

	1-leaf sorghum				4-leaf sorghum			
	Manhattan		Rossville		Manhattan		Rossville	
Application date	6/24/2009	6/10/2010	6/12/2009	6/1/2010	7/1/2009	6/18/2010	6/22/2009	6/3/2010
Time of day	7:30 AM	8:00 AM	11:30 AM	9:45 AM	7:30 AM	9:00 AM	9:30 AM	4:30 PM
Crop height (cm)	1-3	2-3	1-3	2-3	15-20	10-11	10-15	6-8
Air temperature (C)	26	25	24	28	19	28	29	29
Relative humidity %	74	84	69	69	76	75	68	47
Wind speed (m s^{-1})	1.8	3.4	3.1	2.2	1.1	4	1.1	1.6
Wind direction	WSW	S	ESE	S	S	S	S	S
Dew presence	No	Yes	No	No	Yes	No	No	No
Soil temperature (C)	22	24	20	21	18	24	23	23
Soil moisture	Excellent	Excellent	Excellent	Excellent	Excellent	Wet	Excellent	Good
Cloud cover %	10	0	60	40	0	10	0	10

Table 2-2. Weather data at time of applications to 7- and 10-leaf sorghum at Manhattan and Rossville in 2009 and 2010.

	7-leaf sorghum				10-leaf sorghum			
	Manhattan		Rossville		Manhattan		Rossville	
Application date	7/9/2009	6/28/2010	7/2/2009	6/15/2010	7/17/2009	7/6/2010	7/13/2009	6/28/2010
Time of day	8:15 AM	8:00 AM	12:45 PM	2:30 PM	4:15 PM	9:15 AM	10:30 AM	9:45 AM
Crop height (cm)	25	24-33	25-30	23-28	60-65	53-63	60-70	71-81
Air temperature (C)	26	21	31	29	27	27	28	25
Relative humidity %	72	77	54	67	47	83	86	74
Wind speed (m s^{-1})	1.3	0	0.9	1.3	1.3	0	0.9	0.4
Wind direction	SE	E	S	NW	NW	ESE	ENE	W
Dew presence	Yes	Yes	No	No	No	Yes	No	No
Soil temperature (C)	21	24	21	24	21	22	20	24
Soil moisture	Good	Good	Good	Wet	Good	Excellent	Excellent	Good
Cloud cover %	10	90	100	70	15	75	70	50

Table 2-3. Weather data at time of applications to 13- and flag-leaf sorghum at Manhattan and Rossville in 2009 and 2010.

	13-leaf sorghum				Flag leaf sorghum			
	Manhattan		Rossville		Manhattan		Rossville	
Application date	7/26/2009	7/16/2010	7/21/2009	7/9/2010	8/10/2009	7/28/2010	8/3/2009	7/15/2010
Time of day	12:00 PM	9:30 AM	8:15 PM	12:25 PM	5:15 PM	7:30 AM	9:30 AM	11:00 AM
Crop height (cm)	90-100	97-114	76-91	101-114	101-110	112-124	101-110	101-117
Air temperature (C)	30	29	21	29	28	26	28	31
Relative humidity %	57	83	87	64	58	87	75	76
Wind speed (m s^{-1})	1.3	0	0	2	0.9	0	0.7	0
Wind direction	E	N	NW	NE	N	S	W	N
Dew presence	No	Yes	Yes	No	Yes	Yes	No	No
Soil temperature (C)	19	23	19	26	26	27	18	25
Soil moisture	Good	Wet	Excellent	Excellent	Good	Dry	Excellent	Excellent
Cloud cover %	0	85	0	25	5	0	0	80

Table 2-4. Visible injury to grain sorghum 7 and 14 DAT as affected by sorghum leaf stage at time of application and pyrasulfotole & bromoxynil rate at Manhattan and Rossville, KS in 2009 and 2010.

		Injury 7 DAT				Injury 14 DAT			
		Manhattan		Rossville		Manhattan		Rossville	
Leaf stage	Rate[a]	2009	2010	2009	2010	2009	2010	2009	2010
	g ha^{-1}	---------- % ----------							
1	246	3	6	11	6	3	11	3	13
	492	11	9	18	11	9	13	9	29
4	246	8	18	12	6	5	21	7	11
	492	15	26	29	15	8	24	29	26
7	246	12	13	12	7	10	4	7	5
	492	21	14	19	9	14	5	14	6
10	246	13	14	13	10	9	7	10	7
	492	16	16	19	16	13	10	14	11
13	246	11	7	7	7	6	7	6	3
	492	14	11	11	11	9	12	8	6
Flag leaf	246	0	1	6	13	1	1	3	13
	492	2	2	8	15	4	2	5	15
LSD ≤ 0.05		3	3	4	1	ns	2	4	3

[a]Indicates rate of pyrasulfotole & bromoxynil + atrazine 560 g ha^{-1} + ammonium sulfate 2.8 kg ha^{-1} pooled over the addition of 2,4-D ester.

Table 2-5. Visible injury to grain sorghum 21 and 28 DAT as affected by sorghum leaf stage at time of application and pyrasulfotole & bromoxynil rate at Manhattan and Rossville, KS in 2009 and 2010.

		Injury 21 DAT				Injury 28 DAT			
		Manhattan		Rossville		Manhattan		Rossville	
Leaf stage	Rate[a]	2009	2010	2009	2010	2009	2010	2009	2010
	g ha^{-1}	---------- % ----------							
1	246	3	10	1	10	3	5	0	9
	492	7	12	7	19	6	5	4	20
4	246	4	15	5	16	1	4	4	13
	492	7	20	23	30	3	7	18	24
7	246	3	0	4	2	0	0	1	0
	492	6	0	9	3	0	0	4	0
10	246	4	3	8	3	3	3	3	2
	492	6	5	11	4	4	4	5	3
13	246	4	7	2	2	4	7	3	2
	492	6	12	5	3	6	12	5	3
Flag leaf	246	1	1	5	13	1	1	5	13
	492	4	2	6	15	4	2	6	15
LSD ≤ 0.05		ns	2	4	3	2	2	3	4

[a]Indicates rate of pyrasulfotole & bromoxynil + atrazine 560 g ha^{-1} + ammonium sulfate 2.8 kg ha^{-1} pooled over the addition of 2,4-D ester.

Table 2-6. Visible injury to grain sorghum as affected by pyrasulfotole & bromoxynil averaged over rate with or without 2,4-D applied to different sorghum leaf stages 7 and 14 DAT at Manhattan and Rossville, KS in 2009 and 2010 and 21 DAT at Manhattan in 2009.

		Injury 7 DAT				Injury 14 DAT				Injury 21 DAT
		Manhattan		Rossville		Manhattan		Rossville		Manhattan
Leaf stage	2,4-D ester[a]	2009	2010	2009	2010	2009	2010	2009	2010	2009
	g ha^{-1}	---------- % ----------								
1	0	7	6	14	9	5	10	5	19	4
	140	8	8	15	9	7	13	7	23	6
4	0	9	22	22	11	6	21	17	21	4
	140	13	22	18	10	7	25	18	17	7
7	0	16	14	17	8	10	6	12	5	4
	140	18	13	14	8	14	4	9	5	5
10	0	16	16	17	14	13	8	14	10	6
	140	14	15	15	13	10	9	11	8	4
13	0	12	9	12	9	7	9	10	4	5
	140	13	9	6	9	8	9	4	5	5
Flag leaf	0	1	1	7	14	2	1	3	14	2
	140	2	2	8	14	3	2	5	15	4
LSD ≤ 0.05		ns	ns	ns	ns	2	2	ns	3	2

[a]Indicates rate of 2,4-D ester tank-mixed with pyrasulfotole & bromoxynil + atrazine 560 g ha^{-1} + ammonium sulfate 2.8 kg ha^{-1}.

Table 2-7. Visible injury to grain sorghum as affected by rate of pyrasulfotole & bromoxynil applied alone or in combination with 2,4-D ester to different sorghum leaf stages 21 and 28 DAT at Manhattan, KS, 2010.

			Injury	
Leaf stage	Treatment[a]	Rate	21 DAT	28 DAT
		g ha^{-1}	------- % -------	
1	P&B[b]	246	7	4
	P&B	492	13	7
	P&B + 2,4-D ester	246 + 140	13	7
	P&B + 2,4-D ester	492 + 140	11	4
4	P&B	246	14	4
	P&B	492	18	6
	P&B + 2,4-D ester	246 + 140	16	5
	P&B + 2,4-D ester	492 + 140	22	8
7	P&B	246	0	0
	P&B	492	0	0
	P&B + 2,4-D ester	246 + 140	0	0
	P&B + 2,4-D ester	492 + 140	0	0
10	P&B	246	2	2
	P&B	492	5	3
	P&B + 2,4-D ester	246 + 140	4	5
	P&B + 2,4-D ester	492 + 140	6	6
13	P&B	246	7	7
	P&B	492	12	12
	P&B + 2,4-D ester	246 + 140	7	7
	P&B + 2,4-D ester	492 + 140	12	12
Flag leaf	P&B	246	1	1
	P&B	492	2	2
	P&B + 2,4-D ester	246 + 140	1	1
	P&B + 2,4-D ester	492 + 140	2	2
	LSD ≤ 0.05		3	2

[a]All treatments include atrazine 560 g ha^{-1} + ammonium sulfate 2.8 kg ha^{-1}.

[b]P&B = pyrasulfotole & bromoxynil.

Table 2-8. Grain sorghum height as affected by pyrasulfotole & bromoxynil averaged over rate applied alone or in combination with 2,4-D ester to different sorghum leaf stages at Manhattan and Rossville, KS, in 2009 and 2010.

		Plant height			
		Manhattan		Rossville	
Leaf stage	2,4-D ester[a]	2009	2010	2009	2010
	g ha^{-1}	----------cm----------			
1	0	128	130*	135	116
	140	130	131	134	120
4	0	131	129**	134	120
	140	128	126**	133	119
7	0	128	128**	131	122
	140	123**	127**	130*	118
10	0	128	127**	132	115*
	140	132	129**	138	116
13	0	124**	129**	129**	117
	140	127*	135	136	118
Flag Leaf	0	123**	128**	130	118
	140	124**	131	131	120
LSD ≤ 0.05		4.5	3	6	ns
Untreated check		130	133	134	124

[a]Indicates rate of 2,4-D ester tank-mixed with pyrasulfotole & bromoxynil + atrazine 560 g ha^{-1} + ammonium sulfate 2.8 kg ha^{-1}.

**Indicates significant difference of $\alpha \leq 0.05$ when compared to untreated check.

*Indicates significant difference of $\alpha \leq 0.10$ when compared to untreated check.

Table 2-9. Grain sorghum height as affected by rate of pyrasulfotole & bromoxynil applied to different sorghum leaf stages at Manhattan, KS, in 2009.

Leaf stage	Rate[a]	Height
	g ha^{-1}	cm
1	246	129
	492	128
4	246	129
	492	129
7	246	125**
	492	127*
10	246	128
	492	131
13	246	127*
	492	124**
Flag Leaf	246	126**
	492	122**
LSD ≤ 0.05		3
Untreated check		130

[a]Indicates rate of pyrasulfotole & bromoxynil + atrazine 560 g ha^{-1} + ammonium sulfate 2.8 kg ha^{-1}.

**Indicates significance $\alpha \leq 0.05$ when compared to untreated check.

*Indicates significance $\alpha \leq 0.10$ when compared to untreated check.

Table 2-10. Days to flowering from sorghum emergence, heads per plant, and seed per panicle as affected by timing of application averaged across all herbicide treatments at Manhattan and Rossville, KS in 2009 and 2010.

	Days to flowering				Heads per plant			Seeds per panicle		
	Manhattan		Rossville		Manhattan	Rossville	Rossville	Manhattan	Rossville	Rossville
Leaf stage	2009	2010	2009	2010	2009		2010	2009		2010
1	60.1**	55.7**	67.8*	61.1**	1.10	1.07	0.96	2340	2120	1200
4	59.3**	57.8**	68.9**	60.4**	1.05	1.00**	0.97	2340	2000**	1210
7	57.8	54.4	66.8	56.1	1.08	1.06	0.98	2110**	1930**	1410*
10	57.8	55.8**	65.8	56.1	1.07	1.07	1.01	2090**	1870**	1070*
13	58.2	55.0**	66.1	55.3	1.05	1.04	1.01	1930**	1890**	1060*
Flag leaf	57.4	54.8*	65.9	55.4	1.09	1.06	1.01	1630**	1760**	820**
LSD ≤ 0.05	0.7	0.5	1.1	1.0	0.03	0.04	0.04	130	113	119
Untreated check	58.0	54.1	66.1	56.1	1.08	1.07	0.97	2445	2245	1243

**Indicates significance $\alpha \leq 0.05$ when compared to untreated check.

*Indicates significance $\alpha \leq 0.10$ when compared to untreated check.

Table 2-11. Grain sorghum 1000 kernel weight and yield as affected by application timing averaged across herbicide treatments of pyrasulfotole & bromoxynil at Manhattan and Rossville, KS, in 2009 and 2010.

	1000 kernel weight			Yield		
	Manhattan	Rossville	Rossville	Manhattan	Rossville	Rossville
Leaf stage	2009		2010	2009		2010
	---------- g ----------			---------- kg ha^{-1} ----------		
1	26.8	30.9	33.9**	8750**	9700*	5520
4	26.1	30.8	34.3**	8200**	8720**	5740
7	25.7	30.1	33.2**	7840**	8580**	6470*
10	28.4*	34.0**	34.3**	8530**	9650*	5230
13	29.0**	33.8**	34.8**	7760**	9400**	5160
Flag leaf	35.1**	35.0**	34.1**	8140**	9030**	3920*
LSD ≤ 0.05	1.0	1.1	0.7	637	637	655
Untreated check	26.9	31.4	32.1	9446	10650	5596

**Indicates significance $\alpha \leq 0.05$ when compared to untreated check.

*Indicates significance $\alpha \leq 0.10$ when compared to untreated check.

Table 2-12. Grain sorghum yield, heads per plant, and 1000 kernel weight as affected by timing of application of pyrasulfotole & bromoxynil with or without 2,4-D ester at Manhattan and Rossville, KS in 2009.

		Manhattan			Rossville	
Leaf stage	2,4-D ester[a]	Heads per plant	Seed per panicle	Yield	1000 kernel weight	
	g ha^{-1}			kg ha^{-1}	--------- g ----------	
1	0	1.08	2420	9070	27.4	31.4
	140	1.12	2250*	8430**	26.2	30.5
4	0	1.08	2270	8280**	26.5	31.2
	140	1.01**	2410	8120**	25.7	30.5
7	0	1.08	2200**	8520**	26.5	30.2
	140	1.08	2030**	7160**	24.9*	30.1
10	0	1.06	2060**	8400**	28.3	33.4*
	140	1.08	2110**	8660**	28.6**	34.6*
13	0	1.05	1980**	7740**	28.4**	32.7
	140	1.04	1890**	7780**	29.7*	34.9*
Flag leaf	0	1.10	1710**	8370**	34.8*	34.7*
	140	1.09	1550**	7910**	35.4*	35.3*
	LSD ≤ 0.05	0.05	ns	901	1.5	1.6
	Untreated check	1.08	2450	9450	26.9	31.4

[a]Indicates rate of 2,4-D ester tankmixed with pyrasulfotole & bromoxynil + atrazine 560 g ha^{-1} + ammonium sulfate 2.8 kg ha^{-1}.

**Indicates significance $\alpha \leq 0.05$ when compared to untreated check.

*Indicates significance $\alpha \leq 0.10$ when compared to untreated check.

Chapter 3 - Grain Sorghum Response to Pyrasulfotole and Bromoxynil and Growth Regulators

ABSTRACT

Pyrasulfotole & bromoxynil can cause injury to grain sorghum. Preliminary research indicated that growth regulator herbicides applied with pyrasulfotole & bromoxynil may reduce the crop injury. Field experiments were conducted in 2009 and 2010 near Manhattan, KS, to evaluate grain sorghum response to pyrasulfotole & bromoxynil (P&B) applied alone or with growth regulator herbicides. Pyrasulfotole & bromoxynil, 1:8 ratio, was applied at 246 g ha^{-1} alone or with 2,4-D ester, 2,4-D amine, or dicamba at 140 or 280 g ha^{-1} to 3- or 6-leaf sorghum. All treatments included atrazine + ammonium sulfate at 0.56 + 2.8 kg ha^{-1}. Pyrasulfotole & bromoxynil applied alone or with dicamba provided the least amount of injury at both timings. Injury from 2,4-D amine and ester increased with the higher rate with the most injury observed being 35 and 31% from tank-mixing 280 g 2,4-D amine or ester to P&B, 14 DAT. Injury declined by 28 DAT to 8% or less for all treatments. Averaged over herbicide treatments, 3-leaf treated sorghum flowered 1.2 d later, had fewer heads per plant, had more seeds per panicle, and greater 1000 kernel weight than the 6-leaf treated sorghum. Averaging over growth regulator herbicides, the addition of 280 g of growth regulator to P&B delayed flowering 1.4 to 2 d compared to P&B alone which also delayed flowering by 1.4 d compared to the untreated sorghum. The addition of 140 g 2,4-D amine or ester increased heads per plant by 5 to 7% compared to P&B alone. Pyrasulfotole & bromoxynil applied to 3- or 6-leaf sorghum reduced heads per plant 11 and 9% compared to the untreated check. Seeds per panicle were similar among herbicide treatments but was 10% greater on 3-leaf than 6-leaf treated sorghum. The 3-

leaf treated sorghum produced 10% more seeds per panicle but had similar 1000 kernel weight and grain yield than the untreated sorghum. Adding either rate of 2,4-D amine to P&B reduced 1000 kernel weight and yield compared to P&B alone. The highest rate of all growth regulators applied to 6-leaf sorghum reduced 1000 kernel weight and grain yield compared to P&B alone. Pyrasulfotole & bromoxynil tended to reduce yield compared to the untreated check, however, yield loss from weed infestations would be greater than that observed with P&B. This suggests that P&B would be a useful option for growers to manage broadleaf weeds in grain sorghum.

Nomenclature: Pyrasulfotole & bromoxynil; sorghum, *Sorghum bicolor* (L.) Moench. SORBI; 2,4-D amine, 2,4-D ester, dicamba.

Key words: auxins, growth regulator herbicides, growth stages, HPPD-inhibiting herbicides, injury, safening, yield components.

INTRODUCTION

Grain sorghum is an important crop in the Great Plains region of the United States. In 2010, Kansas produced 4.3 million kg of grain sorghum, making it the largest sorghum producing state in the US (Anonymous 2011a). Climate varies widely across Kansas with annual precipitation totals ranging from 117 cm in eastern Kansas to 35 cm in western Kansas (Anonymous 2011b). The lack of precipitation coupled with extreme high temperatures can make crop production challenging. Grain sorghum is considered a relatively drought tolerant C_4 plant, able to perform better than other crops such as soybeans and corn when moisture is limited and temperatures are high (Stahlman and Wicks 2000).

Weeds compete with grain sorghum for water, light, and nutrients, which can reduce crop yields (Feltner et al. 1969; Graham et al. 1988). Yield reductions have been observed from weed competition in grain sorghum, thus adequate weed control is needed at early stages of sorghum growth to avoid large yield reductions (Knezevic et al. 1997; Burnside and Wicks 1967). Burnside et al. (1967) reported no reductions in yield if weeds were controlled by three weeks after planting and as much as 55% yield reduction when weeds were left uncontrolled until eight weeks after planting. Weeds reduce grain yield through direct competition, increased harvest difficulty, and increased disease and insect pressure (Moore et al. 2004; Zimdahl 1999; Burnside and Wicks 1967).

Weed control is achieved through cultural, mechanical, and chemical practices, however chemical control is important especially in reduced or no-tillage systems (Brown et al. 2004; Regehr 1998). A 2003 survey indicated that 90% of sorghum hectares in Kansas received an application of herbicide (Anonymous 2011c). Current herbicides used in sorghum production include preemergence applied (PRE) chloroacetamides and triazines followed by post-emergence

applied (POST) acetolactate synthase (ALS) inhibitors, triazines, growth regulators, or protoporphyrinogen oxidase (protox) inhibitor herbicides (Thompson et al. 2011). The PRE herbicides require moisture for activation to provide adequate weed control. Sorghum is commonly grown where moisture is limited, which can result in poor weed control with PRE herbicides (Tapia et al. 1997). This stresses the importance and need for effective POST herbicide programs if the PRE herbicides fail.

Applying growth regulators POST to grain sorghum has been reported to cause visual crop injury such as leaf-rolling, stunting, and lodging (Wiese and Rea 1958; Peterson et al. 2010; Phillips 1958). In addition to causing visual injury, flowering delays can occur from applications of 2,4-D applied before 7- to 9-leaf grain sorghum (Wiese and Rea 1958). Phillips (1958) observed malformed brace root development with 2,4-D rates ranging from 280 to 1120 g ha^{-1}. The same study also found differences between formulations of 2,4-D and that 2,4-D ester at 140 g ha^{-1} and more caused yield reductions when applied to 9- to 11-leaf sorghum. The results were not consistent year to year. Yield reductions also have been reported when 2,4-D was applied during pollination (Wiese and Rea 1958; Phillips 1958). Other growth regulators such as dicamba have been reported to delay flowering when applied two to three weeks after sorghum emergence (Peeper et al. 1970). The same study showed that dicamba reduces peduncle length, increased tillering, and reduced grain yields.

Atrazine is the most common herbicide used in Kansas and is applied to 79% of the sorghum hectares (Anonymous 2011c). Heavy reliance and repeated use of triazine herbicides in corn and sorghum cropping systems have resulted in the development of triazine-resistant populations of Palmer amaranth (*Amaranthus palmeri* S. Wats.), common waterhemp (*Amaranthus rudis* Sauer), redroot pigweed (*Amaranthus retroflexus* L.), downy brome (*Bromus*

tectorum L.), and kochia (*Kochia scoparia* L. Schrad.) in Kansas (Heap 2011; Peterson 1999). Additionally, ALS-inhibiting resistant biotypes of Palmer amaranth, common waterhemp, Russian thistle (*Salsola tragus* L.), kochia, common cocklebur (*Xanthium strumarium* L.), shattercane (*Sorghum bicolor* L. Moench), and common sunflower (*Helianthus annuus* L.) have been reported (Heap 2011; Peterson 1999). Herbicide resistant weeds have led to changes in production practices such as increased tillage, crop rotation, tank-mixing herbicides with different modes of action, and the use of directed herbicides (Peterson et al. 1999; Abit 2009).

Because many weed species are developing resistance to herbicides commonly used in sorghum, a solution is needed to gain control of these resistant weeds. One possible solution to control the resistant weeds is to use a hydroxyphenylpyruvate dioxygenase (HPPD)-inhibitor herbicide called pyrasulfotole. Pyrasulfotole controls many summer annual broadleaf weed species such as pigweeds, kochia, and common sunflower (Olson et al. 2011). The enzyme, HPPD is an important component for the production of plastioquinone (PQ), which is involved in transporting electrons in the electron transport chain of photosynthesis, and is a cofactor for the synthesis of carotenoids (Freigang et al. 2008). Carotenoids are needed to quench triplet chlorophyll and reactive oxygen species (ROS) which cause membrane damage (Freigang et al. 2008). Pyrasulfotole became commercially available in 2008, and is labeled for use in wheat, barley, and triticale. The commercial product available is a premix of pyrasulfotole and bromoxynil in a 1:8 ratio.

Most HPPD-inhibiting herbicides are currently registered for use in corn production systems and only mesotrione is labeled for use in sorghum as a PRE herbicide. Abit et al. (2009) examined 85 sorghum hybrids to determine differences in susceptibility to mesotrione. Pioneer 84G62, Pioneer 85G01, and Triumph TR 438 were the most susceptible hybrids, whereas Dekalb

DKS35-70, Frontier F222E, and Asgrow Seneca were the most tolerant. Abit and Al-Khatib (2009) also investigated sorghum absorption, translocation, and crop tolerance of mesotrione applied POST to a sensitive sorghum hybrid and a more tolerant sorghum hybrid and concluded that there were few differences in absorption and translocation between the two hybrids. Abit and Al-Khatib (2009) concluded that differences in the metabolism rate resulted in the differential injury observed between the two hybrids. Because mesotrione causes injury to sorghum when applied POST, one can expect other HPPD inhibitors to also cause injury.

Sorghum injury symptoms from HPPD-inhibitors may possibly be reduced with the addition of growth regulator herbicides. Brown et al. (2004) observed a safening response in sorghum when 2,4-D or dicamba were applied with metsulfuron to 3- or 4-leaf sorghum by reducing visual injury and yield loss. Other growth regulators such as fluroxypyr or clopyralid did not reduce injury or provide adequate weed control. Olson et al. (2011) evaluated the response of sorghum to early and late POST applications of pyrasulfotole & bromoxynil with and without 2,4-D ester or dicamba. Of the seven sites, only two sites indicated a safening affect from the addition of 2,4-D ester and dicamba to pyrasulfotole & bromoxynil when applied early POST, however when applied late-POST no significant safening from 2,4-D ester. Dicamba safened pyrasulfotole & bromoxynil by 7% compared to pyrasulfotole &bromoxynil applied alone at Garden City.

The objectives of this research were to evaluate grain sorghum response to pyrasulfotole & bromoxynil with and without various growth regulator herbicides applied at two vegetative growth stages and to determine if different growth regulator herbicide rates reduce visible injury or increase sorghum yield response to pyrasulfotole & bromoxynil when evaluated in a weed-free situation.

MATERIALS AND METHODS

Field experiments were conducted at Kansas State University Agronomy Department fields at Ashland Bottoms south of Manhattan, KS in 2009 and 2010. In 2009 the soil was a Reading Silt loam (Fine-silty, mixed, superactive, mesic Pachic Argiudolls) with a pH of 6.3 and 2.3% organic matter and in 2010 the soil was a Wymore silty clay loam (fine, montmorillonitic, mesic, Aquic Argiudolls) with a pH of 6.0 and 2.5% organic matter. Nitrogen at 247 kg ha^{-1} was applied and incorporated into the soil prior to planting in both years.

Pioneer grain sorghum hybrid '84G62' was planted in 76 cm rows at 143,000 seeds ha^{-1} on June 18, 2009 and June 3, 2010. The hybrid was reported to be among the most susceptible hybrids to mesotrione and may also be susceptible to pyrasulfotole & bromoxynil (Abit et al. 2009). Plots were 3.1 m wide by 8.2 m long and were maintained weed-free with a pre-emergence application of s-metolachlor at 1.41 kg ha^{-1} + atrazine at 1.12 kg ha^{-1}, followed by hand weeding throughout the growing season. Herbicide treatments were applied with a CO_2 pressurized backpack sprayer equipped with TurboTee[1] 11002 flat fan nozzle tips calibrated to deliver 140 L ha^{-1} at a pressure of 234 kPa.

Herbicide treatments were applied to 3- and 6-leaf grain sorghum. The seven herbicide treatments were pyrasulfotole & bromoxynil at 246 g ha^{-1} applied alone and tank-mixed with 2,4-D amine, 2,4-D ester, or dicamba at 140 or 280 g ha^{-1}. The 246 g ha^{-1} is considered the suggested use rate for sorghum and the growth regulator rates used are 0.25 and 0.5 the normal use rate. (Personal communication, Bayer Crop Science). All treatments included atrazine + ammonium sulfate at 0.56 + 2.8 kg ha^{-1}. Atrazine was included with each treatment because of the synergism between PSII inhibitors and HPPD inhibitors (Hess 2000; Abendroth et al. 2006). The inclusion

of ammonium sulfate was to condition the carrier as required by the pyrasulfotole & bromoxynil label. The weather data at each application date is presented in Table 3-1.

Sorghum injury was rated visually 7, 14, 21, and 28 days after treatment (DAT) using a scale of 0 to 100% where 0% = no injury and 100% = mortality. Plant population was determined by counting 8.2 m of row 10 d after sorghum emergence. Flowering date was recorded when 50% of the main stems reached half-bloom and was presented as days to flowering from crop emergence. Grain sorghum height was determined in September from three individual plant measurements in each plot. Heads were counted during grain fill from 8.2 m of row. Sorghum yield was determined by mechanical harvesting the middle two rows in each plot. Moisture content was determined with a grain analyzer[2], and grain yield was adjusted to 14% moisture. Grain samples were collected from each plot and 1000 kernel weight was determined. Seeds per panicle was also calculated.

The experimental design was a randomized complete block with a factorial arrangement of two application times, three growth regulator herbicides, and two rates for each growth regulator herbicide. An untreated check was included in each experiment and treatments were replicated four times. All data were subjected to analysis of variance using PROC MIXED in SAS[3] and means were separated by using Fisher's protected LSD < 0.05. Contrasts were used to detect differences of yield injury components between pyrasulfotole & bromoxynil alone and pyrasulfotole & bromoxynil with growth regulators, across growth regulator rates, and between pyrasulfotole & bromoxynil alone and the untreated check. Treatment responses revealed differences between years for all injury ratings, thus data for these response variables are presented separately. Differences between years for days to flowering and plant height was not observed so the data were pooled.

RESULTS AND DISCUSSION

Visible Injury

All herbicide treatments at both application timings in both years visibly injured grain sorghum (Table 3-2). In general, visible injury increased with the addition of growth regulator herbicides to pyrasulfotole & bromoxynil, however, visible injury decreased over time from 7 to 28 DAT. Pyrasulfotole & bromoxynil applied without growth regulators consistently provided similar or less visible injury at all evaluation timings compared to treatments containing 2,4-D amine, 2,4-D ester, or dicamba. This suggests that the growth regulator herbicides did not provide safening as reported by Olson et al. (2011) or Brown et al. (2004). Visible injury symptoms from pyrasulfotole & bromoxynil consisted of general leaf chlorosis, necrotic leaf margins and leaf tips, necrotic spotting on treated leaves, red-brownish banding on treated leaves, and stunting. In addition to pyrasulfotole & bromoxynil injury, treatments containing growth regulator herbicides caused buggy whipping, plant leaning, minor lodging, and green snap consistent with that reported by Brown et al. (2004) and Peterson et al. (2010).

Injury symptoms were greatest 7 DAT in 2009; however, in 2010 the most injury observed was 7 DAT with the 6-leaf application timing and 14 DAT with the 3-leaf application timing (Table 3-2). Injury was greater in 2010 than in 2009. The greater injury in 2010 could be attributed to greater relative humidity at the time of application for both growth stages (Table 3-1). Herbicides enter leaf surfaces more easily when the humidity is high because the solution tends to evaporate off the leaf surface slower, allowing a greater time for absorption (Rao 2000). The leaf cuticle is thinner and plant water content is greater in high humidity conditions which allow herbicides to diffuse faster across the cuticle and into cell walls. This facilitates symplastic movement to phloem tissue allowing fast translocation within the plant (Rao 2000).

In 2009 no injury differences were observed between herbicide treatments and application timing 7 and 14 DAT (Table 3-2). In 2010, pyrasulfotole & bromoxynil with 2,4-D ester at both rates or with 2,4-D amine at the high rate applied to 3-leaf sorghum caused more injury 7 DAT than pyrasulfotole & bromoxynil applied alone. Additionally at 7 DAT, pyrasulfotole & bromoxynil tank-mixed with 2,4-D amine at 280 g ha^{-1} applied to 6-leaf sorghum caused the most crop injury at 30%. The addition of the highest rates of 2,4-D ester or dicamba or the low rate of 2,4-D amine with pyrasulfotole & bromoxynil applied to 6-leaf sorghum also caused more injury than pyrasulfotole & bromoxynil alone in 2010. In 2009 by 14 DAT, injury diminished to 9% or less for all treatments except pyrasulfotole & bromoxynil with the low rate of dicamba applied to 3-leaf sorghum which also caused some lodging. In 2010, injury was greatest 14 DAT when both rates of 2,4-D amine and 2,4-D ester were applied with pyrasulfotole & bromoxynil to 3-leaf sorghum. Injury from herbicides applied to 6-leaf sorghum declined from 7 to 14 DAT in 2010 (Table 3-2). Injury continued to decline by 21 DAT, however, pyrasulfotole & bromoxynil applied with the higher rates of 2,4-D amine, 2,4-D ester, or dicamba caused the greatest injury to 3-leaf sorghum in 2009. In 2009, injury was less than 10% by 21 DAT with all treatments at both timings, indicating sorghum was recovering from the initial crop injury. In 2010, no differences were observed among herbicide treatments at 21 DAT, however, injury tended to be greater from the 3-leaf application than the 6-leaf application. Evidence of stunting remained 28 DAT, generally with 2,4-D amine or the highest rate of 2,4-D ester and dicamba applied to 3-leaf sorghum. In 2009 and 2010, 6-leaf treated sorghum had little injury still evident and all injury ratings were 4% or less by 28 DAT. The injury observed 28 DAT was less than that reported by Rosales-Robles et al. (2005), who reported 14% injury 28 DAT from 2,4-D amine applied at 140 g ha^{-1}.

Values of sorghum injury were averaged over growth regulator rates from pyrasulfotole & bromoxynil + 2,4-D amine, 2,4-D, ester, or dicamba were contrasted to pyrasulfotole & bromoxynil applied alone (Table 3-3). At 7 DAT pyrasulfotole & bromoxynil + 2,4-D amine, 2,4-D ester, or dicamba applied to 3-leaf sorghum had more injury of 15, 12, and 11% than pyrasulfotole & bromoxynil alone at 7% in 2009 while no differences were observed between treatments in 2010. By 14 DAT in 2009, growth regulator injury declined and differences were less than 4% between pyrasulfotole & bromoxynil alone and treatments with growth regulators applied to 3-leaf sorghum. Unlike that observed in 2009, 2,4-D amine and 2,4-D ester applied to 3-leaf sorghum caused 30 and 29% injury compared to only 13% injury with pyrasulfotole & bromoxynil alone at 14 DAT. Injury from growth regulators declined by 21 DAT in 2010 and by 28 DAT, injury declined to 5% or less. The addition of 2,4-D amine applied to 6-leaf sorghum caused 11 and 25% injury in 2009 and 2010 and is greater than pyrasulfotole & bromoxynil alone with 6 and 12% injury by 7 DAT. These treatments continued to cause more injury than pyrasulfotole & bromoxynil applied alone through 21 DAT. The addition of 2,4-D ester or dicamba to pyrasulfotole & bromoxynil applied to 6-leaf sorghum had less injury than that observed from 2,4-D amine and was not different than that observed by pyrasulfotole & bromoxynil alone in 2009 and 2010 except 2010 7 DAT. Overall in 2009, the addition of any growth regulator caused greater injury when applied to 3-leaf sorghum. Pyrasulfotole & bromoxynil applied alone to 6-leaf sorghum had little to no injury 21 and 28 DAT and adding 2,4-D amine, 2,4-D ester, or dicamba to pyrasulfotole & bromoxynil caused similar low levels of injury to pyrasulfotole & bromoxynil applied alone 21 or 28 DAT in 2010. Dicamba caused the least visual injury across all application timings and years, and sorghum recovered from the injury faster than sorghum treated with 2,4-D amine or 2,4-D ester. Safening metsulfuron with

2,4-D or dicamba in grain sorghum as reported by Brown et al. (2004) was not observed in this study with growth regulators and pyrasulfotole & bromoxynil. Generally, additional injury was observed from the growth regulators compared to pyrasulfotole & bromoxynil alone.

The impact of rate of growth regulator herbicideaveraged over growth regulator herbicides on sorghum injury was contrasted to injury from pyrasulfotole & bromoxynil alone (Table 3-4). Generally, increasing the rate of growth regulator herbicides from 140 g ha^{-1} to 280 g ha^{-1} caused greater injury at both application timings in both years. Adding 140 g ha^{-1} growth regulators to pyrasulfotole & bromoxynil caused similar injury at 7 DAT to pyrasulfotole & bromoxynil alone when applied to 3- or 6-leaf sorghum in both years. The trend continued on 6-leaf treated sorghum 14 DAT however, the addition of growth regulators caused more injury to 3-leaf sorghum compared to pyrasulfotole & bromoxynil alone in 2010. By 21 DAT, 3-leaf treated sorghum had 3 to 7% more injury with the addition of growth regulators compared to pyrasulfotole & bromoxynil applied alone. Evidence of crop injury from the addition of 140 g ha^{-1} growth regulators was 5% or less by 28 DAT. At 7 DAT, the 280 g ha^{-1} rate of growth regulators had 9 and 5% more sorghum injury compared to pyrasulfotole & bromoxynil alone applied at the 3- and 6-leaf application timings, respectively, in 2009. In 2010, 280 g ha^{-1} growth regulators applied to 3-leaf sorghum had similar injury to that caused by pyrasulfotole & bromoxynil alone 7 DAT, however, the higher rate of growth regulators averaged 23% injury to 6-leaf sorghum which was greater than pyrasulfotole & bromoxynil applied alone. At 14 DAT, injury from 280 g ha^{-1} growth regulator declined to 7 and 9% on 3- and 6-leaf sorghum, but this injury was 3 and 5% greater than that caused by pyrasulfotole & bromoxynil alone in 2009. Injury from growth regulators applied to 3-leaf sorghum increased from 7 to 14 DAT in 2010 and 9 to 15% more injury was observed than injury from pyrasulfotole & bromoxynil alone.

Injury was less at the 6-leaf timing 14 DAT, but the 280 g ha^{-1} growth regulators caused 6% more injury than pyrasulfotole & bromoxynil applied alone in 2010. In both years, the high rate of growth regulators increased injury by 6 and 7% compared to pyrasulfotole & bromoxynil applied alone to 3-leaf sorghum, 21 DAT. Injury was similar with pyrasulfotole & bromoxynil applied alone and the addition of 280 g ha^{-1} growth regulators to 6-leaf sorghum 21 or 28 DAT in both years.

Yield Components

In August 2010, a storm event occurred at Ashland Bottoms producing strong winds, which caused lodging to sorghum plants in the experiment. The storm damage was so severe that yield components could not be measured accurately thus yield components from 2009 only will be presented.

No interactions occurred between application timing and herbicide treatment when $\alpha < 0.05$, thus results were pooled over application timings (Table 3-5). The addition of 2,4-D amine or 2,4-D ester at both rates, or dicamba at 280 g ha^{-1} delayed flowering by 0.9 to 2.3 d compared to pyrasulfotole & bromoxynil applied alone across both years. Sorghum treated with 2,4-D amine at 280 g ha^{-1} reduced sorghum height by 3 cm. No other plant height differences were observed. Sorghum treated with the addition of 140 g ha^{-1} of 2,4-D amine or 2,4-D ester to pyrasulfotole & bromoxynil had an increase of 0.06 to 0.08 heads per plant compared to pyrasulfotole & bromoxynil applied alone. Sorghum treated with pyrasulfotole & bromoxynil alone or tank-mixed with growth regulators did not affect seeds per panicle. The addition of 2,4-D amine reduced 1000 kernel weight and reduced yield compared to sorghum treated with pyrasulfotole & bromoxynil alone. Increasing the rate of 2,4-D amine produced similar heads per plant and seeds per panicle to pyrasulfotole & bromoxynil alone, but 1000 kernel weight was

reduced, causing the greatest yield reduction of 1200 kg ha^{-1}. Adding 280 g ha^{-1} of 2,4-D ester reduced yield by 640 kg ha^{-1} compared to pyrasulfotole & bromoxynil alone. Sorghum treated with 2,4-D ester at 140 g ha^{-1} or dicamba at either rate yielded similar to sorghum treated with pyrasulfotole & bromoxynil alone even though dicamba at 280 g ha^{-1} reduced 1000 kernel weight. These results differed from Phillips (1958) who reported no yield reductions with 1.12 kg ha^{-1} 2,4-D applied to 5- to 7-leaf sorghum grown in weed-free conditions.

Days to flower and yield component results were pooled over herbicide treatments to compare application timings (Table 3-6). Yield or plant height was not affected by application timing. Differences between application timings were observed with days to flower, heads per plant, seeds per panicle, and 1000 kernel weight. Sorghum treated at the 3-leaf stage flowered 1.2 d later and had 0.11 fewer heads per plant compared to sorghum treated at the 6-leaf stage. This agrees with Wiese and Rea (1958) who observed 2,4-D at 280 ha^{-1} to 1120 g ha^{-1} applied to 2- to 3-leaf treated sorghum flowered 1 d later than 6- to 7-leaf treated sorghum. Sorghum treated at 3-leaf produced more seeds per panicle and had increased 1000 kernel weight compared to 6-leaf treated sorghum.

The impact of different growth regulators averaged across rate on days to flowering, plant height, and sorghum yield components were contrasted to pyrasulfotole & bromoxynil alone (Table 3-7). Flowering was delayed 1.5 to 1.6 d with the addition of 2,4-D amine to pyrasulfotole & bromoxynil applied to 3- or 6-leaf sorghum. Tank-mixing 2,4-D ester with pyrasulfotole & bromoxynil delayed flowering 1.7 d compared to pyrasulfotole & bromoxynil alone when applied to 3-leaf sorghum. Dicamba applied to 3- or 6-leaf sorghum or 2,4-D ester applied to 6-leaf sorghum did not delay flowering compared to sorghum treated with pyrasulfotole & bromoxynil alone. These data differ from Peeper et al. (1970) who reported sorghum flowering

delays compared to an untreated check when 300 or 600 g ha^{-1} dicamba was applied 2 or 11 d after emergence.

Adding growth regulators to pyrasulfotole & bromoxynil did not affect plant height regardless of application timing (Table 3-7). Heads per plant were similar with all treatments applied to 3-leaf sorghum, however applying 2,4-D amine with pyrasulfotole & bromoxynil to 6-leaf sorghum caused a 0.07 heads $plant^{-1}$ increase compared to pyrasulfotole & bromoxynil alone. Seeds per panicle were similar when pyrasulfotole & bromoxynil was applied alone or with growth regulators at either leaf stage.

Applying 2,4-D amine with pyrasulfotole & bromoxynil to 3- or 6-leaf sorghum reduced 1000 kernel weight by 1.5 and 3.1 g respectively, compared to pyrasulfotole & bromoxynil alone (Table 3-7). 2,4-D amine applied to 3- and 6-leaf sorghum reduced kernel weight reduced grain yield 730 to 1290 kg ha^{-1}. Including 2,4-D ester or dicamba with pyrasulfotole & bromoxynil appeared to have little to no affect on grain yield. These results differed from reports by Phillips (1958) who reported lower yields from 2,4-D ester compared to 2,4-D amine. The use of low 2,4-D rates in this experiment may explain these differences.

Days to flowering, plant height, and sorghum yield components data were pooled over growth regulator herbicides and the means of growth regulator rate were contrasted to pyrasulfotole & bromoxynil alone (Table 3-8). Generally, adding growth regulators to pyrasulfotole & bromoxynil delayed flowering from 0.3 to 2 d compared to pyrasulfotole & bromoxynil alone. Increasing growth regulator rates to 280 g ha^{-1} delayed flowering by 2 and 1.4 d when applied to 3- or 6-leaf sorghum, respectively. These data agree with Wiese and Rea (1958) who observed 1 to 3 d flowering delays when 2,4-D at 280 to 1120 g ha^{-1} was applied to 2- to 7-leaf sorghum. Plant height was not affected with growth regulator rate compared to

pyrasulfotole & bromoxynil applied alone regardless of application timing. Heads per plant were generally lower with 3-leaf treated sorghum, however, the addition of growth regulators did not reduce heads per plant compared to pyrasulfotole & bromoxynil alone at either timing. A slight increase of heads per plant was observed with the 140 g ha^{-1} growth regulator rate applied to 6-leaf sorghum. The high rate of growth regulators at both application timings reduced grain yield by 8%. Although the low rate of growth regulators applied to 6-leaf sorghum increased heads per plant to 1.23, slight reductions in seeds per panicle and kernel weight were evident, slightly reducing grain yield compared to pyrasulfotole & bromoxynil alone. Increasing the growth regular rate in 6-leaf sorghum produced similar heads per plant and seeds per panicle to pyrasulfotole & bromoxynil alone, but reduced 1000 kernel weight by 2.2 g which reduced overall grain yield by 8%.

The response of days to flower, plant height, and yield components from pyrasulfotole & bromoxynil applied alone to 3- and 6-leaf sorghum were contrasted to the untreated check (Table 3-9). Applying pyrasulfotole & bromoxynil to 3-leaf sorghum delayed flowering by 1.4 d and no differences were observed when treating 6-leaf sorghum compared to the untreated check. Plant height was not affected by pyrasulfotole & bromoxynil at either application timings. Pyrasulfotole & bromoxynil applied to 3-leaf sorghum reduced heads per plant but sorghum plants were able to compensate by increasing seeds per panicle by 10%. The 1000 kernel weights only tended to be lower when treated with pyrasulfotole & bromoxynil compared to the untreated check. Applications to 6-leaf sorghum with pyrasulfotole & bromoxynil reduced heads per plant by 0.08, while seeds per panicle and kernel weight remained similar to the untreated check. Yield tended to be reduced from applying pyrasulfotole & bromoxynil to 3- and 6-leaf sorghum compared to the untreated check.

This study demonstrated that the addition of growth regulators to pyrasulfotole & bromoxynil caused more injury than pyrasulfotole & bromoxynil applied alone and that growth regulators did not safen pyrasulfotole & bromoxynil from visual injury. The 3-leaf treated sorghum required additional time to fully recover from injury compared to 6-leaf treated sorghum. Both rates of 2,4-D amine tank-mixed with pyrasulfotole & bromoxynil appeared to be more injurious than 2,4-D ester or dicamba by causing visual injury, delaying flowering, reducing 1000 kernel weight, and grain yield. Additionally, the high rate of 2,4-D ester also caused excessive crop injury and reduced grain yield. Dicamba was the safest of the three growth regulator herbicides evaluated with only the high rate reducing yields compared to pyrasulfotole & bromoxynil applied alone. This study suggests that applying pyrasulfotole & bromoxynil at 246 g ha^{-1} + 560 g ha^{-1} atrazine to sorghum without growth regulators is the safest, however if added weed control is necessary, 140 g ha^{-1} of 2,4-D ester or dicamba may be used effectively without causing significant yield reductions.

SOURCES OF MATERIALS

1TeeJet Spraying Systems, Wheaton, IL 60189-7900.

2Dickey-John GACII grain analysis computer, Dickey-John Corporation, P.O. Box 10, Auburn, IL 62165.

3SAS version 8.2, SAS Institute Inc., 100 SAS Campus Drive, Cary, NC 27513.

ACKNOWLEDGEMENTS

The authors thank Bayer Crop Science and the Kansas Grain Sorghum Commission for funding the project.

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Table 3-1. Weather data for time of application to 3- and 6-leaf grain sorghum at Manhattan, KS in 2009 and 2010.

	3-leaf collar		6-leaf collar	
Application Date	6/28/2009	6/15/2010	7/6/2009	6/22/2010
Time of day	8:30 AM	12:20 PM	10:30 AM	7:30 AM
Crop Height (cm)	8-10	5-8	18-20	18-22
Air Temperature (C)	27	27	26	27
% Relative Humidity	62	81	66	79
Wind Speed ($m\ s^{-1}$)	1.1	1.8	0.7	3.1
Wind Direction	North	Northeast	North	South
Dew Presence	Yes	No	No	Yes
Soil Temperature (C)	21	24	20	23
Soil Moisture	Good	Wet	Excellent	Wet
% Cloud Cover	0	80	25	40

Table 3-2. Visible injury of grain sorghum at 7, 14, 21, and 28 DAT as affected by pyrasulfotole & bromoxynil applied alone or with different growth regulator herbicides and rates applied at two growth stages at Manhattan, KS in 2009 and 2010.

		Visible injury							
		7 DAT		14 DAT		21 DAT		28 DAT	
Growth regulator[a]	Rate	2009	2010	2009	2010	2009	2010	2009	2010
3-leaf timing	g ha^{-1}	---------- % ----------							
none	0	7	13	3	13	1	8	2	0
2,4-D amine	140	12	15	5	24	6	14	6	4
2,4-D amine	280	18	16	5	35	8	18	8	5
2,4-D ester	140	8	16	6	27	3	16	7	1
2,4-D ester	280	16	18	9	31	8	16	7	5
Dicamba	140	7	13	12	16	4	10	4	2
Dicamba	280	14	14	4	18	7	10	6	4
6-leaf timing									
none	0	6	12	4	4	4	2	0	1
2,4-D amine	140	8	19	7	10	4	3	0	1
2,4-D amine	280	14	30	8	16	9	8	2	4
2,4-D ester	140	6	14	4	7	1	3	0	0
2,4-D ester	280	10	20	5	8	5	3	0	1
Dicamba	140	6	12	6	3	3	1	0	0
Dicamba	280	11	20	8	8	4	5	0	1
LSD ≤ 0.05		ns	3	ns	7	2	ns	2	2

[a]All treatments include pyrasulfotole & bromoxynil at 246 g ha^{-1} + atrazine 560 g ha^{-1}+ ammonium sulfate 2.8 kg ha^{-1}.

Table 3-3. Visible injury of grain sorghum at 7, 14, 21, 28 DAT as affected by pyrasulfotole & bromoxynil + growth regulator herbicides applied at two growth stages in Manhattan, KS, 2009 and 2010.

	Visible injury							
	7 DAT		14 DAT		21 DAT		28 DAT	
Growth regulator[a]	2009	2010	2009	2010	2009	2010	2009	2010
3-leaf timing	---------- % ----------							
2,4-D amine	15 *(0.0001)*[b]	16 *(0.2186)*	7 *(0.0002)*	30 *(0.0001)*	7 *(0.0001)*	16 *(0.0001)*	7 *(0.0001)*	5 *(0.0001)*
2,4-D ester	12 *(0.0043)*	17 *(0.0784)*	5 *(0.0230)*	29 *(0.0001)*	5 *(0.0001)*	16 *(0.0001)*	7 *(0.0001)*	3 *(0.0001)*
Dicamba	11 *(0.0404)*	13 *(1)*	5 *(0.0230)*	17 *(0.1609)*	5 *(0.0001)*	10 *(0.2229)*	5 *(0.0033)*	3 *(0.0001)*
none	7	13	3	13	1	8	2	0
6-leaf timing								
2,4-D amine	11 *(0.0023)*	25 *(0.0001)*	9 *(0.0016)*	13 *(0.0019)*	6 *(0.0048)*	5 *(0.0528)*	1 *(0.1681)*	2 *(0.0321)*
2,4-D ester	8 *(0.1384)*	17 *(0.0098)*	6 *(0.5772)*	7 *(0.2563)*	3 *(0.5545)*	3 *(0.4538)*	0 *(1.0)*	0 *(0.5823)*
Dicamba	8 *(0.0897)*	16 *(0.0784)*	6 *(0.3738)*	5 *(0.7145)*	3 *(0.8823)*	3 *(0.5395)*	0 *(1.0)*	0 *(0.4640)*
none	6	12	4	4	4	2	0	1

[a]All treatments include pyrasulfotole & bromoxynil 246 g ha^{-1} + atrazine 560 g ha^{-1} + ammonium sulfate 2.8 kg ha^{-1}.

[b]Values in parenthesis indice p-values when contrasted to pyrasulfotole & bromoxynil alone.

Table 3-4. Visible injury of grain sorghum at 7, 14 ,21 and 28 DAT as affected by pyrasulfotole & bromoxynil + growth regulator herbicide rate applied to grain sorghum at two growth stages in Manhattan, KS, 2009 and 2010.

		Visible injury							
		7 DAT		14 DAT		21 DAT		28 DAT	
Application timing	Growth regulator rate[a]	2009	2010	2009	2010	2009	2010	2009	2010
3-leaf	g ha^{-1}	---------- % ----------							
	0	7	13	3	13	1	8	2	0
	140	9 (*0.2070*)[b]	14 *(0.5270)*	4 *(0.0011)*	22 *(0.0009)*	4 *(0.0003)*	13 *(0.0002)*	5 *(0.0002)*	2 *(0.0001)*
	280	16 *(0.0001)*	16 *(0.1357)*	7 *(0.0001)*	28 *(0.0001)*	7 *(0.0001)*	15 *(0.0002)*	7 *(0.0001)*	5 (*0.0001)*
6-leaf	0	6	12	4	4	4	2	0	1
	140	7 *(0.4276)*	15 *(0.1239)*	5 *(0.6932)*	6 *(0.3673)*	3 *(0.2132)*	2 *(0.7720)*	0 *(1.0)*	0 *(0.3657)*
	280	11 *(0.0005)*	23 *(0.0001)*	9 *(0.0004)*	10 *(0.0162)*	6 *(0.0069)*	5 *(0.0427)*	1 *(0.3270)*	2 *(0.1245)*

[a]All treatments include pyrasulfotole & bromoxynil 246 g ha^{-1} + atrazine 560 g ha^{-1} + ammonium sulfate 2.8 kg ha^{-1}.

[b]Values in parenthesis indicate p-values when contrasted to pyrasulfotole & bromoxynil applied without growth regulators.

Table 3-5. Days to flowering from grain sorghum emergence, plant height, heads per plant, 1000 kernel weight, seeds per panicle, and grain yield in response to pyrasulfotole & bromoxynil applied alone or with growth regulators at Manhattan, KS, in 2009 and 2010.

		2009 & 2010		2009			
Growth regulator[a]	Rate	Days to flower	Plant height	Heads per plant	Seeds per panicle	1000 kernel weight	Yield
	g ha^{-1}	days	cm	#	#	g	kg ha^{-1}
none	0	56.1	128	1.12	2310	27.4	9500
2,4-D amine	140	57.0	127	1.20	2210	25.3	8680
2,4-D amine	280	58.4	125	1.12	2300	24.8	8300
2,4-D ester	140	57.1	128	1.18	2240	27.0	9460
2,4-D ester	280	57.6	128	1.12	2220	26.8	8860
dicamba	140	56.2	129	1.15	2230	27.6	9420
dicamba	280	57.6	130	1.14	2340	26.2	9150
LSD ≤ 0.05		0.8	3	0.06	ns	1.0	480

[a]All treatments include pyrasulfotole & bromoxynil 246 g ha^{-1} + atrazine 560 g ha^{-1} + ammonium sulfate 2.8 kg ha^{-1}.

Table 3-6. Days to flowering from grain sorghum emergence, heads per plant, 1000 kernel weight, and seeds per panicle as affected by timing of application in 2009 and 2010.

	2009 & 2010	2009			
Application timing	Days to flower	Heads per plant	Seeds per panicle	1000 kernel weight	Yield
	days	#	#	g	kg ha^{-1}
3-leaf sorghum	57.7	1.09	2380	26.9	9170
6-leaf sorghum	56.5	1.20	2150	26.0	8940
LSD ≤ 0.05	0.4	0.03	80	0.6	ns

Table 3-7. Days to flowering from grain sorghum emergence, plant height, heads per plant, 1000 kernel weight, seeds per panicle, and grain yield as affected by application timing of pyrasulfotole & bromoxynil + growth regulator herbicides.

	2009 & 2010		2009			
Growth regulator[a]	Days to flower	Plant height	Heads per plant	Seeds per panicle	1000 kernel weight	Yield
3-leaf timing	days	cm	#	#	g	kg ha^{-1}
2,4-D amine	58.1 *(0.0129)*[b]	127 *(0.8320)*	1.10 *(0.8620)*	2450 *(0.7915)*	25.8 *(0.0173)*	8820 *(0.0153)*
2,4-D ester	58.2 *(0.0082)*	128 *(0.9747)*	1.09 *(0.7023)*	2310 *(0.2215)*	27.3 *(0.9532)*	9200 *(0.2301)*
dicamba	57.6 *(0.0732)*	130 *(0.3356)*	1.09 *(0.7544)*	2350 *(0.4549)*	27.2 *(0.8826)*	9290 *(0.3810)*
none	56.5	128	1.10	2420	27.3	9550
6-leaf timing						
2,4-D amine	57.3 *(0.0129)*	124 *(0.2253)*	1.22 *(0.0485)*	2060 *(0.1476)*	24.3 *(0.0001)*	8160 *(0.0001)*
2,4-D ester	56.4 *(0.2306)*	128 *(0.9106)*	1.20 *(0.1147)*	2140 *(0.5983)*	26.4 *(0.1164)*	9120 *(0.2473)*
dicamba	56.2 (*0.4384)*	129 *(0.7426)*	1.20 *(0.1688)*	2220 *(0.7912)*	26.6 *(0.1689)*	9280 *(0.5552)*
none	55.8	128	1.15	2190	27.4	9450

[a]All treatments include pyrasulfotole & bromoxynil 246 g ha^{-1} + atrazine 560 g ha^{-1} + ammonium sulfate 2.8 kg ha^{-1}.

[b]Values in parenthesis indicate p-values when contrasted to pyrasulfotole & bromoxynil applied without growth regulators.

Table 3-8. Days to flower from sorghum emergence, plant height, heads per plant, 1000 kernel weight, seeds per panicle, and grain yield as affected by pyrasulfotole & bromoxynil + growth regulators applied at two growth stages in Manhattan, KS, 2009 and 2010.

		2009 & 2010		2009			
Growth regulator rate[a]		Days to flower	Plant height	Heads per plant	Seeds per panicle	1000 kernel weight	Yield
3-leaf	g ha^{-1}	days	cm	#	#	g	kg ha^{-1}
	none	56.5	128	1.10	2420	27.3	9550
	140	57.4 *(0.0981)*[b]	128 *(0.8139)*	1.11 *(0.7494)*	2380 *(0.6429)*	26.9 *(0.4560)*	9380*(0.5459)*
	280	58.5 *(0.0020)*	128 *(0.7770)*	1.07 *(0.3525)*	2360 *(0.4541)*	26.7 *(0.2938)*	8830*(0.0108)*
6-leaf	none	55.8	128	1.15	2190	27.4	9450
	140	56.1 *(0.5296)*	128 *(0.9308)*	1.23 *(0.0144)*	2070 *(0.1466)*	26.4 *(0.0824)*	8990*(0.0948)*
	280	57.2 *(0.0136)*	127 *(0.6307)*	1.18 *(0.3161)*	2210 *(0.8049)*	25.2 *(0.0004)*	8710 *(0.0093)*

[a]All treatments include pyrasulfotole & bromoxynil 246 g ha^{-1} + atrazine 560 g ha^{-1} + ammonium sulfate 2.8 kg ha^{-1}.

[b]Values in parenthesis indicate p-values when contrasted to pyrasulfotole & bromoxynil applied without growth regulators.

Table 3-9. Days to flower, plant height, heads per plant, 1000 kernel weight, seeds per panicle, and grain yield as affected by pyrasulfotole & bromoxynil applied at two growth stages at Manhattan, KS, in 2009 and 2010.

	2009 & 2010		2009			
Treatment	Days to flower	Plant height	Heads per plant	Seeds per panicle	1000 kernel weight	Yield
	days	cm	#	#	g	kg ha^{-1}
Untreated Check	55.1	130	1.23	2200	28.4	10020
3-leaf pyrasulfotole & bromoxynil[a]	56.5 *(0.0476)*[b]	128 *(0.4310)*	1.10 *(0.0034)*	2420 *(0.0362)*	27.3 *(0.1251)*	9550 (*0.1637*)
6-leaf pyrasulfotole & bromoxynil[a]	55.8 *(0.3045)*	128 *(0.4988)*	1.15 *(0.0453)*	2190 *(0.9678)*	27.4 *(0.1660)*	9450 (*0.0956*)

[a]Treatments include pyrasulfotole & bromoxynil 246 g ha^{-1} + atrazine 560 g ha^{-1} + ammonium sulfate 2.8 kg ha^{-1}.

[b]Values in parenthesis indicate p-values when contrasted to pyrasulfotole & bromoxynil applied without growth regulators.

Chapter 4 - Palmer Amaranth Differential Response to Pyrasulfotole and Bromoxynil

ABSTRACT

Palmer amaranth, a serious weed in Kansas crops, has developed resistance to several herbicide modes of action in recent years. Greenhouse and field experiments were conducted near Manhattan, Kansas to evaluate the response of two suspected resistant (R1 & R2), and one susceptible (S), Palmer amaranth populations to pyrasulfotole & bromoxynil, in a 1:8 ratio, atrazine, and tembotrione. Herbicides were applied to 7 and 22 cm Palmer amaranth in the greenhouse and field experiments, respectively. Control was evaluated 14 days after treatment (DAT) in the field experiments and 28 DAT in the greenhouse experiments. Percent mortality was determined at each evaluation. Percent mortality increased as pyrasulfotole & bromoxynil rates increased in both field and greenhouse experiments. R1 and R2 populations had a resistance index (RI) of 11.0 and 5.2 with pyrasulfotole & bromoxynil in the greenhouse and 6.6 and 4.8 in the field. Tembotrione at 92 g ha^{-1} controlled the S population 100% in both greenhouse and field experiments, and controlled 66 and 82% of R1 and R2 in the greenhouse 28 DAT, and 40 and 63% in the field 14 DAT. Atrazine alone at 1120 g ha^{-1} controlled R1 and R2 6% 28 DAT in the greenhouse and 4 to 10% of R1 and R2 14 DAT in the field. The addition of atrazine to tembotrione or bromoxynil & pyrasulfotole increased control compared to either herbicide applied alone. The rate of pyrasulfotole & bromoxynil to kill 50% of the R1 and R2 populations was 661 and 313 g ha^{-1} in the greenhouse experiments, and 543 and 392 g ha^{-1} in the field experiments. The use rate of pyrasulfotole & bromoxynil at 246 g ha^{-1} would provide commercially unacceptable control of the R1 or R2 Palmer amaranth populations.

Nomenclature: Pyrasulfotole & bromoxynil; atrazine; tembotrione; Palmer amaranth, *Amaranathus palmeri* S. Wats.

Key words: HPPD-inhibitor resistance, herbicide-resistant Palmer amaranth, pyrasulfotole & bromoxynil.

INTRODUCTION

Palmer amaranth (*Amaranthus palmeri* S. Wats.) is a troublesome summer annual weed that is widely distributed across the Southern half of the United States and infests many crops such as corn, soybeans, grain sorghum, and cotton (Horak et al. 1994; Massinga et al. 2001; Klingaman and Oliver 1994; Rowland et al. 1999; Moore et al. 2004). Palmer amaranth is fast growing, a prolific seed producer, and has a long germination window, which are all attributes to its weedy characteristic (Keeley et al. 1987; Steckel et al. 2004; Horak and Loughin 2000). Additionally, Palmer amaranth populations exist that are resistant to Acetolactate synthase (ALS)-inhibitors, dinitroanilines, glycines, and photosystem II-inhibitor herbicides in thirteen states (Heap 2011).

Palmer amaranth is a dioecious plant that can reach heights over 2.7 m, has a growth rate of 0.21 cm GDD^{-1}, and produces over 600,000 seeds $plant^{-1}$ (Horak and Loughin 2000; Keeley et al. 1987). These traits make Palmer amaranth capable of reducing crop yield by competing for light, nutrients, and water (Radosevich et al. 2007). For example, 8 plants m^{-1} row reduced corn grain yield by 91%, and reduced soybean yields by 78% (Massinga et al. 2001; Bensch et al. 2003). Grain sorghum LAI and panicle weight reductions of 63 and 83% were observed with mixed stands of smooth pigweed and Palmer amaranth at 12 plants m^{-2} (Graham et al. 1988).

Producers that shifted from conventional till to reduced or no-till, to conserve soil moisture, reduce erosion, and improve soil physical properties, extensively rely on chemical methods to control Palmer amaranth as well as other weeds in the field (Burnside et al. 1980; Wicks et al. 1988). Monocropping and the use of herbicide resistant crops, such as glyphosate tolerance, have led to the repeated use of herbicides with the same mode of action on fields for many years (Regehr and Morishita 1989; Culpepper et al. 2006). Due to the obligate out-crossing

of Palmer amaranth and repeated selection pressure from intensive herbicide use in cropping systems, herbicide-resistant accessions have developed (Martin 2000; Heap 2011).

Palmer amaranth resistance to glyphosate was first reported in Georgia cotton fields in 2006, and resistant populations are currently found in nine states (Heap 2011; Culpepper et al. 2006). Gaines et al. (2011) reported glyphosate resistance is caused by increased plant's genomic copy number of the enzyme 5-enlopyruvylshikimate-2-phosphate synthase (EPSPS) and that 30 to 50 EPSPS copies are needed to survive glyphosate rates from 0.5 to 1.0 kg ha^{-1}. A study in Arkansas reported 12.5 kg ha^{-1} of glyphosate was needed to control a resistant Palmer amaranth biotype 95% (Norsworthy et al. 2008).

ALS-inhibitor resistant Palmer amaranth was first confirmed in Kansas in 1991, only nine years after the first ALS-inhibiting herbicide was commercialized (Heap 2011; Horak and Peterson 1995). Resistance to herbicides in the chemical groups of sulfonylureas, imidazolinones, and sulfonamides currently infest approximately 2.2 million ha^{-1} in the US. Resistance to ALS-inhibiting herbicides is generally due to a single amino acid change in the active binding site in most species including Palmer amaranth (Woodworth et al. 1996).

Triazine herbicides are commonly used in corn and sorghum production and Palmer amaranth resistance to triazine was first reported in 1995 (Heap 2011). The resistance mechanism is often due to an altered target site in the plastoquinone protein (Q_b) within photosystem II. A change in the amino acid sequence of the Q_b protein causes the altered binding site which reduces the binding of triazine herbicides (Foes et al. 1998; Hess 2000).

The 4-Hydroxyphenylpryruvate dioxygenase (HPPD)-inhibiting herbicides such as isoxaflutole, mesotrione, pyrasulfotole, tembotrione, and topramezone provide good control of annual broadleaf weeds including Palmer amaranth (Thompson et al. 2011). The HPPD enzyme

is essential for the biosynthesis of plastoquinone (PQ) and tocopherols in plants (Freigang et al. 2008). Plastoquinone is an important co-factor in electron transport during photosynthesis that shuttles electrons from photosystem II to photosystem I, where sunlight is converted into chemical energy (Freigang et al. 2008). Plastoquinone is also an important cofactor for desaturase enzymes such as phytoene, which are needed for carotenoid biosynthesis. Carotenoids protect the chloroplasts from excessive sunlight by quenching excess triplet energy allowing chlorophyll to return to a ground energy state (Freigang et al. 2008; Hess 2000). When the HPPD enzyme is blocked, PQ levels decline and increases sensitivity to photosystem II-inhibitors. Blocking the HPPD enzyme also stops carotenoid synthesis, leaving the plant with bleached foliage due to excess energy destroying chlorophyll and membranes, ultimately resulting in plant death (Freigang et al. 2008). Atrazine or other PSII-inhibitors are commonly applied with HPPD-inhibitors due to the synergism between these herbicides (Abendroth et al. 2006).

Currently, there are no reported cases of Palmer amaranth resistance to HPPD-inhibitors; however, in 2010 HPPD-inhibitor resistant tall waterhemp (*Amaranthus tuberculatus* Sauer) was reported in Illinois and Iowa (Hausman et al. 2011; McMullan and Green 2011). In Illinois, repeated use of mesotrione, topramezone, or tembotrione for 7 years in continuous seed corn production resulted in a population developing 10-fold resistance to mesotrione based on greenhouse experiments (Hausman et al. 2011). Additionally, tembotrione and topramezone provided inadequate control. Tank-mixing atrazine with mesotrione, tembotrione, or topramezone increased efficacy 15 to 30% even though the population was resistant to atrazine. Preliminary research on the same waterhemp population by Tranel et al. (2011) indicated a non-target resistance mechanism inherited by a single-dominant gene. The population was also resistant to ALS-inhibiting herbicides.

A field in Iowa that had been in a seed corn-soybean rotation for 10 years had tall waterhemp survive a mesotrione + atrazine tank-mix that was applied to the seed corn. HPPD-inhibitors were used each year seed corn was grown. Greenhouse experiments conducted by McMullan and Green (2011) reported that the population resistance index (RI) was 7.7, 10.5, and 28.2 when treated with mesotrione, atrazine, or thifensulfuron, respectively. Similar trends were observed to that of Hausman et al. (2011) when atrazine, mesotrione, tembotrione, and topramezone provided inadequate control of a suspected resistant waterhemp population.

In 2009, a sorghum plot was established in Stafford County, KS to demonstrate weed control in sorghum with pyrasulfotole & bromoxynil, in a 1:8 ratio. A population of Palmer amaranth was not controlled with pyrasulfotole & bromoxynil at 246 g ha^{-1} (Personal communication, Bayer Crop Science 2009). According to the grower, there was no prior history of HPPD-inhibitor herbicide use on the field; however, previous use of mesotrione in nearby fields was common and may have increased the likelihood of resistance. The objective of this study was to determine if Palmer amaranth populations collected from the field responds differently than a known susceptible population to pyrasulfotole & bromoxynil, tembotrione, and atrazine in greenhouse and field environments.

MATERIALS AND METHODS

Plant Materials

In October 2009, Palmer amaranth seed were collected from a grower's field in Stafford County, KS where pyrasulfotole & bromoxynil did not control Palmer amaranth. Seed were collected from surviving plants within the pyrasulfotole & bromoxynil-treated area (R1). Another collection of seed was gathered from the opposite end of the same field that was not treated with pyrasulfotole & bromoxynil (R2). The seed were threshed from R1 and R2 plants,

placed in storage at -5 C for 30 days, and stored at room temperature until planted. A susceptible (S) population was collected from Ashland Bottoms research station near Manhattan, KS.

Greenhouse Study

In January-March 2010, seed from R1, R2, and S populations were sown in 13-cm diameter containers filled with Miracle-Gro potting mix[1]. Plants were grown under greenhouse conditions of 29/25 ± 2 C day/night temperature with a 16/8-h day/night period. The supplemental light intensity was 84 µmol m^{-2} s^{-1}. Plants were watered with flood irrigation as needed. Seedlings were thinned to four plants per container. The Palmer amaranth populations were treated when plants were 7 to 15 cm in height with six rates of pyrasulfotole & bromoxynil at 62, 123, 246, 492, 984, and 1968 g ha^{-1} + ammonium sulfate 2.5% v/v; pyrasulfotole & bromoxynil 246 g ha^{-1} + atrazine 1120 g ha^{-1} + ammonium sulfate 2.5% v/v; atrazine 1120 g ha^{-1} + crop oil concentrate[2] (COC) 1% v/v; tembotrione 92 g ha^{-1} + COC 1% v/v; and tembotrione 92 g ha^{-1} + atrazine 1120 g ha^{-1} + COC 1% v/v. Pyrasulfotole & bromoxynil at 246 g ha^{-1} was the suggested use rate (Bayer Crop Science, personal communication 2009). An untreated check of each population was included. Herbicides were applied with a bench-type sprayer[3] equipped with an 80015LP[4] spray tip to deliver 187 L ha^{-1} at 138 kPa.

Visual injury ratings of control were made 28 days after treatment (DAT) using a scale of 0 to 100% where 0% = no injury and 100% = dead. At 28 DAT, plant mortality was determined by dividing the number of dead plants by the total number of plants in each pot.

Field Study

In July 2010, seed from R1, R2, and S populations were sown in 50- by 35-cm flats filled 5-cm deep with Miracle-Gro potting mix in the greenhouse. At the cotyledon to 1-leaf stage, individual Palmer amaranth seedlings were transplanted to 6.5- by 6.5-cm containers. The

roots were rinsed free of soil to ensure no seeds were attached to the transplants. Nine days after establishing in pots, plants were transplanted to the field at the Ashland Bottoms research station near Manhattan, KS. Four plants of each of three biotypes were transplanted into 3- by 3-m plots, placed in 76 cm rows spaced 30 cm apart within the rows for a total of 12 plants per plot. The Palmer amaranth populations were treated when plants were 15 to 27 cm in height with pyrasulfotole & bromoxynil at 62, 123, 246, 492, 984, and 1968 g ha^{-1} + ammonium sulfate 2.5% v/v; pyrasulfotole & bromoxynil 246 g ha^{-1} + atrazine 1120 g ha^{-1} + ammonium sulfate 2.5% v/v; atrazine 1120 g ha^{-1} + crop oil concentrate[2] (COC) 1% v/v; tembotrione 92 g ha^{-1} + COC 1% v/v. An untreated check of each population was included. Herbicide treatments were applied with a CO_2 pressurized backpack sprayer equipped with TurboTee[4] 11002 flat fan nozzle tips to deliver 140 L ha^{-1} at a pressure of 234 kPa. Weather data were recorded at the time of application (Table 4-1).

Visual ratings of herbicide injury of R1, R2, and S populations were made 14 DAT as previously described. Plant mortality was determined at 14 DAT as described previously. Surviving plants were clipped at the ground level, dried at 65 C for 7 days, and then weighed.

The experimental design for the greenhouse study was a randomized complete block with a factorial arrangement of 11 herbicide treatments by 3 populations, with four replications and was conducted twice, one month apart. The experimental design for the field study was a split plot arrangement with herbicide treatment as the main plot and Palmer amaranth population as sub-plot, replicated four times and was conducted twice, two weeks apart. All data were subjected to ANOVA using SAS[5] 8.1, and means were separated using Fishers protected LSD $\leq$ 0.05. Nonlinear regression analysis of the six pyrasulfotole & bromoxynil rates were used to

determine the rate required to cause 50% injury (ED_{50}) and 50% mortality (LD_{50}). The control or mortality response, y, herbicide rate, 'x' were:

$$y = \frac{a}{1 + \exp(-(-x - ED_{50})/b))}$$

where 'a' is the upper limit or 100%, 'b' is the slope. The R1, R2, and S calculated values for each parameter are shown in Table 4-5 and Table 4-6. Resistance index (RI) was calculated by dividing the ED_{50} or LD_{50} of R1 or R2 populations by the ED_{50} or LD_{50} of the S population.

RESULTS AND DISCUSSION

Greenhouse Study

All treatments injured or controlled the R1, R2, and S populations, 28 DAT. The R1 and R2 populations were injured less by pyrasulfotole & bromoxynil than the S population. Injury symptoms appeared approximately 4 d after application. Pyrasulfotole & bromoxynil caused leaf chlorosis, leaf necrosis, and some bleaching to the terminal bud to all populations. Tembotrione caused foliar bleaching, and leaf necrosis injury symptoms similar to that observed by Hausman et al. (2011). Atrazine symptoms consisted of leaf chlorosis and slight necrosis in R1 and R2 populations to severe necrosis in the S population.

Tembotrione alone or tank-mixed with atrazine provided the greatest injury to the R1 and R2 populations and completely controlled the S population by 28 DAT (Table 4-2). A trend of increasing injury to the R1 and R2 populations was observed when tembotrione was applied with atrazine. Atrazine applied alone caused initial injury to R1 and R2 populations following application, however, injury declined to 8 to 20% by 28 DAT. Atrazine alone provided the least control of all populations. Injury was not consistent with that observed in triazine-resistant weeds

having an altered binding site from a change in the amino acid sequence of the Q_b protein (Foes et al. 1998; Hess 2000).

All herbicide treatments provided greater plant mortality in the S population than the R1 or R2 populations (Table 4-2). Atrazine alone controlled 6% of R1 or R2 populations and 78% of the S population. This suggests that the R1 and R2 populations are triazine-resistant and that a few individuals in the S population may also be triazine-resistant. The S population controlled 85% or more with the remaining treatments. Pyrasulfotole & bromoxynil and pyrasulfotole & bromoxynil + atrazine failed to provide adequate control of R1 and R2 populations; however the addition of atrazine increased injury by 21% in the R2 than the R1 population. The R1 population was collected from plants surviving a pyrasulfotole & bromoxynil application, thus decreases the likelihood of producing susceptible offspring and may explain why control of the R1 population with HPPD-inhibitor herbicides was less than control of the R2 population. The R1 and R2 mortality increased with the addition of atrazine to tembotrione by 22 and 16%, respectively. A similar trend of greater control of R2 than R1 population was observed with the tembotrione treatments.

Regression models show that injury on all populations increased with increasing rates of pyrasulfotole & bromoxynil, 28 DAT (Figure 4-1). All model parameters are shown in Table 4-5 and Table 4-6. The R1 population had the highest ED_{50} of 576 g ha^{-1} at 28 DAT which was greater than the ED_{50} of R2 and S populations. The resistance index (RI) for R1 and R2 populations 28 DAT was 9.7 and 4.6 which were similar to results reported by McMullan and Green (2011) who observed a 7.7 RI with tall waterhemp when treated with mesotrione. The S populations reached 100% injury with 123 g ha pyrasulfotole & bromoxynil.

The LD_{50} for R1 and R2 were 2.7 and 1.3 times greater than the suggested use rate of pyrasulfotole and bromoxynil in grain sorghum (Figure 4-2). Eight times the suggested use rate, 1968 g ha^{-1}, provided only 90% mortality of R1 or R2. The R1 and R2 populations clearly responded differently to pyrasulfotole & bromoxynil than the S population, which strongly suggests that R1 and R2 populations are resistant to pyrasulfotole & bromoxynil.

Field Study

No treatment by experiment interaction was observed for injury or mortality so data were pooled over experiments. All treatments injured R1, R2, and S Palmer amaranth populations as observed in the greenhouse (Table 4-3). Injury ranged from leaf chlorosis to leaf necrosis with atrazine, pyrasulfotole & bromoxynil, and tembotrione. Pyrasulfotole & bromoxynil and tembotrione treatments also caused foliar bleaching and stunting similar to that observed in the greenhouse. Generally, the R1 population was less responsive to pyrasulfotole & bromoxynil, pyrasulfotole & bromoxynil + atrazine, and tembotrione, followed by R2 and the S populations. The R1 and R2 populations displayed the less injury from atrazine at 1120 g ha^{-1} than S population, which is consistent with the greenhouse findings 28 DAT. The addition of atrazine to pyrasulfotole & bromoxynil completely controlled the S population and increased injury of R1 and R2 15 to 28%. Injury was greater when atrazine was added with pyrasulfotole & bromoxynil. Injury to R1 and R2 populations was less than that observed in the greenhouse with all treatments.

Tembotrione caused the greatest mortality to R1, R2, and S populations (Table 4-3). R1 and R2 populations had greater mortality to treatments in the greenhouse compared to that observed in the field. Atrazine alone provided the least mortality in all three populations, suggesting that atrazine resistance is present in all three populations. The addition of atrazine to

pyrasulfotole & bromoxynil increased mortality in all three populations compared either to atrazine or pyrasulfotole & bromoxynil applied alone (Table 4-4) . Surviving plant dry weights followed similar trends to injury and plant mortality. Tembotrione caused the greatest decrease in dry weight followed by pyrasulfotole & bromoxynil + atrazine. Atrazine applied to R1 or R2 populations had did not reduce plant dry weight compared to the untreated plants indicating these populations are resistant. Pyrasulfotole & bromoxynil alone applied to R1 plants did not reduce plant dry weights compared to the untreated check. All other treatments caused dry weight reductions to R1, R2 and S populations.

Increasing the rate of pyrasulfotole & bromoxynil increased Palmer amaranth injury to all three populations (Figure 4-3). The ED_{50} for R1, R2, and S were 465, 321, and 58 g ha^{-1} at 14 DAT. As observed in the greenhouse, the ED_{50} for R1 was greater than that of R2. The S population ED_{50} was identical to that in the greenhouse. New growth on surviving plants was evident by 14 DAT. The R1 and R2 populations had RI values of 6.6 and 4.8 when treated with pyrasulfotole & bromoxynil (Figure 4-3). The LD_{50} rate for the R1 population was lower in the field compared to greenhouse observations, while R2 and S populations LD_{50} increased in the field (Figure 4-4). The LD_{50} for R1, R2, and S populations were 2.2, 1.6, and 0.3 times the suggested use rate.

Similar trends were observed in the greenhouse and field experiments. R1 appeared to have a slightly greater tolerance to HPPD-inhibitors than R2, but R2 was less responsive to atrazine applied alone. Neither R1 nor R2 populations were controlled 100% with any herbicide treatment. However, all treatments provided more control of the S population than the R1 or R2 populations. Since these populations responded differently, results suggest that HPPD-inhibitors should not be used in the field where these R populations were collected. Palmer amaranth in the

field study generally had a lower response to herbicides compared to greenhouse results. This could be due to the slightly larger plant size when herbicides were applied to Palmer amaranth in the field, or by morphological differences caused by differing environments between the greenhouse and field conditions. The addition of atrazine to pyrasulfotole & bromoxynil or tembotrione increased control in both greenhouse and field experiments which agrees with Abendroth et al. (2006) who stated PSII inhibitors increase HPPD inhibitor efficacy. This suggests that if sorghum producers are to use pyrasulfotole & bromoxynil in their fields, that atrazine should be added to reduce the risk of developing HPPD-inhibitor resistant Palmer amaranth.

Clearly these results show that there is a differential response between the S and R populations and that the R populations will not be controlled by the use rate of pyrasulfotole & bromoxynil in a producers sorghum field.

SOURCES OF MATERIALS

[1]Miracle-gro moisture control potting mix, Scotts miracle-gro products inc, 1411 Scottslawn Road, Marysville, OH 43041.

[2]Prime Oil, Terra International Inc., P. O. Box 6000, Sioux City, IA 51102-6000.

[3]Research track sprayer, De Vries Manufacturing, RR 1, Box 184, Hollandale, MN 56045.

[4]TeeJet, Spraying Systems Co., Wheaton IL 60189-7900.

[5]SAS Institute Inc., 100 SAS Campus Drive, Cary, NC 27513.

ACKNOWLEDGEMENTS

The authors thank Bayer Crop Science for partial funding.

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Table 4-1. Weather data at the time of application for field experiments 1 and 2.

	Experiment 1	Experiment 2
Application Date	7/18/2010	8/4/2010
Time of day	7:00 PM	7:00 AM
Crop height (cm)	18-30	15-25
Air temperature	24	34
Relative humidity %	76	86
Wind speed (m^{-1} s^{-1})	0	0
Wind direction	South Southeast	South
Dew presence	No	No
Soil temperature	28	29
Soil moisture	good	dry
Cloud cover %	10	0

Table 4-2. Palmer amaranth populations R1, R2, and S visual injury and mortality to pyrasulfotole & bromoxynil, atrazine, and tembotrione 28 DAT pooled over two greenhouse experiments.

		Injury			Mortality		
Treatment	Rate	R1	R2	S	R1	R2	S
	g ha^{-1}	---------- % ----------					
Pyrasulfotole & bromoxynil*	246	43	43	88	25	34	85
Pyrasulfotole & bromoxynil + atrazine*	246 + 1120	36	53	97	26	47	97
Atrazine**	1120	20	8	78	6	6	78
Tembotrione**	92	79	85	100	66	81	100
Tembotrione + atrazine**	92 + 1120	94	97	100	88	97	100
LSD ≤ 0.05		---------- 19 ----------			---------- 21 ----------		

*Includes ammonium sulfate 2.5% v/v.

**Includes crop oil concentrate 1% v/v.

Table 4-3. Palmer amaranth populations R1, R2, and S visual injury to pyrasulfotole & bromoxynil, atrazine, and tembotrione 7 and 14 DAT pooled over two field experiments.

		Injury 14 DAT			Mortality		
Treatment	Rate	R1	R2	S	R1	R2	S
	g ha^{-1}	---------- % ----------					
Pyrasulfotole & bromoxynil*	246	12	24	89	6	18	84
Pyrasulfotole & bromoxynil + atrazine*	246 + 1120	27	52	100	19	39	100
Atrazine**	1120	10	4	70	9	0	59
Tembotrione**	92	63	86	100	40	63	100
LSD ≤ 0.05		---------- 20 ----------			---------- 23.0 ----------		

*Includes ammonium sulfate 2.5% v/v.

**Includes crop oil concentrate 1% v/v.

Table 4-4. Palmer amaranth populations R1, R2, and S mortality and biomass in response to pyrasulfotole & bromoxynil, atrazine, and tembotrione 14 DAT pooled over two field experiments.

		Dry Weight		
Treatment	Rate	R1	R2	S
	g ha^{-1}	---------- g ----------		
Pyrasulfotole & bromoxynil*	246	29	19	7
Pyrasulfotole & bromoxynil + atrazine*	246 + 1120	16	10	0
Atrazine**	1120	34	45	12
Tembotrione**	92	8	4	0
Untreated		31	32	36
LSD ≤ 0.05		-------- 16.0 --------		

* Includes ammonium sulfate 2.5% v/v.

**Includes crop oil concentrate 1% v/v.

Table 4-5. Parameter estimates of R1, R2, and S population dose response curves to pyrasulfotole & bromoxynil control 28 and 14 DAT in greenhouse and field experiments, respectively.

	Greenhouse				Field			
Population	*a*	*b*	x_0	R^2	*a*	*b*	x_0	R^2
R1	94	403	576	0.86	54	152	465	0.99
R2	90	169	269	0.97	65	185	321	0.95
S	98	3	57	0.99	93	19	58	0.98

Table 4-6. Parameter estimates of R1, R2, and S population dose response curves to pyrasulfotole & bromoxynil mortality 28 and 14 DAT in greenhouse and field experiments, respectively.

	Greenhouse				Field			
Population	*a*	*b*	x_0	R^2	*a*	*b*	x_0	R^2
R1	90	324	661	0.84	43	139	543	0.99
R2	89	172	313	0.96	56	179	392	0.97
S	98	3	60	0.98	92	30	82	0.99

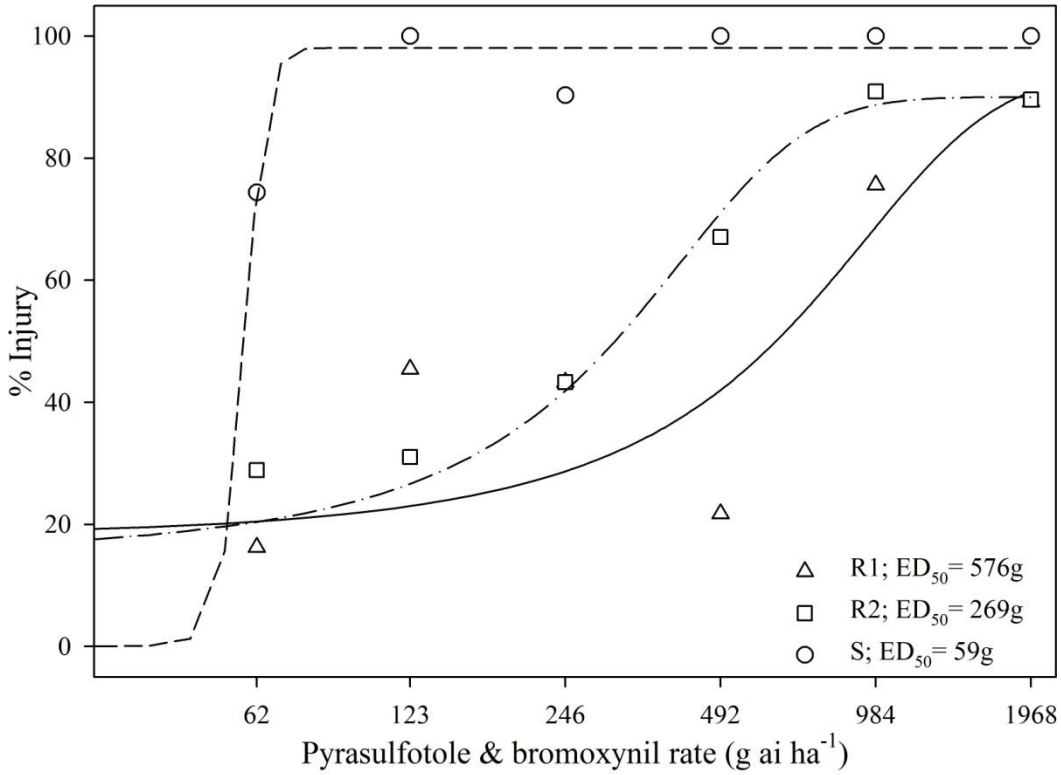

Figure 4-1. Palmer amaranth populations R1, R2, and S visual injury 28 DAT to various pyrasulfotole & bromoxynil rates pooled over two greenhouse experiments.

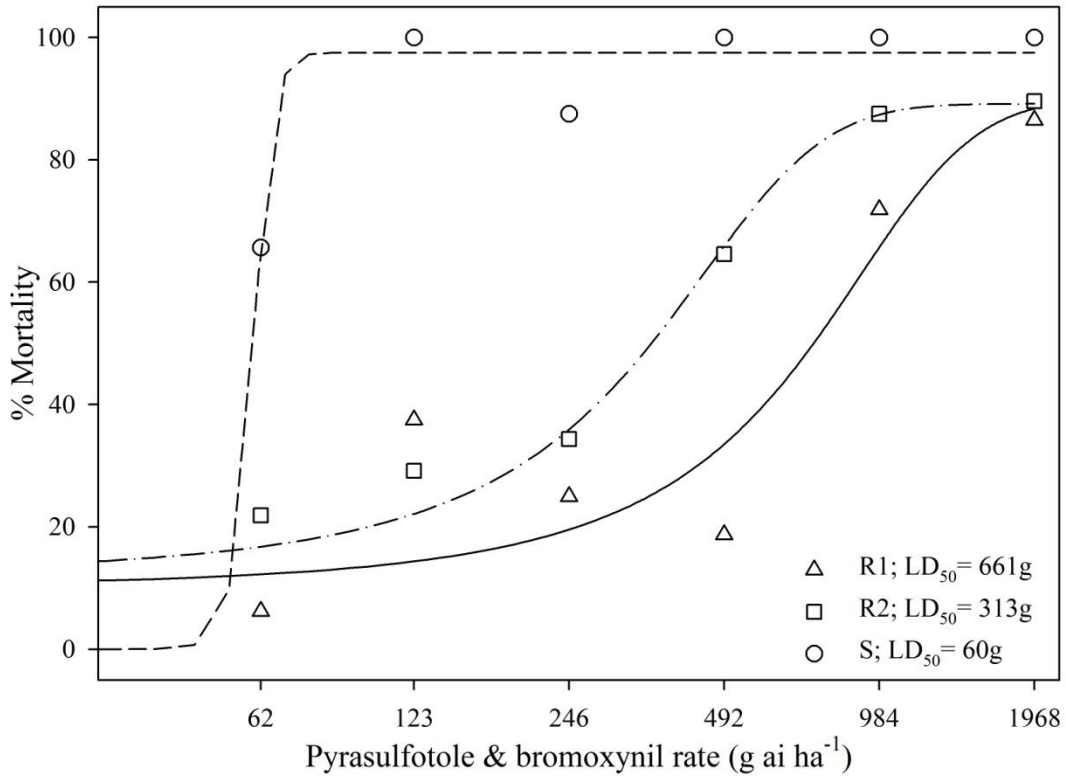

Figure 4-2. Mortality of R1, R2, and S Palmer amaranth populations 28 DAT to various pyrasulfotole & bromoxynil rates pooled over two greenhouse experiments.

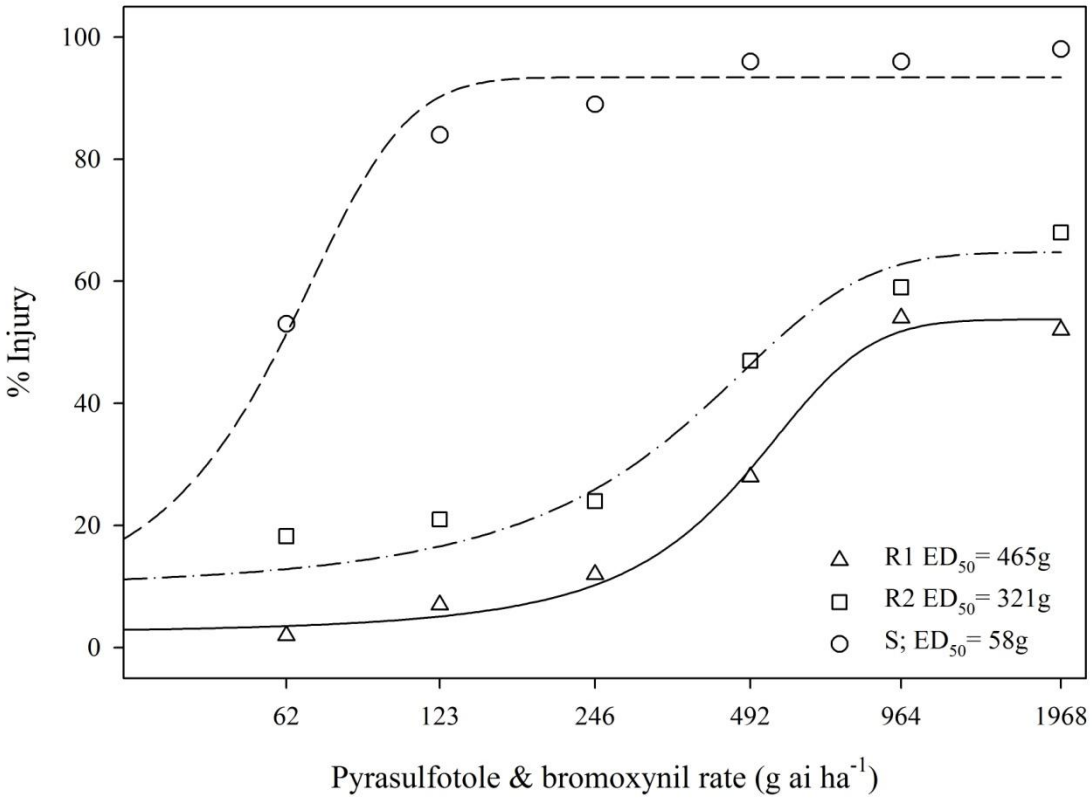

Figure 4-3. Palmer amaranth populations R1, R2, and S visual injury 14 DAT to various pyrasulfotole & bromoxynil rates pooled over two field experiments.

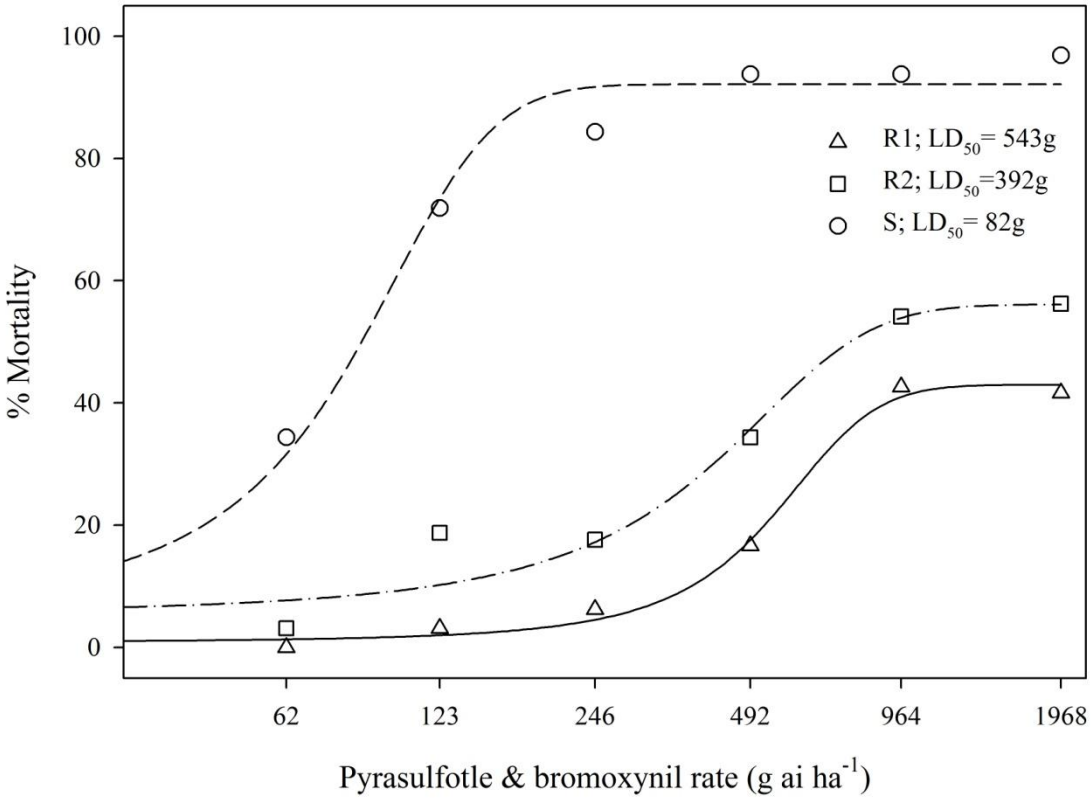

Figure 4-4. Palmer amaranth populations R1, R2, and S mortality 14 DAT from various pyrasulfotole & bromoxynil rates pooled over two field experiments.

Appendix A - Chapter 2 Data

Table A-1. Visible injury to grain sorghum as affected by rate and application timing of pyrasulfotole & bromoxynil applied alone or in combination with 2,4-D ester 7, 14, 21, and 28 days after treatment (DAT) at Manhattan, KS in 2009.

			Injury			
Leaf stage	Treatment[a]	Rate	7 DAT	14 DAT	21 DAT	28 DAT
		g ha^{-1}	---------- % ----------			
1	P&B[b]	246	3	2	1	3
	P&B	492	12	8	7	6
	P&B + 2,4-D ester	246 + 140	4	4	4	4
	P&B + 2,4-D ester	492 + 140	11	10	7	6
4	P&B	246	5	4	3	0
	P&B	492	13	7	6	3
	P&B + 2,4-D ester	246 + 140	10	5	5	3
	P&B + 2,4-D ester	492 + 140	17	8	8	4
7	P&B	246	10	7	3	0
	P&B	492	22	13	5	0
	P&B + 2,4-D ester	246 + 140	15	13	4	0
	P&B + 2,4-D ester	492 + 140	21	15	6	0
10	P&B	246	13	10	5	3
	P&B	492	18	15	7	4
	P&B + 2,4-D ester	246 + 140	12	8	4	2
	P&B + 2,4-D ester	492 + 140	15	11	5	3
13	P&B	246	10	6	4	4
	P&B	492	13	8	6	6
	P&B + 2,4-D ester	246 + 140	12	5	4	4
	P&B + 2,4-D ester	492 + 140	15	10	6	6
Flag leaf	P&B	246	0	1	1	1
	P&B	492	1	3	3	3
	P&B + 2,4-D ester	246 + 140	1	2	3	3
	P&B + 2,4-D ester	492 + 140	4	4	5	5
	LSD ≤ 0.05		4	3	2	2

[a]All treatments include atrazine 560 g ha^{-1} + ammonium sulfate 2.8 kg ha^{-1}.

[b]P&B = pyrasulfotole & bromoxynil.

Table A-2. Visible injury to grain sorghum as affected by rate and application timing of pyrasulfotole & bromoxynil applied alone or in combination with 2,4-D ester 7, 14, 21, and 28 days after treatment (DAT) at Manhattan, KS in 2010.

			Injury			
Leaf stage	Treatment[a]	Rate	7 DAT	14 DAT	21 DAT	28 DAT
		g ha^{-1}	---------- % ----------			
1	P&B[b]	246	4	9	7	4
	P&B	492	9	12	13	7
	P&B + 2,4-D ester	246 + 140	7	13	13	7
	P&B + 2,4-D ester	492 + 140	10	13	11	4
4	P&B	246	18	20	14	4
	P&B	492	26	22	18	6
	P&B + 2,4-D ester	246 + 140	19	23	16	5
	P&B + 2,4-D ester	492 + 140	26	27	22	8
7	P&B	246	12	5	0	0
	P&B	492	16	6	0	0
	P&B + 2,4-D ester	246 + 140	14	4	0	0
	P&B + 2,4-D ester	492 + 140	13	4	0	0
10	P&B	246	15	6	2	2
	P&B	492	16	11	5	3
	P&B + 2,4-D ester	246 + 140	14	7	4	5
	P&B + 2,4-D ester	492 + 140	16	10	6	6
13	P&B	246	7	7	7	7
	P&B	492	11	12	12	12
	P&B + 2,4-D ester	246 + 140	7	7	7	7
	P&B + 2,4-D ester	492 + 140	12	12	12	12
Flag leaf	P&B	246	1	1	1	1
	P&B	492	2	2	2	2
	P&B + 2,4-D ester	246 + 140	1	1	1	1
	P&B + 2,4-D ester	492 + 140	2	2	2	2
	LSD ≤ 0.05		3	3	3	2

[a]All treatments include atrazine 560 g ha^{-1} + ammonium sulfate 2.8 kg ha^{-1}.

[b]P&B = pyrasulfotole & bromoxynil.

Table A-3. Visible injury to grain sorghum as affected by rate and application timing of pyrasulfotole & bromoxynil applied alone or in combination with 2,4-D ester 7, 14, 21, and 28 days after treatment (DAT) at Rossville, KS in 2009.

			Injury			
Leaf stage	Treatment[a]	Rate	7 DAT	14 DAT	21 DAT	28 DAT
		g ha^{-1}	---------- % ----------			
1	P&B[b]	246	9	3	0	0
	P&B	492	20	8	6	3
	P&B + 2,4-D ester	246 + 140	13	4	1	1
	P&B + 2,4-D ester	492 + 140	17	9	8	6
4	P&B	246	13	8	5	5
	P&B	492	32	26	19	17
	P&B + 2,4-D ester	246 + 140	11	6	4	4
	P&B + 2,4-D ester	492 + 140	26	31	26	19
7	P&B	246	13	7	4	1
	P&B	492	22	16	10	4
	P&B + 2,4-D ester	246 + 140	12	6	4	1
	P&B + 2,4-D ester	492 + 140	16	2	7	3
10	P&B	246	15	12	10	4
	P&B	492	20	16	12	5
	P&B + 2,4-D ester	246 + 140	11	9	6	2
	P&B + 2,4-D ester	492 + 140	19	13	10	5
13	P&B	246	10	9	3	5
	P&B	492	13	12	7	6
	P&B + 2,4-D ester	246 + 140	4	3	0	0
	P&B + 2,4-D ester	492 + 140	8	5	3	4
Flag leaf	P&B	246	6	2	4	4
	P&B	492	8	4	5	5
	P&B + 2,4-D ester	246 + 140	7	4	5	5
	P&B + 2,4-D ester	492 + 140	9	5	6	6
	LSD ≤ 0.05		5	6	6	5

[a]All treatments include atrazine 560 g ha^{-1} + ammonium sulfate 2.8 kg ha^{-1}.

[b]P&B = pyrasulfotole & bromoxynil.

Table A-4. Visible injury to grain sorghum as affected by rate and application timing of pyrasulfotole & bromoxynil applied alone or in combination with 2,4-D ester 7, 14, 21, and 28 days after treatment (DAT) at Rossville, KS in 2010.

			Injury			
Leaf stage	Treatment[a]	Rate	7 DAT	14 DAT	21 DAT	28 DAT
		g ha^{-1}	---------- % ----------			
1	P&B[b]	246	6	9	7	6
	P&B	492	12	28	20	19
	P&B + 2,4-D ester	246 + 140	7	17	13	13
	P&B + 2,4-D ester	492 + 140	11	29	18	22
4	P&B	246	7	13	17	11
	P&B	492	16	29	32	22
	P&B + 2,4-D ester	246 + 140	6	10	16	15
	P&B + 2,4-D ester	492 + 140	14	24	28	27
7	P&B	246	7	5	2	0
	P&B	492	8	6	3	0
	P&B + 2,4-D ester	246 + 140	7	5	2	0
	P&B + 2,4-D ester	492 + 140	9	6	4	0
10	P&B	246	11	7	3	3
	P&B	492	17	12	4	4
	P&B + 2,4-D ester	246 + 140	10	7	3	2
	P&B + 2,4-D ester	492 + 140	16	9	3	3
13	P&B	246	7	3	2	2
	P&B	492	11	6	3	3
	P&B + 2,4-D ester	246 + 140	8	3	2	2
	P&B + 2,4-D ester	492 + 140	10	6	4	4
Flag leaf	P&B	246	13	12	12	12
	P&B	492	15	15	15	15
	P&B + 2,4-D ester	246 + 140	13	14	14	14
	P&B + 2,4-D ester	492 + 140	15	16	16	16
	LSD ≤ 0.05		2	5	4	5

[a]All treatments include atrazine 560 g ha^{-1} + ammonium sulfate 2.8 kg ha^{-1}.

[b]P&B = pyrasulfotole & bromoxynil.

Table A-5. Days to flowering from sorghum emergence and plant height as affected by rate and application timing of pyrasulfotole & bromoxynil applied alone or in combination with 2,4-D ester at Manhattan, KS in 2009 and 2010.

			Days to Flowering		Plant Height	
Leaf stage	Treatment[a]	Rate	2009	2010	2009	2010
		g ha^{-1}			------ cm ------	
1	P&B[b]	246	59.8	55.3	129	129
	P&B	492	60.8	55.5	127	131
	P&B + 2,4-D ester	246 + 140	59.3	56.5	129	131
	P&B + 2,4-D ester	492 + 140	60.8	55.5	130	132
4	P&B	246	59.0	57.8	130	127
	P&B	492	59.5	57.8	132	131
	P&B + 2,4-D ester	246 + 140	58.8	57.3	128	128
	P&B + 2,4-D ester	492 + 140	59.8	58.5	127	125
7	P&B	246	56.5	54.3	127	127
	P&B	492	58.8	54.8	130	129
	P&B + 2,4-D ester	246 + 140	59.8	54.3	122	127
	P&B + 2,4-D ester	492 + 140	57.5	54.3	125	126
10	P&B	246	58.3	55.5	126	130
	P&B	492	57.5	55.3	129	125
	P&B + 2,4-D ester	246 + 140	57.8	56.0	131	129
	P&B + 2,4-D ester	492 + 140	58.3	56.5	133	130
13	P&B	246	58.3	55.3	126	130
	P&B	492	58.5	54.8	122	128
	P&B + 2,4-D ester	246 + 140	58.0	55.0	128	137
	P&B + 2,4-D ester	492 + 140	58.0	55.0	126	133
Flag leaf	P&B	246	58.0	54.8	125	130
	P&B	492	57.3	54.5	122	126
	P&B + 2,4-D ester	246 + 140	57.0	54.8	127	131
	P&B + 2,4-D ester	492 + 140	57.3	55.0	122	131
	Untreated Check		58.0	54.1	130	133
	LSD ≤ 0.05		1.5	1.0	4	5

[a]All treatments include atrazine 560 g ha^{-1} + ammonium sulfate 2.8 kg ha^{-1}.

[b]P&B = pyrasulfotole & bromoxynil.

Table A-6. Days to flowering from sorghum emergence and plant height as affected by rate and application timing of pyrasulfotole & bromoxynil applied alone or in combination with 2,4-D ester at Rossville, KS in 2009 and 2010.

			Days to Flowering		Plant Height	
Leaf stage	Treatment[a]	Rate	2009	2010	2009	2010
		g ha^{-1}			------ cm ------	
1	P&B[b]	246	66.3	59.0	133	114
	P&B	492	68.3	61.5	136	119
	P&B + 2,4-D ester	246 + 140	67.8	61.5	135	125
	P&B + 2,4-D ester	492 + 140	69.0	62.3	134	115
4	P&B	246	68.3	58.5	133	119
	P&B	492	70.0	60.8	136	120
	P&B + 2,4-D ester	246 + 140	68.3	60.3	130	118
	P&B + 2,4-D ester	492 + 140	69.3	32.0	135	120
7	P&B	246	66.8	55.3	129	118
	P&B	492	68.0	56.0	134	126
	P&B + 2,4-D ester	246 + 140	66.0	56.3	130	118
	P&B + 2,4-D ester	492 + 140	66.3	56.8	131	118
10	P&B	246	64.5	56.5	132	113
	P&B	492	65.8	56.5	132	117
	P&B + 2,4-D ester	246 + 140	66.0	54.8	137	115
	P&B + 2,4-D ester	492 + 140	67.0	56.5	138	118
13	P&B	246	66.0	55.3	128	116
	P&B	492	66.8	55.3	130	117
	P&B + 2,4-D ester	246 + 140	65.5	55.0	137	120
	P&B + 2,4-D ester	492 + 140	66.3	55.5	136	117
Flag leaf	P&B	246	65.5	54.8	130	118
	P&B	492	65.0	55.8	131	117
	P&B + 2,4-D ester	246 + 140	67.3	55.5	131	120
	P&B + 2,4-D ester	492 + 140	66.0	56.0	131	120
	Untreated Check		66.3	56.1	134	124
	LSD ≤ 0.05		2.1	1.9	6	10

[a]All treatments include atrazine 560 g ha^{-1} + ammonium sulfate 2.8 kg ha^{-1}.

[b]P&B = pyrasulfotole & bromoxynil.

Table A-7. Heads plant^{-1}, seeds panicle^{-1}, 1000 kernel weight, and grain yield as affected by rate and application timing of pyrasulfotole & bromoxynil applied alone or in combination with 2,4-D ester at Manhattan, KS in 2009.

Leaf stage	Treatment[a]	Rate	Heads plant^{-1}	Seeds panicle^{-1}	kernel weight	Yield
		g ha^{-1}			g	kg ha^{-1}
1	P&B[b]	246	1.11	2330	27.9	9244
	P&B	492	1.05	2508	26.9	8897
	P&B + 2,4-D ester	246 + 140	1.14	2239	26.1	8294
	P&B + 2,4-D ester	492 + 140	1.09	2262	26.3	8558
4	P&B	246	1.08	2322	26.0	8431
	P&B	492	1.09	2226	27.1	8135
	P&B + 2,4-D ester	246 + 140	1.03	2306	25.7	7894
	P&B + 2,4-D ester	492 + 140	1.00	2509	25.8	8354
7	P&B	246	1.10	2270	26.8	8924
	P&B	492	1.06	2123	26.3	8108
	P&B + 2,4-D ester	246 + 140	1.08	1999	25.1	7103
	P&B + 2,4-D ester	492 + 140	1.07	2064	24.8	7226
10	P&B	246	1.06	2045	28.0	8446
	P&B	492	1.05	2081	28.5	8355
	P&B + 2,4-D ester	246 + 140	1.08	2094	28.7	8575
	P&B + 2,4-D ester	492 + 140	1.09	2122	28.4	8745
13	P&B	246	1.07	2072	27.1	7985
	P&B	492	1.04	1881	29.7	7501
	P&B + 2,4-D ester	246 + 140	1.05	1924	28.8	7866
	P&B + 2,4-D ester	492 + 140	1.05	1853	30.5	7685
Flag leaf	P&B	246	1.08	1737	34.5	8386
	P&B	492	1.12	1688	35.2	8355
	P&B + 2,4-D ester	246 + 140	1.11	1648	35.3	8258
	P&B + 2,4-D ester	492 + 140	1.07	1460	35.6	7565
	Untreated Check		1.08	2445	26.8	9446
	LSD ≤ 0.05		0.07	260	2.0	777

[a]All treatments include atrazine 560 g ha^{-1} + ammonium sulfate 2.8 kg ha^{-1}.

[b]P&B = pyrasulfotole & bromoxynil.

Table A-8. Heads plant^{-1}, seeds panicle^{-1}, 1000 kernel weight, and grain yield as affected by rate and application timing of pyrasulfotole & bromoxynil applied alone or in combination with 2,4-D ester at Rossivlle, KS in 2009.

Leaf stage	Treatment[a]	Rate	Heads plant^{-1}	Seeds panicle^{-1}	kernel weight	Yield
		g ha^{-1}			g	kg ha^{-1}
1	P&B[b]	246	1.08	2141	31.4	9848
	P&B	492	1.08	5131	31.4	9969
	P&B + 2,4-D ester	246 + 140	1.05	2050	30.6	9398
	P&B + 2,4-D ester	492 + 140	1.08	2165	30.4	9582
4	P&B	246	1.00	1982	31.4	8674
	P&B	492	1.03	1977	31.0	8769
	P&B + 2,4-D ester	246 + 140	1.00	1994	31.1	8753
	P&B + 2,4-D ester	492 + 140	0.97	2072	29.9	8689
7	P&B	246	1.04	1966	29.8	8437
	P&B	492	1.03	1904	30.5	8402
	P&B + 2,4-D ester	246 + 140	1.10	1904	30.2	8750
	P&B + 2,4-D ester	492 + 140	1.08	1929	29.9	8744
10	P&B	246	1.09	1921	34.2	9929
	P&B	492	1.03	1969	32.7	9643
	P&B + 2,4-D ester	246 + 140	1.09	1756	34.8	9414
	P&B + 2,4-D ester	492 + 140	1.09	1844	34.5	9630
13	P&B	246	1.05	1811	32.9	9011
	P&B	492	1.03	1925	32.4	9126
	P&B + 2,4-D ester	246 + 140	1.07	1964	35.4	10272
	P&B + 2,4-D ester	492 + 140	1.02	1870	34.4	9199
Flag leaf	P&B	246	1.03	1862	34.1	9312
	P&B	492	1.11	1773	35.3	9266
	P&B + 2,4-D ester	246 + 140	1.09	1690	35.2	8905
	P&B + 2,4-D ester	492 + 140	1.02	1703	35.4	8642
	Untreated Check		1.07	2245	31.5	10650
	LSD ≤ 0.05		0.07	228	2.2	1282

[a]All treatments include atrazine 560 g ha^{-1} + ammonium sulfate 2.8 kg ha^{-1}.

[b]P&B = pyrasulfotole & bromoxynil.

Table A-9. Heads plant^{-1}, seeds panicle^{-1}, 1000 kernel weight, and grain yield as affected by rate and application timing of pyrasulfotole & bromoxynil applied alone or in combination with 2,4-D ester at Rossville, KS in 2010.

Leaf stage	Treatment[a]	Rate	Heads plant^{-1}	Seeds panicle^{-1}	kernel weight	Yield
		g ha^{-1}			g	kg ha^{-1}
1	P&B[b]	246	0.97	1276	34.4	6025
	P&B	492	0.97	1188	33.3	5341
	P&B + 2,4-D ester	246 + 140	0.94	1043	33.4	5886
	P&B + 2,4-D ester	492 + 140	0.97	1305	34.5	4845
4	P&B	246	0.97	1093	33.6	5020
	P&B	492	0.97	1131	34.3	5468
	P&B + 2,4-D ester	246 + 140	0.96	1246	34.2	6094
	P&B + 2,4-D ester	492 + 140	0.98	1384	35.1	6391
7	P&B	246	0.99	1548	33.0	7002
	P&B	492	0.96	1385	33.4	6206
	P&B + 2,4-D ester	246 + 140	0.98	1376	33.1	6469
	P&B + 2,4-D ester	492 + 140	0.99	1334	33.5	6210
10	P&B	246	1.01	1103	33.8	5115
	P&B	492	1.02	1119	34.4	5728
	P&B + 2,4-D ester	246 + 140	1.02	1074	34.3	5355
	P&B + 2,4-D ester	492 + 140	0.99	999	34.7	4718
13	P&B	246	1.07	1006	34.9	5054
	P&B	492	1.00	1090	34.5	5333
	P&B + 2,4-D ester	246 + 140	0.98	1092	34.2	5022
	P&B + 2,4-D ester	492 + 140	1.00	1039	35.6	5214
Flag leaf	P&B	246	0.99	865	34.7	4272
	P&B	492	1.04	841	34.0	4035
	P&B + 2,4-D ester	246 + 140	1.02	849	34.0	4060
	P&B + 2,4-D ester	492 + 140	0.98	709	34.0	3312
	Untreated Check		0.97	1243	32.1	5596
	LSD ≤ 0.05		0.08	238	1.5	1309

[a]All treatments include atrazine 560 g ha^{-1} + ammonium sulfate 2.8 kg ha^{-1}.

[b]P&B = pyrasulfotole & bromoxynil.

Appendix B - Chapter 3 Data

Table B-1. Visible injury of grain sorghum with pyrasulfotole & bromoxynil applied alone or with growth regulator herbicides applied 7, 14, 21, and 28 DAT at two growth stages at Manhattan, KS, in 2009 and 2010.

		Visible injury							
		7 DAT		14 DAT		21 DAT		28 DAT	
Growth regulator[a]	Rate	2009	2010	2009	2010	2009	2010	2009	2010
3-leaf timing	g ha^{-1}	---------- % ----------							
none	0	7	13	3	13	1	8	2	0
2,4-D amine	140	12	15	5	24	6	14	6	4
2,4-D amine	280	18	16	5	35	8	18	8	5
2,4-D ester	140	8	16	6	27	3	16	7	1
2,4-D ester	280	16	18	9	31	8	16	7	5
Dicamba	140	7	13	12	16	4	10	4	2
Dicamba	280	14	14	4	18	7	10	6	4
6-leaf timing									
none	0	6	12	4	4	4	2	0	1
2,4-D amine	140	8	19	7	10	4	3	0	1
2,4-D amine	280	14	30	8	16	9	8	2	4
2,4-D ester	140	6	14	4	7	1	3	0	0
2,4-D ester	280	10	20	5	8	5	3	0	1
Dicamba	140	6	12	6	3	3	1	0	0
Dicamba	280	11	20	8	8	4	5	0	1
Untreated Check		0	0	0	0	0	0	0	0
LSD ≤ 0.05		4	4	3	6	2	4	2	2

[a]All treatments include pyrasulfotole & bromoxynil at 246 g ha^{-1} + atrazine 560 g ha^{-1}+ ammonium sulfate 2.8 kg ha^{-1}.

Table B-2. Days to flower from sorghum emergence, plant height, heads plant^{-1}, seeds panicle^{-1}, 1000 kernel weight, and grain yield as affected by pyrasulfotole & bromoxynil applied alone or with growth regulator herbicides at two growth stages at Manhattan, KS, 2009 and 2010.

		Days to flower		Plant height		2009			
Growth regulator[a]	Rate	2009	2010	2009	2010	Heads plant^{-1}	Seeds panicle^{-1}	Kernel weight	Yield
3-leaf timing	g ha^{-1}			cm				g	kg ha^{-1}
none	0	57.3	55.8	128	127	1.10	2422	27.3	9549
2,4-D amine	140	58.8	56.0	129	125	1.13	2436	25.9	9008
2,4-D amine	280	60.0	57.5	130	123	1.06	2456	25.7	8634
2,4-D ester	140	58.5	57.5	133	123	1.10	2379	27.4	9688
2,4-D ester	280	59.8	57.0	130	124	1.08	2240	27.3	8709
Dicamba	140	58.5	55.3	131	129	1.11	2331	27.3	9455
Dicamba	280	60.3	56.3	132	130	1.08	2376	27.1	9133
6-leaf timing									
none	0	56.8	54.8	124	133	1.15	2191	27.4	9453
2,4-D amine	140	58.3	55.0	126	126	1.26	1982	24.7	8348
2,4-D amine	280	59.5	56.5	124	122	1.18	2133	24.0	7964
2,4-D ester	140	57.3	55.0	130	128	1.25	2093	26.6	9229
2,4-D ester	280	58.5	55.0	129	127	1.16	2193	26.2	9003
Dicamba	140	56.8	54.3	128	130	1.19	2119	27.9	9392
Dicamba	280	57.5	56.3	128	131	1.21	2311	25.2	9172
Untreated Check		57.0	53.3	128	130	1.23	2195	28.4	10020
LSD ≤ 0.05		1.8	1.4	4	6	0.08	211	1.4	671

[a]All treatments include pyrasulfotole & bromoxynil at 246 g ha^{-1} + atrazine 560 g ha^{-1}+ ammonium sulfate 2.8 kg ha^{-1}.