KOCHIA CONTROL WITH PREEMERGENCE HERBICIDES IN SOYBEANS; DOSE RESPONSE OF THREE KOCHIA POPULATIONS TO GLYPHOSATE; AND RESPONSE OF CORN, SOYBEAN, AND GRAIN SORGHUM TO SAFLUFENACIL

by

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Abstract

Kochia (*Kochia scoparia* (L.) Schrad) is a troublesome and highly competitive weed in many cropping systems in the Great Plains region. It has traditionally been controlled using postemergence (POST) applications of glyphosate, however control is becoming inconsistent. Use of preemergence (PRE) herbicides may help to control kochia. Objectives of this research were to (1) Evaluate the efficacy of selected PRE herbicides in combination with POST applied glyphosate for controlling kochia in soybeans, (2) evaluate a kochia population (Norton) response to various rates of glyphosate compared to previously characterized highly susceptible (Syracuse) and moderately resistant (Ingalls) kochia populations, and (3) quantify the effects of herbicide rate, planting depth, soil pH, and soil type on corn, soybean, and grain sorghum tolerance to saflufenacil. Field studies showed that glyphosate applied alone did not always provide adequate season-long kochia control. In general, PRE herbicide treatments provided effective kochia control. These data suggest that a sequential herbicide program with a PRE herbicide treatment followed by POST glyphosate will provide the most consistent kochia control in soybeans and help minimize the risk of developing herbicide resistant kochia. Greenhouse studies confirmed great variability in kochia susceptibility to glyphosate across three different kochia populations. In general, as glyphosate rates increased, kochia control increased with all three populations. At the field use rate of glyphosate, the Syracuse kochia population was controlled 94% 21 days after treatment (DAT), whereas the Ingalls and Norton populations were controlled 26 and 41% respectively. Nonlinear regression analysis for each population indicated the glyphosate rate required to cause 50% visible control (GR50) was 1.6, 1.1, and 0.31 times the field use rate of 870 g ae/ha for the Ingalls, Norton, and Syracuse kochia populations. Greenhouse studies indicated that soil type had the greatest impact on saflufenacil injury to corn,

soybeans, and sorghum, with crop injury consistently being greater on a fine sandy loam soil with 0.9% organic matter than a silt loam soil with 3.9% organic matter. Soil pH, saflufenacil rate, and seed depth also may influence the risk of crop injury from saflufenacil, but were less important than soil type.

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Chapter 1 - Kochia Control with Preemergence Herbicides in Soybeans

ABSTRACT

Field experiments were conducted near Cimarron in southwest Kansas in 2009, Hays in west-central Kansas in 2009 and 2010, and Norton in northwest Kansas in 2010, to evaluate the efficacy of selected pre-emergence (PRE) herbicides in combination with postemergence (POST) applied glyphosate for control of kochia in soybeans. Kochia control was evaluated 2, 4, 8, and 10 weeks after planting (WAP) at all sites except for Hays 2009, which was evaluated at 5 and 10 WAP. At Hays in 2009, all herbicides except for clomazone and imazaquin provided at least 95% kochia control 5 WAP. At 10 WAP, all treatments provided 100% kochia control. In 2010, most treatments provided at least 98% kochia control throughout the growing season. Clomazone provided 93% control at 4 WAP. At Cimarron, all herbicide treatments provided greater than 95% control of kochia except flumioxazin at 2 and 4 WAP. At 8 WAP, all treatments provided more than 90% kochia control, with sulfentrazone and sulfentrazone & cloransulam providing the highest control. Glyphosate controlled 95% of kochia at 8 WAP, but less than 65% of kochia at 10 WAP. At Norton, all treatments provided greater than 95% kochia control except imazaquin at 2 and 4 WAP. All treatments provided greater than 95% kochia control at both 8 and 10 WAP, with 99% or better control 8 WAP. Glyphosate applied alone did not always provide adequate season-long kochia control. PRE herbicide treatments can provide effective kochia control in soybeans and may be critical to help manage glyphosate-resistant kochia.

INTRODUCTION

Kochia (*Kochia scoparia* (L.) Schrad), also known as fireweed, is originally from eastern Europe and western Asia (Dodd and Moore 1993). Kochia is a C4 plant, which makes it extremely well adapted to hot, dry climates (Dodd and Moore 1993). It was originally brought to the United States in the early 1900's as an ornamental plant and has been utilized as a drought-resistant forage crop (Undersander et al. 1990; Phillips and Launchbaugh 1958). During the early part of the 20th century there were only a few scattered populations of kochia present in the northern Great Plains (Becker, 1978). However, the kochia population increased dramatically during the 1930's in Kansas (Gates 1941) and North Dakota (Stevens 1946). Since then it has spread and is now present in much of the midwestern and western United States (Menalled and Smith 2007) and is a troublesome and highly competitive weed in many cropping systems.

Because of kochia's ability to compete, it has been found to have a detrimental effect on the yield of many crops grown in the western USA. For example, season-long kochia competition reduced soybean yields by 30% (Forcella 1985). Kochia competition from 6 plants per meter of row reduced sunflower yield by 27% (Durgan et al. 1990). Uncontrolled triazine resistant kochia reduced grain sorghum yields as much as 85% (Wicks et al. 1994). For each kilogram per hectare of kochia biomass produced, corn grain yields decreased 0.33 kilograms per hectare (Wicks et al. 1993). Another study showed that when kochia was allowed to compete with sugarbeet for a full season, yield was reduced to 225 kilogram per hectare compared to 49,177 kilogram per hectare when kochia was removed 4 weeks after sugarbeet emergence (Weatherspoon and Schweizer 1969).

Kochia traditionally has been controlled using preemergence (PRE) and postemergence (POST) herbicides that belong to several different herbicide modes of action (MOA). PRE herbicides used to control kochia include acetolactate synthase (ALS)-inhibitors, microtubule-inhibitors, protoporphyrinogen oxidase (PPO)-inhibitors, seedling shoot inhibitors, hydroxyphenyl-pyruvate-dioxygenase (HPPD)-inhibitors, and photosynthetic (PSII)-inhibitor herbicides (Thompson et.al. 2011). POST herbicides used for kochia control include plant growth regulators, PPO-inhibitors, ALS-inhibitors, HPPD-inhibitors, and enolpyruvyl-shikimate-phosphate synthase (EPSPS)-inhibitors (Thompson et.al. 2011). In 1976, a kochia population in Kansas was confirmed resistant to a PSII-inhibitor. This was the first time a kochia population was confirmed to be resistant to an herbicide (Heap 2012). Since then kochia populations in 20 states in the USA, as well as in Canada and the Czech Republic have developed resistance to four different herbicide MOA, including growth regulators, PSII-inhibitors, ALS-inhibitors, and just recently glyphosate, which is an EPSPS-inhibitor (Waite 2008, Heap 2012).

The ability of kochia to develop herbicide resistance so quickly and easily when compared to other weeds is possibly a function of several factors including reproduction and dispersal characteristics, short seed life, and genetic diversity within the species (Burnside et. al. 1981; Eberlein and Fore 1984; Guttieri et. al. 1998; Mengistu and Messersmith 2002). A short seed life and limited seed dormancy results in a rapid turnover rate in the population and thus can result in quicker selection and genetic shifts in a population (Conard and Radosevich 1979).

In addition to herbicides, cultural practices and tillage also can be utilized to control kochia in soybean. Narrowing crop row spacing will allow soybeans to close canopy quicker than those that are spaced farther apart. This will shade the ground and suppress new flushes of kochia (Wax and Pendelton 1968). Tillage can help control kochia by killing emerged plants and

seed burial. Studies have shown that burying the seed of kochia 20 millimeters deep can severely reduce the ability of that seed to germinate and emerge (Schwinghamer and Van Acker 2008). Because of poor establishment after deep burial and lack of dormancy, cultivation remains a good option for kochia control in certain situations. However, the advent of herbicide resistant crops, especially those resistant to glyphosate, has caused a shift in cultural practices and has led many producers to adopt some form of conservation or no-till crop production system (Jordan et al. 1997; Moseley and Hagood 1990). This is especially the case in western Kansas where limited rainfall combined with high winds are prompting producers to switch to these practices to conserve moisture and topsoil. In these types of operations, there is little or no tillage, which means that producers rely almost exclusively on herbicides to control weed populations.

As of 2009, over 90% of the soybeans grown in the United States were glyphosate-resistant, along with around 70% of cotton and 60-65% of corn (Duke 2009). Because of the tendency for kochia to develop resistance to herbicides, and especially with the confirmation of kochia populations with resistance to glyphosate, alternative control methods must be considered. Since tillage is not a choice in conservation tillage systems, the use of PRE and POST herbicides with different MOA may help to curb the development of herbicide resistant kochia populations. The objective of this research was to evaluate the efficacy of selected PRE herbicides in combination with POST applied glyphosate for controlling kochia in soybeans.

MATERIALS AND METHODS

Field experiments were conducted near Cimarron in southwest Kansas in 2009, Hays in west-central Kansas in 2009 and 2010, and Norton in northwest Kansas in 2010, to evaluate the efficacy of selected herbicides for control of kochia in soybeans. The experimental site near Cimarron was located 8 miles north and 2 miles west of Cimarron in a producer's field that had previously been planted to corn and had a natural infestation of kochia. The field had Ness clay and Spearville silty clay loam soils and was conventionally tilled. The experimental site at Hays was located at the Kansas State University Agricultural Research Center. The crop in the field the previous year had been a failed wheat crop in 2009, and grain sorghum in 2010. Experiments both years were conducted on a Harney silt loam soil using conventional-tillage. The experimental site near Norton was located 9 miles west and 4 miles north of Norton in a producer's field that had previously been planted to corn and had a natural infestation of kochia. The field had a Holdrege silt loam soil and was conventionally-tilled. All sites were non-irrigated except for the Cimarron site, which was irrigated using a pivot irrigation system.

Glyphosate-resistant soybeans were planted using a standard row planter with 76 cm rows at Cimarron on May 2nd, 2009. At Hays, glyphosate-resistant soybeans were planted using a standard row planter with 76 cm rows on May 14 and June 2 in 2009 and 2010 respectively. At Norton, glyphosate-resistant soybeans were planted in 38 cm rows by the producer using a drill on June 4, 2010. Soybean plots were 2.3 m wide and 7.6 m long at all sites. Herbicides were applied with a CO_2 pressurized backpack sprayer equipped with TeeJet 8002VS nozzle tips calibrated to spray 140 L ha^{-1}.

At Hays, kochia seeds were sown across the plot area the day before planting each year. Other weeds present included punturevine (*Tribulus terrestris* L.), tumble pigweed (*Amaranthus albus* L.), and Palmer amaranth (*Amaranthus palmeri* L.). A natural kochia population was present at Cimarron and Norton, so no additional kochia seed was spread. Other weeds included velvetleaf (*Abutilon theophrasti* Medik.) and yellow foxtail (*Setaria glauca* (L.) Beauv) at Cimarron and large crabgrass (*Digitaria sanguinalis* (L.) Scop.), Russian thistle (*Salsola iberica* Sennen & Pau), and Palmer amaranth (*Amaranthus palmeri* L.) at Norton.

PRE treatments at Cimarron and Hays in 2009 included flumioxazin at 72 and 108 g ai/ha, sulfentrazone at 210 and 315 g ai/ha, clomazone at 841 g ai/ha, imazaquin at 138 g ai/ha, sulfentrazone & cloransulam at 263 & 34 g ai/ha, sulfentrazone & metribuzin at 203 & 305 g ai/ha, flumioxazin & chlorimuron at 85 & 30 g ai/ha, pendimethalin plus flumioxazin at 1597 plus 72 g ai/ha, pendimethalin plus sulfentrazone at 1597 plus 210 g ai/ha, clomazone plus sulfentrazone at 841 plus 210 g ai/ha, *S*-metolachlor & fomesafen plus metribuzin at 1216 & 266 plus 424 g ai/ha. PRE treatments were followed 4 weeks later by a POST application of glyphosate at a typical field use rate of 870 g ae/ha. In addition, glyphosate was applied POST at 870 and 1740 g/ha alone and a non-treated control plot was included for comparison. POST glyphosate treatments were applied when kochia was 15-20 cm tall and soybeans were in the V2-V3 growth stage. Dry ammonium sulfate was added to all glyphosate treatments at 3.8 kg/ha. PRE applications were made on May 3rd and May 18th, and POST applications were made on June 5th and June 19th at Cimarron and Hays respectively.

PRE treatments at Norton and Hays in 2010 remained the same as in 2009 except one treatment, flumioxazin & chlorimuron at 85 & 30 g ai/ha was omitted and six treatments, flumioxazin & chlorimuron at 63 & 22 and 105 & 37 g ai/ha, sulfentrazone & cloransulam at 196

& 25 g ai/ha, saflufenacil at 25 g ai/ha, saflufenacil & imazethapyr at 25 & 70 g ai/ha, and flumioxazin & pyroxasulfone at 71 & 90 g ai/ha were added. POST glyphosate treatments remained the same. Glyphosate was applied when kochia was 15-20 cm tall and soybeans were in the V2-V3 growth stage. PRE applications were made on June 8 and June 2, at Norton and Hays respectively, and POST applications were made on July 8th at both locations.

Visual control ratings on a scale of 0 to 100%, where 0 equals no control and 100% equals complete control were determined 2, 4, 8, and 10 weeks after planting (WAP) for kochia and general weed control for Cimarron, Hays 2010, and Norton, and 5 and 10 WAP for kochia control for Hays 2009. The experimental design was a randomized complete block with four replications. Untreated control treatments were omitted from statistical analyses of weed control data. All ratings were subjected to ANOVA using PROC GLM in SAS and means were separated using LSD at $P \leq 0.05$. All data were tested for homogeneity of variance, subjected to ANOVA, and sites are presented separately because of a significant site effect.

RESULTS AND DISCUSSION

At Cimarron in 2009, general weed control was 92% or higher and not significantly different among all PRE treatments 2 WAP (Table 1.1). General weed control with PRE treatments at 4 WAP varied, with clomazone providing the least control at 66%. Pendimethalin plus flumioxazin, flumioxazin at 108 g/ha, and flumioxazin at 72 g/ha provided 78, 79, and 81% control respectively. All other treatments provided greater than 91% control (Table 1.1). At 8 WAP, the 1X and 2X POST rates of glyphosate and clomazone followed by (fb) glyphosate provided the least control at 74, 74, and 81% control respectively. All other herbicide treatments provided greater than 88% control (Table 1.1). At 10 WAP, the 1X and 2X POST rates of

glyphosate, clomazone fb glyphosate, imazaquin fb glyphosate, and flumioxazin at 72 g/ha fb glyphosate provided the least control at 59, 61, 70, 79 and 81% respectively (Table 1.1).

At Hays in 2010, general weed control was 92% or higher and not significantly different among all PRE treatments 2 WAP (Table 1.2). At 4 WAP, clomazone provided the least control at 48%. Clomazone plus sulfentrazone and sulfentrazone at 315 g/ha controlled 63 and 73% of weeds respectively. All other treatments provided 83% or greater weed control (Table 1.2). At 8 WAP, all treatments provided greater than 86% control (Table 1.2). POST glyphosate treatments provided 90% or greater weed control at 8 WAP, but no more than 88% control 10 WAP (Table 1.2).

At Norton in 2010, general weed control 2 WAP was 98% or higher with all PRE treatments except imazaquin, which provided 85% control (Table 1.3). At 4 WAP, clomazone, saflufenacil, and sulfentrazone & cloransulam at 263 & 34 g/ha provided the least control at 74, 76, and 83% respectively. All other treatments provided 91% or greater control (Table 1.3). Following the postemergence glyphosate applications, all treatments provided 98% or greater general weed control at 8 and 10 WAP (Table 1.3).

Kochia control 2 WAP at Cimarron in 2009 was excellent, with all treatments providing 100% control, with the exception of flumioxazin at 72 g/ha which provided 93% control (Table 1.4). At 4 WAP, control of kochia with pendimethalin plus flumioxazin at 1597 plus 72 g/ha and flumioxazin at 72 g/ha dropped to 95, and 83% respectively. All other treatments provided at least 99% control (Table 1.4). At 8 WAP, all treatments provided 95% or better kochia control and were not different among treatments (Table 1.4). Control of kochia ranged from 50 to 100% control 10 WAP. Glyphosate at 870 g/ha or 1740 g/ha, and clomazone at 841 g/ha fb glyphosate, provided the least kochia control at 50, 63, and 64% respectively. All other treatments provided

93% or greater kochia control, including several treatments that provided complete control (Table1.4).

At Hays in 2009, kochia control 5 WAP was 100% with all treatments except imazaquin, clomazone, and flumioxazin at 72 g/ha which provided 90, 90, and 95% kochia control respectively (Table 1.5). All treatments provided complete kochia control by 10 WAP, following the POST glyphosate treatment (Table 1.5).

Control of kochia at Hays in 2010 was similar to that of 2009. Kochia control 2 WAP was 98% or higher and not significantly different among PRE treatments (Table 1.6). At 4 WAP, kochia control was 98% or higher with all treatments except clomazone, which provided 93% control (Table 1.6). Kochia control 8 and 10 WAP was 100% for all treatments (Table 1.6).

At Norton in 2010, all treatments except imazaquin provided 94% or greater kochia control at 2 and 4 WAP (Table 1.7). At 8 WAP, kochia control was 99% or higher and not significantly different among all treatments. All treatments provided 96% or greater kochia control 10 WAP (Table 1.7).

Season-long kochia control was 96% or greater across all PRE fb glyphosate and glyphosate alone treatments at Hays both years and at Norton in 2010. This could be due to a couple of factors. First, timely rainfall occurred following the PRE treatments in both years at Hays and at Norton. The adequate soil moisture would keep the residual herbicides active in the soil and that may have impacted overall PRE herbicide performance (Stickler 1969). Another factor was the timing of planting and that both sites were conventionally tilled just prior to planting. Kochia has the ability to germinate early in the season (Schwinghamer and Van Acker 2008). Delay of planting could allow a majority of kochia to emerge and be controlled by the tillage operation prior to planting. This could significantly decrease the kochia populations after

planting. At Cimarron, PRE herbicide control of kochia was generally good to excellent throughout the growing season, with only three treatments providing less than 95% control. The lower level of late season kochia control at Cimarron with some PRE treatments and glyphosate compared to the other sites may have been due to the earlier planting date and irrigation at that site. An earlier planting date likely would result in more kochia germination and emergence after planting. Irrigation would help activate the PRE herbicides for good kochia control early in the season (Stickler 1969). However, regular watering would also facilitate quicker degradation of the PRE herbicides and stimulate more flushes of kochia later in the season that would be more likely to escape control from both the PRE treatments and the POST glyphosate treatments. While glyphosate controlled emerged kochia, it has no residual activity, meaning that any kochia which emerged after glyphosate application would not have been controlled.

PRE and glyphosate treatments provided good season-long general weed control at Hays and Norton in 2010 and early to mid-season general weed control at Cimarron in 2009. This data suggests that a sequential herbicide program with a PRE herbicide treatment followed by POST glyphosate will probably provide the most consistent kochia control and help minimize the risk of developing herbicide resistant kochia.

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Table 1.1. General weed control 2, 4, and 10 weeks after planting (WAP) at Cimarron in 2009.

Herbicide	Rate g/ha	Application Timing[1]	2 WAP[2]	4 WAP[2]	8 WAP	10 WAP
			----------% Control----------			
flumioxazin	72	PRE	92	81	88	81
flumioxazin	108	PRE	96	79	90	85
sulfentrazone	210	PRE	98	94	95	88
sulfentrazone	315	PRE	100	99	98	95
clomazone	841	PRE	98	66	81	70
sulfentrazone & cloransulam	263 & 34	PRE	100	99	98	96
sulfentrazone & metribuzin	203 & 305	PRE	99	94	96	94
flumioxazin & chlorimuron	85 & 30	PRE	93	96	98	89
pendimethalin + flumioxazin	1597 + 72	PRE	95	78	89	85
pendimethalin + sulfentrazone	1597 + 210	PRE	100	91	95	95
clomazone + sulfentrazone	841 + 210	PRE	95	84	94	91
S-metolachlor & fomesafen + metribuzin	1216 & 266 + 424	PRE	98	93	98	95
imazaquin	138	PRE	96	95	94	79
glyphosate	870	POST			74	59
glyphosate	1740	POST			74	61
LSD (0.05)			NS	17	6	10

[1]All preemergence (PRE) herbicide treatments were followed by postemergence (POST) applied glyphosate at 870 g /ha 4 WAP.

[2]Ratings for PRE herbicides only.

Table 1.2. General weed control 2, 4, 8, and 10 weeks after planting (WAP) at Hays in 2010.

Herbicide	Rate g/ha	Application Timing[1]	2 WAP[2]	4 WAP[2]	8 WAP	10 WAP
			----------% Control----------			
flumioxazin	72	PRE	98	88	94	91
flumioxazin	108	PRE	100	86	86	85
sulfentrazone	210	PRE	100	89	95	93
sulfentrazone	315	PRE	100	73	88	88
clomazone	841	PRE	100	48	93	85
sulfentrazone & cloransulam	197 & 25	PRE	100	95	96	98
sulfentrazone & cloransulam	263 & 34	PRE	98	85	96	98
sulfentrazone & metribuzin	203 & 305	PRE	100	98	98	98
flumioxazin & chlorimuron	64 & 22	PRE	100	98	96	96
flumioxazin & chlorimuron	106 & 36	PRE	100	99	98	98
pendimethalin + flumioxazin	1597 + 72	PRE	100	93	99	95
pendimethalin + sulfentrazone	1597 + 210	PRE	100	98	98	98
clomazone + sulfentrazone	841 + 210	PRE	100	63	97	92
S-metolachlor & fomesafen + metribuzin	1216 & 266 + 424	PRE	100	99	99	95
saflufenacil	25	PRE	100	83	91	88
saflufenacil & imazethapyr	25 & 71	PRE	100	96	99	96
flumioxazin & pyroxasulfone	71 & 90	PRE	100	98	100	98
imazaquin	138	PRE	100	94	100	100
glyphosate	870	POST			99	88
glyphosate	1740	POST			90	76
LSD (0.05)			NS	22	10	10

[1]All preemergence (PRE) herbicide treatments were followed by postemergence (POST) applied glyphosate at 870 g /ha 4 WAP.

[2]Ratings for PRE herbicides only.

Table 1.3. General weed control 2, 4, 8, and 10 weeks after planting (WAP) at Norton in 2010.

Herbicide	Rate g/ha	Application Timing[1]	2 WAP[2]	4 WAP[2]	8 WAP	10 WAP
			----------% Control----------			
flumioxazin	72	PRE	98	94	99	100
flumioxazin	108	PRE	100	98	100	100
sulfentrazone	210	PRE	100	96	100	100
sulfentrazone	315	PRE	99	97	99	100
clomazone	841	PRE	100	74	99	99
sulfentrazone & cloransulam	197 & 25	PRE	99	100	100	100
sulfentrazone & cloransulam	263 & 34	PRE	100	83	100	100
sulfentrazone & metribuzin	203 & 305	PRE	100	99	99	100
flumioxazin & chlorimuron	64 & 22	PRE	100	98	100	100
flumioxazin & chlorimuron	106 & 36	PRE	100	91	100	100
pendimethalin + flumioxazin	1597 + 72	PRE	100	94	100	99
pendimethalin + sulfentrazone	1597 + 210	PRE	100	99	100	99
clomazone + sulfentrazone	841 + 210	PRE	98	100	100	100
S-metolachlor & fomesafen + metribuzin	1216 & 266 + 424	PRE	99	100	99	99
saflufenacil	25	PRE	100	76	100	100
saflufenacil & imazethapyr	25 & 71	PRE	100	100	100	100
flumioxazin & pyroxasulfone	71 & 90	PRE	100	99	100	100
imazaquin	138	PRE	85	93	100	98
glyphosate	870	POST			100	100
glyphosate	1740	POST			100	99
LSD (0.05)			9	19	NS	NS

[1]All preemergence (PRE) herbicide treatments were followed by postemergence (POST) applied glyphosate at 870 g /ha 4 WAP.

[2]Ratings for PRE herbicides only.

Table 1.4. Kochia control 2, 4, 8, and 10 weeks after planting (WAP) at Cimarron in 2009.

Herbicide	Rate g/ha	Application Timing[1]	2 WAP[2]	4 WAP[2]	8 WAP	10 WAP
			----------% Control----------			
flumioxazin	72	PRE	93	83	98	80
flumioxazin	108	PRE	100	99	98	93
sulfentrazone	210	PRE	100	100	100	99
sulfentrazone	315	PRE	100	100	100	100
clomazone	841	PRE	100	100	99	63
sulfentrazone & cloransulam	263 & 34	PRE	100	100	100	100
sulfentrazone & metribuzin	203 & 305	PRE	100	100	100	100
flumioxazin & chlorimuron	85 & 30	PRE	100	100	100	100
pendimethalin + flumioxazin	1597 + 72	PRE	100	95	100	93
pendimethalin + sulfentrazone	1597 + 210	PRE	100	100	100	100
clomazone + sulfentrazone	841 + 210	PRE	100	100	100	100
S-metolachlor & fomesafen + metribuzin	1216 & 266 + 424	PRE	100	100	99	99
imazaquin	138	PRE	100	100	100	98
glyphosate	870	POST			95	50
glyphosate	1740	POST			95	64
LSD (0.05)			6	10	NS	16

[1]All preemergence (PRE) herbicide treatments were followed by postemergence (POST) applied glyphosate at 870 g /ha 4 WAP.

[2]Ratings for PRE herbicides only.

Table 1.5. Kochia control 5 and 10 weeks after planting (WAP) at Hays in 2009.

Herbicide	Rate g/ha	Application Timing[1]	5 WAP[2]	10 WAP
			-------% Control-------	
flumioxazin	72	PRE	95	100
flumioxazin	108	PRE	100	100
sulfentrazone	210	PRE	100	100
sulfentrazone	315	PRE	100	100
clomazone	841	PRE	90	100
sulfentrazone & cloransulam	263 & 34	PRE	100	100
sulfentrazone & metribuzin	203 & 305	PRE	100	100
flumioxazin & chlorimuron	85 & 30	PRE	100	100
pendimethalin + flumioxazin	1597 + 72	PRE	100	100
pendimethalin + sulfentrazone	1597 + 210	PRE	100	100
clomazone + sulfentrazone	841 + 210	PRE	100	100
S-metolachlor & fomesafen + metribuzin	1216 & 266 + 424	PRE	100	100
imazaquin	138	PRE	90	100
glyphosate	870	POST		100
glyphosate	1740	POST		100
LSD (0.05)			7	NS

[1]All preemergence (PRE) herbicide treatments were followed by postemergence (POST) applied glyphosate at 870 g /ha 5 WAP.

[2]Ratings for PRE herbicides only.

Table 1.6. Kochia control 2, 4, 8, and 10 weeks after planting (WAP) at Hays in 2010.

Herbicide	Rate g/ha	Application Timing[1]	2 WAP[2]	4 WAP[2]	8 WAP	10 WAP
			----------% Control----------			
flumioxazin	72	PRE	98	100	100	100
flumioxazin	108	PRE	100	100	100	100
sulfentrazone	210	PRE	100	100	100	100
sulfentrazone	315	PRE	100	100	100	100
clomazone	841	PRE	100	93	100	100
sulfentrazone & cloransulam	197 & 25	PRE	100	100	100	100
sulfentrazone & cloransulam	263 & 34	PRE	98	98	100	100
sulfentrazone & metribuzin	203 & 305	PRE	100	100	100	100
flumioxazin & chlorimuron	64 & 22	PRE	100	100	100	100
flumioxazin & chlorimuron	106 & 36	PRE	100	100	100	100
pendimethalin + flumioxazin	1597 + 72	PRE	100	100	100	100
pendimethalin + sulfentrazone	1597 + 210	PRE	100	100	100	100
clomazone + sulfentrazone	841 + 210	PRE	100	98	100	100
S-metolachlor & fomesafen + metribuzin	1216 & 266 + 424	PRE	100	100	100	100
saflufenacil	25	PRE	100	100	100	100
saflufenacil & imazethapyr	25 & 71	PRE	100	100	100	100
flumioxazin & pyroxasulfone	71 & 90	PRE	100	100	100	100
imazaquin	138	PRE	100	100	100	100
glyphosate	870	POST			100	100
glyphosate	1740	POST			100	100
LSD (0.05)			NS	5	NS	NS

[1]All preemergence (PRE) herbicide treatments were followed by postemergence (POST) applied glyphosate at 870 g /ha 4 WAP.

[2]Ratings for PRE herbicides only.

Table 1.7. Kochia control 2, 4, 8, and 10 weeks after planting (WAP) at Norton in 2010.

Herbicide	Rate g/ha	Application Timing[1]	2 WAP[2]	4 WAP[2]	8 WAP	10 WAP
			----------% Control----------			
flumioxazin	72	PRE	96	94	99	100
flumioxazin	108	PRE	100	99	100	100
sulfentrazone	210	PRE	100	100	100	100
sulfentrazone	315	PRE	98	98	99	100
clomazone	841	PRE	100	100	100	100
sulfentrazone & cloransulam	197 & 25	PRE	99	100	100	100
sulfentrazone & cloransulam	263 & 34	PRE	100	100	100	100
sulfentrazone & metribuzin	203 & 305	PRE	100	99	99	100
flumioxazin & chlorimuron	64 & 22	PRE	100	100	100	100
flumioxazin & chlorimuron	106 & 36	PRE	100	100	100	100
pendimethalin + flumioxazin	1597 + 72	PRE	100	100	100	100
pendimethalin + sulfentrazone	1597 + 210	PRE	100	95	100	100
clomazone + sulfentrazone	841 + 210	PRE	98	100	100	100
S-metolachlor & fomesafen + metribuzin	1216 & 266 + 424	PRE	99	100	99	99
saflufenacil	25	PRE	100	99	100	100
saflufenacil & imazethapyr	25 & 71	PRE	100	100	100	100
flumioxazin & pyroxasulfone	71 & 90	PRE	100	100	100	100
imazaquin	138	PRE	85	91	100	96
glyphosate	870	POST			100	100
glyphosate	1740	POST			100	100
LSD (0.05)			10	7	NS	3

[1]All preemergence (PRE) herbicide treatments were followed by postemergence (POST) applied glyphosate at 870 g /ha 4 WAP.

[2]Ratings for PRE herbicides only.

Chapter 2 - Dose Response of Three Kochia Populations to Glyphosate

ABSTRACT

Greenhouse experiments were conducted to determine the response of three kochia populations to glyphosate applied at 10 different rates. Kochia seeds were collected from a field near Norton, KS, in 2010 for response comparisons to kochia populations from Syracuse and Ingalls, KS previously characterized as highly susceptible and moderately resistant to glyphosate herbicide. Glyphosate rates included 0, 0.0625, 0.125, 0.25, 0.50, 1, 1.5, 2, 4 and 6 times a typical field use rate of 870 g ae/ha. In general, as the rate of glyphosate increased, visible kochia control increased with all three populations. Symptoms included yellowing of leaves, general chlorosis, stunting, and necrosis of kochia plants. These symptoms occurred at much lower rates for the Syracuse population compared to the Norton or Ingalls populations, but symptoms were observed across all populations at the higher rates. Kochia control below the field use rate of glyphosate was not greater than 10% for either the Ingalls or Norton populations throughout the experiment. However, the Syracuse population was controlled up to 37 and 59% at the 0.25 and 0.5X rates 21 days after treatment (DAT). At the field use rate, the Syracuse kochia population was controlled 94% 21 DAT, while the Ingalls and Norton populations were controlled 26 and 41% respectively. Control of the Ingalls and Norton kochia populations 21 DAT didn't exceed 90% until applied at the 4 and 6X rates of glyphosate. All glyphosate rates at and above the field use rate controlled the Syracuse population over 90% 21 DAT, with complete mortality occurring at the 2, 4, and 6X rates. Nonlinear regression analysis for each population

indicated the glyphosate rate required to cause 50% visible control (GR50) was 1.6, 1.1, and 0.31 times the field use rate for the Ingalls, Norton, and Syracuse kochia populations respectively, indicating the Syracuse kochia population was considerably more susceptible than either of the other two populations. The results of this research give an indication of the great variability in kochia susceptibility to glyphosate across different kochia populations. Because of the increased use of glyphosate, integrated weed management systems that utilize different herbicide modes of action and methods of weed control will be required to effectively manage and reduce the spread of glyphosate-resistant kochia.

INTRODUCTION

Kochia (*Kochia scoparia* (L.) Schrad), also known as fireweed, is originally from eastern Europe and western Asia (Dodd and Moore 1993). It was originally brought to the United States in the early 1900's as an ornamental plant and has been utilized as a drought-resistant forage crop (Undersander et al. 1990; Phillips and Launchbaugh 1958). During the early part of the 20th century there were only a few scattered populations of kochia present in the northern Great Plains (Becker, 1978). However, the kochia population increased dramatically during 1930's in Kansas (Gates 1941) and North Dakota (Stevens 1946). Since then it has spread and is now present in much of the midwestern and western United States and is a troublesome weed in many cropping systems (Menalled and Smith 2007).

Kochia is a C4 plant (Dodd and Moore 1993), which makes it extremely well adapted to the hot, dry climates of the western USA. It is a highly competitive and difficult to control plant in many cropping systems throughout this region for a variety of reasons, including unique germination abilities, a highly developed rooting system, an ability to alter growth habits, and

prolific seed production and dispersal (Phillips and Launchbaugh 1958; Mulugeta et al 1994; Stevens 1932).

Kochia traditionally has been controlled using preemergence (PRE) and postemergence (POST) herbicides that belong to several different herbicide mode of actions (MOA's). PRE herbicides used to control kochia include acetolactate synthase (ALS)-inhibitors, microtubule-inhibitors, protoporphyrinogen oxidase (PPO)-inhibitors, seedling shoot inhibitors, 4-hydroxyphenyl-pyruvate-dioxygenase (HPPD)-inhibitors, and photosynthetic (PSII)-inhibitor herbicides (Thompson et.al. 2011). POST herbicides used for kochia control include plant growth regulators, PPO-inhibitors, ALS-inhibitors, HPPD-inhibitors, and enolpyruvyl-shikimate-phosphate synthase (EPSPS)-inhibitors (Thompson et.al. 2011). In 1976, a kochia population in Kansas was confirmed resistant to a PSII-inhibitor herbicide. This was the first time a kochia population had been confirmed to be resistant to an herbicide (Heap 2012). Since then kochia populations in 20 states in the USA, as well as Canada and the Czech Republic have developed resistance to four different herbicide MOA's, including growth regulators, PSII-inhibitors, ALS-inhibitors, and just recently glyphosate, which is an EPSPS-inhibitor (Waite 2008, Heap 2012).

The ability of kochia to develop herbicide resistance so quickly and easily when compared to other weeds is possibly a function of several factors including reproduction and dispersal characteristics, short seed life, and genetic diversity within the species (Burnside et. al. 1981; Eberlein and Fore 1984; Guttieri et. al. 1998; Mengistu and Messersmith 2002). A short seed life and limited seed dormancy results in a rapid turnover rate in the population and thus can result in quicker selection and genetic shifts in a population (Conard and Radosevich 1979).

In the central and western half of the United States, limited soil moisture and soil erosion are major factors restricting agricultural production. In an effort to conserve moisture and top

soil, many producers have switched to systems that reduce or eliminate tillage operations (Halvorson et al. 2001; Norwood 2001; Smika 1990). In these types of systems, producers rely almost exclusively on herbicides to control weeds. The traditional burndown treatment at planting time includes a burndown herbicide to kill emerged weeds and to prevent other weeds from emerging (Krausz et al. 2000). The advent of herbicide resistant crops has also helped to increase the number of hectares in no-till crop production. The most popular herbicide resistant crop technology has been glyphosate-resistant crops. As of 2009, over 90% of the soybeans, 70% of cotton, and 60 to 65% of corn grown in the United States were glyphosate-resistant (Duke 2009).

Glyphosate is a systemic herbicide used in no-till cropping systems as a burndown, and as an in-crop, nonselective option for weed control in glyphosate-resistant crops. The relatively low cost and broad-spectrum weed control provided by glyphosate are the main reasons for its widespread adoption. Because glyphosate is a non-residual herbicide, it also provides producers with good crop rotation flexibility (Hoss et al. 2003).

Currently there are 23 known weed species with evolved resistance to glyphosate (Heap 2012). Levels of resistance to glyphosate are generally lower compared to other herbicide mode of actions (Feng et al. 2004, Gressel 2002, Owen and Powles 2010). For example, rigid ryegrass resistance to glyphosate is several fold more resistant to glyphosate than a susceptible biotype, whereas the resistance to ALS-inhibiting herbicides is hundreds of folds greater than the susceptible biotypes (Powles et al. 1998). Glyphosate resistance in kochia has been difficult to confirm because of the low level of resistance and interaction with environmental conditions. The objective of this study was to quantify the level of glyphosate resistance of three kochia populations collected from three field sites in western Kansas.

MATERIALS AND METHODS

Seeds were collected near Norton, Kansas in the fall of 2010 from a field with a suspected population of glyphosate-resistant kochia to determine the level of glyphosate resistance compared to kochia populations from Syracuse and Ingalls, Kansas. The Syracuse population was collected from a site where glyphosate has been used infrequently. The Ingalls population was collected from a field with a history of repeated glyphosate use and was suspected to be resistant to glyphosate. Waite (2008) found that the Syracuse population was susceptible to glyphosate while the Ingalls population was moderately resistant.

Kochia seeds from the Syracuse, Ingalls, and Norton sites were planted in the greenhouse on December 15^{th,} 2010, and February 2^{nd,} 2011, in 50 x 35 x 10 cm flats filled with 11 kg of a soil mix. The soil mixture was a 1:1 by volume blend of sand and Morrill loam (fine-loamy, mesic Typic Arguidolls). The soil had 1.0% organic matter and a pH of 7.5. Containers measuring 0.9 L were filled with the same soil mixture and single kochia seedlings were transplanted from the flats on February 9th and April 5th 2011 when kochia plants were 8 to10 cm tall. Plants were grown under greenhouse conditions at 32/22 $\pm$ 2 C day/night temperatures and 16/8 h day/night periods. The supplemental photosynthetic photon flux was 80 µmol/m^2/s. Plants were fertilized weekly with a solution containing 0.40 mg/L nitrogen, 0.34 mg/L phosphorus, and 0.33 mg/L potassium.

This experiment was conducted twice, with the first run of kochia biotypes treated with glyphosate on February 18th and the second run treated with glyphosate on April 13^{th,} 2011, when the plants were 15 to 20 cm in height. Glyphosate was applied at 0, 0.0625, 0.125, 0.25, 0.50, 1, 1.5, 2, 4 and 6 times (X) a typical field use rate of 870 g ae/ha. The glyphosate source for all experiments was Roundup WeatherMax. All treatments included 0.25% (v/v) non-ionic

surfactant and 2.0% (w/v) dry ammonium sulfate. Treatments were applied with a bench-type sprayer calibrated to deliver 187 L/ha at 138 kPa. Visible control symptoms were monitored daily and control ratings were determined at 7, 14, and 21 days after treatment (DAT) on a scale of 0 to 100%, where 0 equals no control and 100% equals mortality. A nonlinear regression analysis was used to determine the glyphosate rate required to cause 50% visible control (GR_{50}) to the kochia populations (Seefeldt et al. 1995). The experiment had a randomized complete block design with six replications. All data were tested for homogeneity of variance, subjected to ANOVA, and pooled across runs because of an insignificant run effect. Means were separated by Fischer's Protected LSD at <0.05.

RESULTS AND DISCUSSION

In general, as the rate of glyphosate increased, kochia control increased with all three populations. Control symptoms included yellowing of leaves, general chlorosis, stunting, and necrosis of kochia plants. These symptoms occurred at much lower rates for the Syracuse population compared to the Norton or Ingalls populations, but symptoms were observed across all populations at the higher rates. Symptoms started to appear 5 DAT and slowly progressed throughout the study.

The Ingalls population generally showed the greatest tolerance to glyphosate throughout the duration of the study. At 7 DAT, only the 4X and 6X rates of glyphosate provided more than 26% control, while the field use rate only provided 24% control (Table 2.1). At 14 DAT, the 2X, 4X and 6X rates provided 44, 89, and 82% control respectively, but none of the other treatments controlled the kochia greater than 22% (Table 2.1). At 21 DAT, the field use rate of glyphosate provided 26% control, while the 1.5, 2, 4, and 6X rates gave 36, 69, 98 and 97% control of the kochia. Kochia control from glyphosate rates less than 1X rate never exceeded 8% throughout

the duration of the experiment.

The Norton kochia population was generally more susceptible to glyphosate than the Ingalls population, but less susceptible than the Syracuse population. At 7 DAT, the 2, 4, and 6X rates of glyphosate controlled Norton kochia 59, 57, and 73% while the 1X rate provided 27% control. All other rates provided no more than 10% control (Table 2.1). At 14 DAT, kochia control from the 1X rate increased to 40%, while the 2, 4, and 6X rates had 72, 81, and 89% control, respectively (Table 2.1). By 21 DAT the 4 and 6X rates had provided at least 90% control while control with the 1X, 1.5, and 2X rates generally was similar to the 14 DAT (Table 2.1). Throughout the study, glyphosate rates below the 1X rate provided no more than 10% control of the Norton kochia.

The Syracuse kochia population generally showed the greatest susceptibility to glyphosate compared to the Ingalls and Norton populations. At 7 DAT, all glyphosate rates above 0.25X provided 30% or greater control on Syracuse kochia, while control for the 0.0625, 0.125, and 0.25X rates was 3, 5, and 15%, respectively. The Syracuse kochia was controlled 70% by the field use rate of glyphosate at 7 DAT. At 14 and 21 DAT, only the 0.0625X and 0.125X rates of glyphosate provided less than 32% kochia control (Table 2.1). At 21 DAT, the field use rate of glyphosate provided 94% control of the Syracuse kochia population, while plant death was achieved by glyphosate rates of 2, 4, and 6X (Table 2.1). Throughout the study, glyphosate rates below the use rate provided more control of the Syracuse kochia than either the Ingalls or Norton kochia populations.

The GR50 values for all three populations were calculated for 21 DAT. The Ingalls kochia population was the most resistant, needing a glyphosate rate of 1.6 times the field use rate to achieve 50% control, followed by the Norton population which required a glyphosate rate of

1.1X (Figure 2.1). The Syracuse kochia population was considerably more susceptible than either of the other two populations, needing a glyphosate rate of only 0.31X to achieve 50% control (Figure 2.1). Farmers and crop advisers generally consider 90% control to be the threshold level of acceptable weed control. To achieve this result at 21 DAT, glyphosate has to be applied at a 4X rate for both the Ingalls and Norton populations, while the standard use rate provided 90% control of the Syracuse kochia (Table 2.1).

The results of this research give an indication of the great variability in kochia susceptibility to glyphosate across and within kochia populations. Kochia resistance to glyphosate has developed at multiple locations in western Kansas, as confirmed by Waite (2008). Because of the reproductive and dispersal characteristics of kochia, glyphosate resistance is likely to spread rapidly and become a major problem for western Kansas farmers, especially for those farmers that practice no-till or in areas that have relied heavily on glyphosate for weed control. Integrated weed management systems that utilize different herbicide modes of action and methods of weed control will be required to effectively manage and reduce the spread of glyphosate-resistant kochia, which threatens the viability of no-till crop production systems.

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Figure 2.1. Kochia Population Dose Response Curves and GR50's 21 DAT.

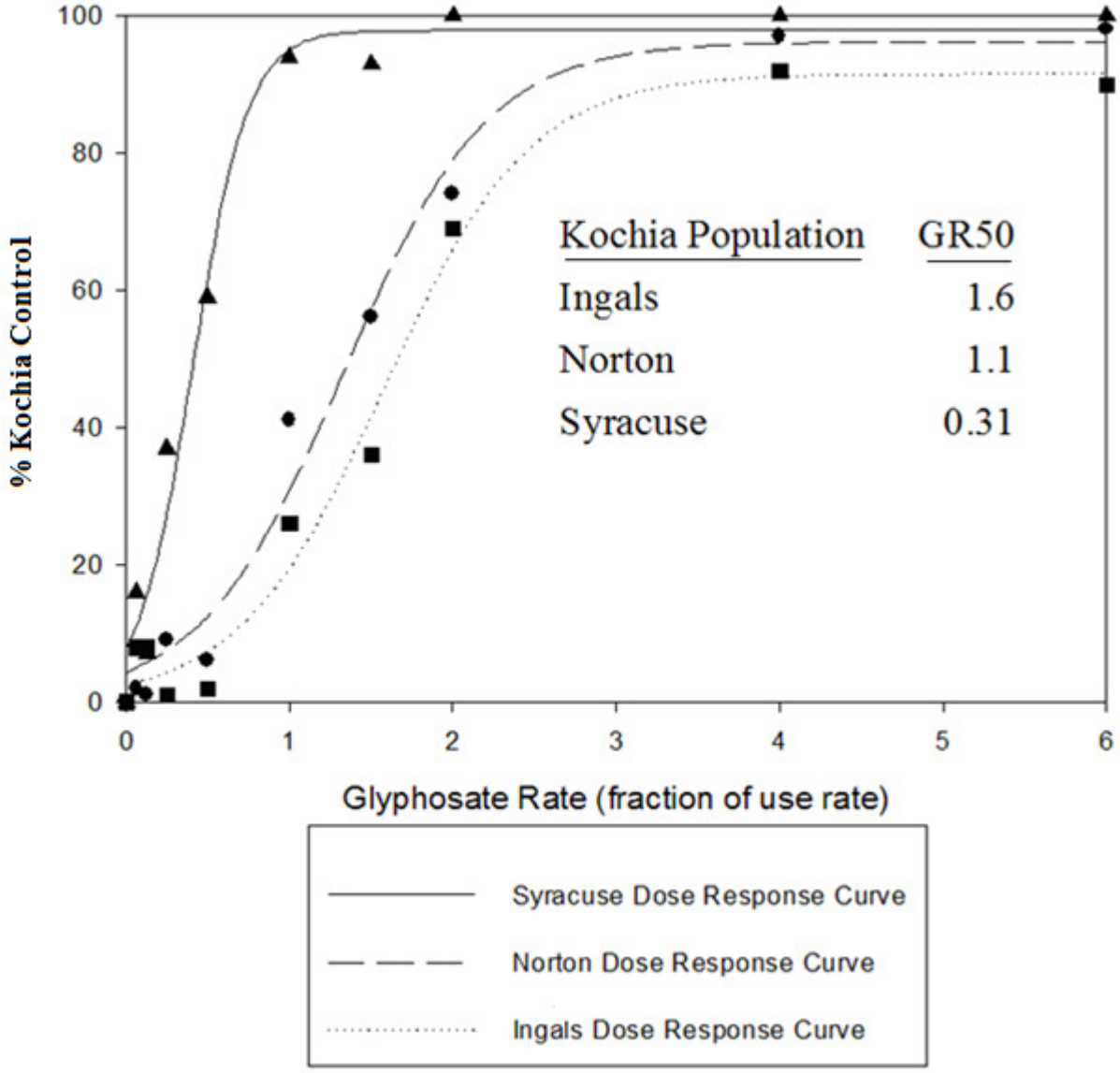

Table 2.1. Visible kochia control 7, 14, and 21 days after treatment (DAT) with glyphosate on three kochia populations.

Glyphosate	Ingalls			Norton			Syracuse		
Rate[1]	7 DAT	14 DAT	21 DAT	7 DAT	14 DAT	21 DAT	7 DAT	14 DAT	21 DAT
	Kochia control, %								
0	0	0	0	0	0	0	0	0	0
0.0625	0	2	8	0	0	2	3	11	16
0.125	1	4	8	0	0	1	5	6	7
0.25	0	0	1	1	1	9	15	32	37
0.5	4	1	2	10	7	6	30	55	59
1	24	22	26	27	40	41	70	88	94
1.5	14	21	36	27	46	56	76	92	93
2	26	44	69	59	72	74	86	99	100
4	57	89	98	57	81	92	84	99	100
6	61	82	97	73	89	90	94	98	100
LSD (0.05)	24	30	33	41	28	29	44	49	52

[1]Rates are the proportion of a glyphosate field use rate of 870 g ae/ha.

Chapter 3 - Corn, Soybean, and Grain Sorghum Response to Saflufenacil

ABSTRACT

Greenhouse experiments were conducted in 2011 to quantify the effects of herbicide rate, planting depth, soil pH, and soil type on corn, soybean, and grain sorghum tolerance to saflufenacil. Two soils, a Reading silt loam soil (Reading) consisting of 2, 74, and 24% sand, silt, and clay, and a Farnum fine sandy loam soil (Farnum) consisting of 82, 12, and 6% sand, silt, and clay. Soil pH was adjusted so that the Reading and Farnum soils had a low pH of 5.7 and 4.9 and a high pH of 7.5 and 8.4 respectively. Five seeds of each crop were planted into flats at evenly spaced intervals at two depths of 2 and 4 cm. Herbicide treatments included saflufenacil at rates of 0, 50, 100, and 150 g ai/ha. Corn injury appeared to be most significantly affected by soil type, with corn planted in the Reading soil being less injured than corn planted in the Farnum soil. In general, corn injury increased with increasing saflufenacil rate on the Farnum soil. The effect of soil pH and seeding depth on corn injury from saflufenacil was inconclusive. Saflufenacil injury to soybeans was variable, but generally was greater in the Farnum than the Reading soil, increased with increasing saflufenacil rate, and was greater at high pH than at low pH. Grain sorghum injury appeared to be influenced the most by soil type, with less injury occurring in the Reading soil than the Farnum soil. Grain sorghum injury was generally greater at the high pH compared to the low pH. This research suggests that several factors can influence the potential for crop injury from saflufenacil on corn, soybean, and grain sorghum, but soil characteristics, especially texture, appear to be the most important factor.

INTRODUCTION

Saflufenacil (N' –[2-chloro-4-fluoro-5-(3-methyl-2,6-dioxo-4-(trifluoromethyl)-3,6-dihydro-1(2H)pyrimidinyl) benzoyl]-N-isopropyl-N-methylsulfimide) is a pyrimidinedione protoporphyrinogen IX oxidase (PPO)–inhibiting herbicide developed by the BASF Corporation (Grossman et. al. 2010). It can provide both foliar and residual control of broadleaf weeds such as kochia (*Kochia scoparia* (L.) Schrad), pigweeds (*Amaranthus* spp. L.), Russian thistle (*Salsola iberica* Sennen & Pau), common cocklebur (*Xanthium strumarium* L.), horseweed (*Conyza canadensis* (L.) Cronq.), common lambsquarters (*Chenopodium album* L.), morningglory (*Ipomoea* spp. L.), common sunflower (*Helianthus annuus* L.), velvetleaf (*Abutilon theophrasti* Medik.), and common ragweed (*Ambrosia artemisiifolia* L.) as a fallow, preplant (PP), or premergence (PRE) treatment in various crops (Thompson et al. 2011). Saflufenacil is translocated primarily in the xylem of plants and can be absorbed by roots, shoots, and leaves, offering rapid burndown as well as soil residual activity (Liebl et al. 2008; Anonymous 2008). Saflufenacil can be used for selective weed control in a variety of crops, including corn, soybean, sorghum, cotton, small grains, and tree crops (Anonymous 2008).

Herbicide availability for uptake by plants from the soil depends on the interaction between the herbicide molecule and the various properties of soil (Bailey and White 1970; Harper 1994; Peter and Weber 1985). Organic matter (OM), clay content, soil pH, water holding capacity, and cation exchange capacity (CEC) are soil factors that can affect herbicide activity (Blumhorst et al. 1990; Corbin et al.1971; Harrison et al. 1976; Weber et al. 1993).

The sum of positive charges of adsorbed cations that a soil can adsorb at a specific pH is its CEC (Foth 1990). Soil CEC primarily is dependent on soil pH, clay content, and organic

matter. The higher the CEC, the greater the number of positively charged ions that can be adsorbed and removed from the soil solution (Kerr et al. 2004). Therefore, CEC can often be inversely correlated with herbicide bioactivity (Kerr et al. 2004). Herbicide bioactivity has also been inversely correlated with OM and soil clay content (Blumhorst et al. 1990; Harper 1994; Peter and Weber 1985).

Saflufenacil is an ionic, moderately acidic, highly aqueous soluble herbicide with a pKa value of 4.41 (Anonymous 2008). Soil bioactivity of other similar acidic herbicides such as 2,4-D, oryzalin, chlorsulfuron, metsulfuron, and sulfentrazone are directly related to soil pH and OM (Anderson and Barrett 1985; Grey et al. 1997; Kerr et al., 2004). Acidic herbicides are repelled by clays under neutral conditions but sorbed through physical bonding mechanisms under acidic conditions when the compounds are in the molecular form (Bailey and White 1970; Weber et al. 1993). For this reason, increasing soil pH causes more acidic herbicide anions to remain in soil solution and be available for uptake by plants. Even though saflufenacil may be affected by soil pH, its pKa value is below 5, meaning it will primarily be in the anionic form in most agricultural soils resulting in small pH effect on soil bioactivity (Hixon 2008).

Studies have been conducted over the past several years to determine the tolerance of several crops to saflufenacil including corn, soybean, and grain sorghum (Soltani et al. 2009, Soltani et al. 2010, Brown et al. 2010).

Soltani et al. (2009) conducted field trials in two Ontario locations to evaluate corn tolerance to saflufenacil at doses of 50, 100 and 200 g/ha applied PRE. Visual corn injury resulting from preemergence saflufenacil treatments caused little or no crop injury, and the author speculated that preemergence applications are safe up to 200 g/ha.

Soltani et al. (2010) conducted field trials at two Ontario locations in 2006 and at Exeter Ontario in 2007 to determine the tolerance of soybean and six other leguminous crops to saflufenacil doses of 100 and 200 g/ha applied PRE. Regardless of rate, saflufenacil injured soybean 25 and 13% one and two weeks after emergence (WAE). Injury decreased with time and at 4WAE soybeans only showed 6 and 22% injury at 100 and 200 g/ha respectively. This led the authors to conclude that PRE applications of saflufenacil in soybeans were safe up to the 100 g/ha rate.

Field trials were conducted by Brown et al. (2010) in 2008 and 2009 to determine the tolerance of grain sorghum to PRE applied saflufenacil at rates of 40, 90 and 180 g/ha at Lubbock, 50, 100, and 200 g/ha at Halfway, and 30, 60, and 100 g/ha at Lamesa, Texas. In general, sorghum injury increased with increasing saflufenacil rates and higher than average rainfall/irrigation after planting in 2009 seemed to increase saflufenacil injury.

Soil properties vary widely across the United States, which can influence the potential for herbicide injury and the rates that can be used safely on various crops. Corn, sorghum and soybean response to saflufenacil has been variable across sites. The objective of this research was to determine the effects of soil type, soil pH, crop seeding depth, and herbicide rate on corn, sorghum, and soybean response to preemergence applications of saflufenacil.

MATERIALS AND METHODS

Two soils were collected for the experiment. One was a Reading silt loam soil (Reading) consisting of 2, 74, and 24% sand, silt, and clay respectively from the Kansas State University Research Farm at Ashland Bottoms near Manhattan, KS in September of 2010. Soil pH was 5.9, CEC was 20.0 meq/100g, and organic matter (OM) was 3.9%. The other was a Farnum fine

sandy loam soil (Farnum) consisting of 82, 12, and 6% sand, silt, and clay respectively, which was collected from the Kansas State University Sandyland Experiment Field near St. John, KS in September of 2010. Soil pH was 5.6, CEC was 2.4 meq/100g, and OM was 0.9%.

Each soil type was divided in half with the pH of one half being lowered to a target pH of 5.6, and the pH of the other half being raised to a target pH of 7.5. The pH of the Reading soil was lowered by mixing 0.26 g of 90% elemental sulfur per kg of soil. The pH of the Farnum soil was already at 5.6, so no pH-lowering amendment was added. To raise the pH of the Reading soil, 2.2 g of Hi-Yield Horticultural Hydrated Lime (131% calcium carbonate equivalent) was mixed per kg of soil. The pH of the Farnum soil was raised by mixing 1.1g of Hi-Yield Horticultural Hydrated Lime per kg of soil. All mixing was done by tumbling the soil and amendments in a cement mixer for 20 minutes. After the soil was mixed, it was steamed and transferred into sealed plastic trash containers and incubated for one month. Soil pH adjustments were based on soil test results and the recommendations provided by Dr. David Mengel (personal communication). After incubating, soil was sent to the Kansas State University Soil Testing Lab to be retested. The Reading soil had a low pH of 5.7 with a CEC of 21.5 and a high pH of 7.5 with a CEC of 20.6. The Farnum soil had a low pH of 4.9 with a CEC of 2.2, and a high pH of 8.4 with a CEC of 5.8. Soil was then transferred to plastic pans measuring 20.5 cm long, 28 cm wide, and 6.5 cm deep with holes in the bottom to facilitate drainage and sub-surface irrigation.

The three crop varieties chosen for this experiment were “Dekalb DKC63-42” corn, “Asgrow AG4403” soybean, and “Pioneer 84G62” grain sorghum. On February 21st and April 4th, five seeds of each crop were planted into the pans at evenly spaced intervals at depths of 2 and 4 cm. Herbicide treatments were applied preemergence on February 22nd and April 5th using

a bench-type sprayer equipped with a single TeeJet 80015LP nozzle tip calibrated to deliver 187 L ha^{-1} at 138 kPa. Herbicide treatments included saflufenacil at rates of 0, 50, 100, and 150 g/ha. All pans were then watered one hour later using the bench-type sprayer equipped with a single TeeJet 1/4TTJ15-VS nozzle tip calibrated to deliver 4.0 L min^{-1} at 138 kPa and cycled repeatedly across the flats until 1 cm of irrigation was applied. Three days later all pans were again watered in the same manner as described before to simulate 1 cm of irrigation or precipitation. Thereafter, pans were sub-irrigated as needed starting February 28th and April 9th. Plants were grown under greenhouse conditions at 32/22 $\pm$ 2 C day/night temperatures and 16/8 h day/night periods. The supplemental photosynthetic photon flux was 80 µmol m^{-2} s^{-1}. Visible injury ratings were recorded at 7 and 14 days after crop emergence (DAE). Injury ratings were based on a scale of 0 to 100% where 0 equals no injury and 100% equals mortality.

The experiment had a 2 by 2 by 2 by 4 factorial treatment arrangement of soil type, pH, planting depth, and herbicide rate in a completely randomized design. All ratings were subjected to ANOVA using PROC MIXED in SAS with soil, pH, depth, and rate factors fixed and replication being random. LSD's were calculated using pdmix800. Treatments were replicated four times and the experiment was repeated. Both runs were tested for homogeneity of variance, subjected to ANOVA, and runs are presented separately because of significant run effects.

RESULTS AND DISCUSSION

Crop injury on corn, soybean, and grain sorghum was statistically similar within runs for both evaluation intervals, so only the 14 DAE data are presented.

Corn

All treatment variables were significant for corn injury in Run 1, with significant interactions for soil × pH and soil × saflufenacil rate (Table 3.1). Corn injury in general was greater on the Farnum soil than the Reading soil, especially at the higher saflufenacil rates and high pH (Tables 3.2 and 3.3). Corn injury was greater at high pH than low pH on the Farnum soil, but less than 8% and not different between low and high pH on the Reading soil (Table 3.2). Corn injury on the Farnum soil increased as saflufenacil rate increased, however, corn injury was less than 10% and not different among saflufenacil rates on the Reading soil (Table 3.3). Corn planted 2 cm deep was injured 21% compared to 12% injury for corn planted 4 cm deep (Table 3.4).

A significant four-way interaction among all variables evaluated occurred for corn injury in Run 2 (Table 3.5). Corn injury was minimal with the Reading soil, regardless of pH, saflufenacil rate, or seeding depth (Table 3.6). Corn injury was minimal at the low saflufenacil rate on the Farnum soil, regardless of pH or soil depth, and also at the medium saflufenacil rate with the high pH. Corn injury on the Farnum soil at the high and medium saflufenacil rate and low pH was erratic and inconsistent for seeding depth, perhaps due to mouse damage during this run.

Overall, corn injury appeared to be most significantly affected by soil type, with corn planted in the Reading soil being less injured than corn planted in the Farnum soil. In general, corn injury increased with increasing saflufenacil rate on the Farnum soil. The effect of soil pH and seeding depth on corn injury from saflufenacil was not consistent and inconclusive.

Soybean

All treatment variables except depth were significant for soybean injury in Run 1, with one significant interaction of soil × pH (Table 3.7). Soybean injury was greater on the Farnum soil than the Reading soil, regardless of pH, and in general greater at the high pH than the low pH for the Farnum soil (Table 3.8). The highest soybean injury occurred on the Farnum soil with the high pH. Soybean injury from saflufenacil was greater at the 150 g/ha rate than the 50 or 100 g/ha rate (Table 3.9).

A significant interaction occurred between soil and pH for soybean injury in Run 2, but seed depth and saflufenacil rate were not significant (Table 3.10). Soybean injury was very high for the low pH on the Farnum soil, but minimal for the high pH on the Farnum soil or with the Reading soil, regardless of pH (Table 3.11). High injury with the low pH and Farnum soil may have been confounded by mouse damage.

Treatment effects on soybean injury in Run 2 were inconclusive, but in Run 1, soybean injury appeared to be most significantly affected by soil type, with soybeans planted in the Reading soil being less injured than corn planted in the Farnum soil. Soybean injury increased with increasing saflufenacil rate and was greater at high pH than at low pH.

Grain Sorghum

A significant three-way interaction of soil × pH × depth occurred for grain sorghum injury in Run 1 (Table 3.12). Grain sorghum injury tended to be greater on the Farnum soil than the Reading soil for each respective depth by pH combination, except at the 4 cm depth and high pH combination, where there was no difference between the soils (Table 3.13). Grain sorghum injury on the Reading soil tended to be greater at the high pH than the low pH regardless of seed depth. Sorghum injury was not different for seed depth at the low pH soil, but tended to be

greater at the 4 cm depth than at the 2 cm depth on the high pH soil. Grain sorghum planted at the 2 cm depth on the Farnum soil tended to be injured more at the high pH than the low pH (Table 3.13).

The three-way interaction of soil × pH × saflufenacil rate significantly affected grain sorghum injury in Run 2 (Table 3.14). Minimal grain sorghum injury occurred with the Reading soil, regardless of pH or saflufenacil rate. Grain sorghum injury on the Farnum soil tended to be higher at the 150 g/ha saflufenacil rate than the 50 or 100 g/ha rates at the high pH, however at the low pH, grain sorghum injury was erratic and inconsistent (Table 3.15).

Overall grain sorghum injury appeared to be influenced the most by soil type, with less injury occurring in the Reading soil than the Farnum soil. Grain sorghum injury was generally greater at the high pH compared to the low pH.

Discussion

Soil type had the greatest impact on injury across the three crops, with crop injury consistently greater in the Farnum soil compared to the Reading soil. The differences due to soils most likely could be correlated to CEC for application to different soil environments. The risk of crop injury is likely much greater on low CEC than on high CEC soils as reported for other herbicides (Kerr 2004; Blumhorst et al. 1990; Harper 1994; Peter and Weber 1985). Soil pH, saflufenacil rate, and seed depth also can influence the risk of crop injury, but results were not consistent in this study. Although not compared directly, corn generally was injured less than soybean or grain sorghum. This is consistent with the labeled rates for saflufenacil, which are higher in corn, where saflufenacil is labeled up to 75 g/ha, than either grain sorghum or soybeans where saflufenacil is only labeled to 50 and 25 g/ha respectively (Anonymous 2008; Thompson et al. 2011). It appears that several factors can influence the potential for crop injury

from PRE applications of saflufenacil on corn, soybean, and grain sorghum, but soil characteristics, especially soil CEC, appears to be the most important factor.

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Table 3.1. ANOVA for corn injury 14 DAE, Run 1.

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	23	20080.98958	873.08650	4.01	<.0001
Error	72	15681.25000	217.79514		
Corrected Total	95	35762.23958			

R-Square	Coeff Var	Root MSE	injury Mean
0.561514	90.52759	14.75788	16.30208

Source	DF	Type I SS	Mean Square	F Value	Pr > F
soil	1	7794.010417	7794.010417	35.79	<.0001
pH	1	2156.510417	2156.510417	9.90	0.0024
soil*pH	1	2156.510417	2156.510417	9.90	0.0024
depth	1	1708.593750	1708.593750	7.84	0.0065
soil*depth	1	6.510417	6.510417	0.03	0.8632
pH*depth	1	2.343750	2.343750	0.01	0.9177
soil*pH*depth	1	94.010417	94.010417	0.43	0.5133
rate	2	1494.270833	747.135417	3.43	0.0378
soil*rate	2	2241.145833	1120.572917	5.15	0.0082
pH*rate	2	572.395833	286.197917	1.31	0.2751
soil*pH*rate	2	569.270833	284.635417	1.31	0.2770
depth*rate	2	182.812500	91.406250	0.42	0.6588
soil*depth*rate	2	494.270833	247.135417	1.13	0.3272
pH*depth*rate	2	285.937500	142.968750	0.66	0.5218
soil*pH*depth*rate	2	322.395833	161.197917	0.74	0.4806

Table 3.2. Corn injury 14 DAE as influenced by soil and pH, averaged over saflufenacil rate and seed depth, Run 1.

pH	Reading	Farnum
	----------% injury----------	
4.9-5.7	7	16
7.5-8.4	7	35
LSD	11	

Table 3.3. Corn injury 14 DAE as influenced by soil and saflufenacil rate, averaged over pH and seed depth, Run 1.

Saflufenacil Rate	Reading	Farnum
	----------% injury----------	
50	8	13
100	7	28
150	6	34
LSD	15	

Table 3.4. Corn injury 14 DAE as influenced by seed depth, averaged over soil, pH, and saflufenacil rate, Run 1.

Seeding Depth	% Injury
2 cm	21
4 cm	12
LSD	6

Table 3.5. ANOVA for corn injury 14 DAE, Run 2.

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	23	27926.48958	1214.19520	4.67	<.0001
Error	72	18733.25000	260.18403		
Corrected Total	95	46659.73958			

R-Square	Coeff Var	Root MSE	injury Mean
0.598514	190.9373	16.13022	8.447917

Source	DF	Type I SS	Mean Square	F Value	Pr > F
soil	1	6353.760417	6353.760417	24.42	<.0001
pH	1	1418.343750	1418.343750	5.45	0.0223
soil*pH	1	1658.343750	1658.343750	6.37	0.0138
depth	1	201.260417	201.260417	0.77	0.3821
soil*depth	1	297.510417	297.510417	1.14	0.2885
pH*depth	1	15.843750	15.843750	0.06	0.8058
soil*pH*depth	1	49.593750	49.593750	0.19	0.6637
rate	2	3345.020833	1672.510417	6.43	0.0027
soil*rate	2	2996.895833	1498.447917	5.76	0.0048
pH*rate	2	1209.812500	604.906250	2.32	0.1051
soil*pH*rate	2	1136.687500	568.343750	2.18	0.1199
depth*rate	2	916.270833	458.135417	1.76	0.1792
soil*depth*rate	2	743.145833	371.572917	1.43	0.2465
pH*depth*rate	2	3547.312500	1773.656250	6.82	0.0019
soil*pH*depth*rate	2	4036.687500	2018.343750	7.76	0.0009

Table 3.6. Corn injury 14 DAE as influenced by soil, pH, saflufenacil rate, and seed depth, Run 2.

	Reading				Farnum			
	4.9-5.7 pH		7.5-8.4 pH		4.9-5.7 pH		7.5-8.4 pH	
Rate	2 cm	4 cm	2 cm	4 cm	2 cm	4 cm	2 cm	4 cm
	% injury							
50	0	0	0	0	4	0	0	0
100	0	0	0	0	63	14	1	5
150	0	0	0	4	14	54	38	8
LSD	43							

Table 3.7. ANOVA for soybean injury 14 DAE, Run 1.

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	23	50849.73958	2210.85824	5.47	<.0001
Error	72	29081.25000	403.90625		
Corrected Total	95	79930.98958			

R-Square	Coeff Var	Root MSE	injury Mean
0.636171	58.55394	20.09742	34.32292

Source	DF	Type I SS	Mean Square	F Value	Pr > F
soil	1	29225.26042	29225.26042	72.36	<.0001
pH	1	6583.59375	6583.59375	16.30	0.0001
soil*pH	1	1794.01042	1794.01042	4.44	0.0386
depth	1	906.51042	906.51042	2.24	0.1385
soil*depth	1	1239.84375	1239.84375	3.07	0.0840
pH*depth	1	44.01042	44.01042	0.11	0.7423
soil*pH*depth	1	219.01042	219.01042	0.54	0.4639
rate	2	4759.89583	2379.94792	5.89	0.0043
soil*rate	2	844.27083	422.13542	1.05	0.3569
pH*rate	2	820.31250	410.15625	1.02	0.3674
soil*pH*rate	2	969.27083	484.63542	1.20	0.3072
depth*rate	2	1053.64583	526.82292	1.30	0.2777
soil*depth*rate	2	885.93750	442.96875	1.10	0.3395
pH*depth*rate	2	1009.89583	504.94792	1.25	0.2926
soil*pH*depth*rate	2	494.27083	247.13542	0.61	0.5451

Table 3.8. Soybean injury 14 DAE as influenced by soil and pH, averaged over saflufenacil rate and seed depth, Run 1.

pH	Reading	Farnum
	----------% injury----------	
4.9-5.7	13	39
7.5-8.4	21	65
LSD	15	

Table 3.9. Soybean injury 14 DAE as influenced by saflufenacil rate, averaged over soil, pH, and seed depth, Run 1.

Saflufenacil Rate (g/ha)	% Injury
50	28
100	31
150	44
LSD	12

Table 3.10. ANOVA for soybean injury 14 DAE, Run 2.

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	23	78453.9063	3411.0394	6.72	<.0001
Error	72	36531.2500	507.3785		
Corrected Total	95	114985.1563			

R-Square	Coeff Var	Root MSE	injury Mean
0.682296	121.1432	22.52506	18.59375

Source	DF	Type I SS	Mean Square	F Value	Pr > F
soil	1	26833.59375	26833.59375	52.89	<.0001
pH	1	25187.76042	25187.76042	49.64	<.0001
soil*pH	1	21450.26042	21450.26042	42.28	<.0001
depth	1	356.51042	356.51042	0.70	0.4047
soil*depth	1	356.51042	356.51042	0.70	0.4047
pH*depth	1	94.01042	94.01042	0.19	0.6682
soil*pH*depth	1	250.26042	250.26042	0.49	0.4847
rate	2	282.81250	141.40625	0.28	0.7576
soil*rate	2	273.43750	136.71875	0.27	0.7646
pH*rate	2	581.77083	290.88542	0.57	0.5662
soil*pH*rate	2	391.14583	195.57292	0.39	0.6815
depth*rate	2	550.52083	275.26042	0.54	0.5836
soil*depth*rate	2	109.89583	54.94792	0.11	0.8975
pH*depth*rate	2	978.64583	489.32292	0.96	0.3861
soil*pH*depth*rate	2	756.77083	378.38542	0.75	0.4780

Table 3.11. Soybean injury 14 DAE as influenced by soil and pH, averaged over saflufenacil rate and seed depth, Run 2.

pH	Reading	Farnum
	----------% injury----------	
4.9-5.7	3	67
7.5-8.4	1	4
LSD	17	

Table 3.12. ANOVA for grain sorghum injury 14 DAE, Run 1.

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	23	13353.90625	580.60462	1.81	0.0303
Error	72	23131.25000	321.26736		
Corrected Total	95	36485.15625			

R-Square	Coeff Var	Root MSE	injury Mean
0.366009	67.87761	17.92393	26.40625

Source	DF	Type I SS	Mean Square	F Value	Pr > F
soil	1	4469.010417	4469.010417	13.91	0.0004
pH	1	1881.510417	1881.510417	5.86	0.0180
soil*pH	1	250.260417	250.260417	0.78	0.3804
depth	1	283.593750	283.593750	0.88	0.3506
soil*depth	1	31.510417	31.510417	0.10	0.7550
pH*depth	1	527.343750	527.343750	1.64	0.2042
soil*pH*depth	1	1881.510417	1881.510417	5.86	0.0180
rate	2	1560.937500	780.468750	2.43	0.0953
soil*rate	2	725.520833	362.760417	1.13	0.3290
pH*rate	2	47.395833	23.697917	0.07	0.9290
soil*pH*rate	2	253.645833	126.822917	0.39	0.6753
depth*rate	2	426.562500	213.281250	0.66	0.5180
soil*depth*rate	2	53.645833	26.822917	0.08	0.9200
pH*depth*rate	2	142.187500	71.093750	0.22	0.8020
soil*pH*depth*rate	2	819.270833	409.635417	1.28	0.2856

Table 3.13. Grain Sorghum injury 14 DAE as influenced by soil, pH, and seeding depth, averaged over saflufenacil rate, Run 1.

Seeding	Reading		Farnum	
Depth	4.9-5.7 pH	7.5-8.4 pH	4.9-5.7 pH	7.5-8.4 pH
	----------% injury----------			
2 cm	13	21	23	42
4 cm	14	30	38	30
LSD	23			

Table 3.14. ANOVA for grain sorghum injury 14 DAE, Run 2.

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	23	8445.83333	367.21014	1.95	0.0168
Error	72	13537.50000	188.02083		
Corrected Total	95	21983.33333			

R-Square	Coeff Var	Root MSE	injury Mean
0.384193	253.1459	13.71207	5.416667

Source	DF	Type I SS	Mean Square	F Value	Pr > F
soil	1	2016.666667	2016.666667	10.73	0.0016
pH	1	126.041667	126.041667	0.67	0.4156
soil*pH	1	234.375000	234.375000	1.25	0.2679
depth	1	204.166667	204.166667	1.09	0.3009
soil*depth	1	66.666667	66.666667	0.35	0.5534
pH*depth	1	9.375000	9.375000	0.05	0.8239
soil*pH*depth	1	26.041667	26.041667	0.14	0.7109
rate	2	572.395833	286.197917	1.52	0.2252
soil*rate	2	913.020833	456.510417	2.43	0.0954
pH*rate	2	1697.395833	848.697917	4.51	0.0142
soil*pH*rate	2	1417.187500	708.593750	3.77	0.0278
depth*rate	2	297.395833	148.697917	0.79	0.4574
soil*depth*rate	2	519.270833	259.635417	1.38	0.2579
pH*depth*rate	2	189.062500	94.531250	0.50	0.6070
soil*pH*depth*rate	2	156.770833	78.385417	0.42	0.6607

Table 3.15. Grain Sorghum injury 14 DAE as influenced by soil, pH, and saflufenacil rate, averaged over depth, Run 2.

Saflufenacil	Reading		Farnum	
Rate (g/ha)	4.9-5.7 pH	7.5-8.4 pH	4.9-5.7 pH	7.5-8.4 pH
	----------% injury----------			
50	1	2	5	3
100	0	0	31	4
150	0	2	3	15
LSD	22			