Response of Susceptible and Putative Resistant Goosegrass (*Eleusine indica*) Populations to Protoporphyrinogen Oxidase Inhibitors

by

William Barrett Head

A thesis submitted to the Graduate Faculty of
Auburn University
in partial fulfillment of the
requirements for the Degree of
Master of Science

Auburn, Alabama
May 5, 2018

Keywords: goosegrass, oxadiazon, herbicide resistance, PPO Inhibitors

Approved by

Dr. Scott McElroy, Professor, Crop, Soils and Environmental Sciences
Dr. Glenn Wehtje, Professor, Crop, Soils and Environmental Sciences
Dr. Steve Li, Associate Professor and Extension Specialist

Abstract

Goosegrass is considered one of the five most troublesome weeds in the world (Holm, 1991). Goosegrass is commonly found in high traffic and stress areas where the desired turf is thin and vulnerable for weed infestations. The use of preemergence herbicides has become important for goosegrass control because of limited options for postemergence control. Studies were conducted to investigate oxadiazon suspected-resistant goosegrass populations from Richmond, VA. The SR populations showed minimal response to increasing oxadiazon rates while susceptible populations showed greater tissue damage and plant necrosis. Research suggests that there is a resistance mechanism within the SR populations. Greenhouse studies were conducted to evaluate the response of oxadiazon-resistant goosegrass to postemergence applications of protoporphyrinogen oxidase inhibitors. Studies were conducted to determine response of five goosegrass ecotypes ('Craft Farms', 'CCV', 'RB' 'Alabama Crop Wild Type', and 'Texas') to oxadiazon, flumioxazin, and sulfentrazone. A dose response curve was developed to predict at which rate a goosegrass population will be inhibited. The rate at which a given herbicide inhibits growth 50 and 90%, also referred to as I_{50} and I_{90} values are commonly presented in dose response research. Estimated *i* values from susceptible (S) seed were several orders of magnitude higher than *i* values from oxadiazon resistant (OR) plants. The OR populations in Auburn University trials required an estimated 13.65 and 3522 kg ai $ha^{-1,}$ respectively, of the population at 42 DAT while the Craft Farms (S) plants required only 0.23 and 2.52 kg ai ha^{-1}, respectively. These data indicate that oxadiazon rates required to achieve commercially acceptable control (90%) were significantly higher for the SR than the S successions.

Acknowledgements

I would like to thank my major advisor, Dr. Scott McElroy, and my advisory committee, Drs. Steve Li and Glenn Wehtje, for their support and guidance while in the M.S. program at Auburn University. I would also like to thank research technician Adam Boyd for his support, help, and patience while in the program. I also wish to express my appreciation to my fellow graduate students for their help and guidance. Finally, I would like to thank my family in Clanton, AL for their love, support, and encouragement while here in Auburn.

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

Literature Review

Weed problems arise when a plant species or a group of species interfere with a man's activities, his health or his pleasures (Fryer, 1979). A weed is commonly defined as a plant growing out of place or where it is unwanted or needed. Weeds exist because of the human ability to judge and select among species in the plant kingdom (Radosevich et al. 1997). There are approximately 250,000 species of plants on the globe, but there are less than 250 plant species that are considered weeds (Radosevich et al. 1997). The most common way to categorize a weed is to classify per its habitat. Two examples of weed classification based on habitat are agrestals and ruderals. Agrestals are weeds found in tilled, arable land from fields of grains and root crops, orchards, hoed gardens, and plantations. (Holzner and Numata 2013). Ruderals are weeds found on garbage sites, dung hills, roadsides, railways lines and other rights of way areas. They spend much of their life cycle reproducing and have little competition for resources from other plants (Cobb 2011). These plants may not cause problems to the desired crop or turf stand but instead are unsightly and unpleasing to the eye. Other subdivisions of classifying weeds are characteristics such as monocot (narrow leaved) or dicot (broad leaved) (Holzner and Numata 2013).

Dicots, or broadleaf plants, are numerous in both crop and turfgrass settings. They include many weedy plant species such as dandelion, clover, spurge, as well as many trees and shrubs. Dicots are different from monocots in that they have two cotyledons in their seed. The vein arrangement in their leaves is "net-like", with flowers in multiples of four or five (Christians 1998). Monocots are mostly grassy weeds but also includes sedges (*Cyperaceae*), lily/onion (*Liliaceae*) and the spiderwort family (*Commelinaceae*). The monocots are flowering plants with one cotyledon. These plants have parallel venation in the leaves, stems that contain vascular

bundles, and parts of the flower in multiples of three (Christians, 1998). The life cycle of plant species is categorized as annual, perennial, or biennial (McElroy et al. 2007).

An annual plant completes its life cycle in a one-year period. The annual weeds consist of summer annuals and winter annuals. The summer annuals germinate in the spring and in the fall with the cooling temperatures. The winter annuals germinate in the fall, live through the winter, produce a seed crop in the spring and die with warming temperatures of summer (Christians 1998). Perennial weeds live for more than two years and may produce many times before dying. These plants maintain the same vegetative growth and root system year after year (Radosevich et al. 1997). Perennial weeds are most difficult to control because of their ability to mimic the turfgrass that is being infested. Because of this, is it difficult to selectively remove the weed with a herbicide without harming the turfgrass (Christians 1998). Biennials are plants that survive longer than one year but die before completing a two-year life cycle. In year one the plant produces vegetative growth from a seedling and forms a rosette. Biennials often mature into large plants with fleshy roots (Radosevich et al. 1997).

Many turfgrass weeds are grasses. Grasses possess a supapical meristem which allows them to adapt to mowing. The meristems of most plants are apical meaning that the new growth is added at the end of the stem each new growing season. The supapical meristems in grasses is located below the tip of the grass blade which allows the adaptation for low mowing heights (Christians 1998).

Goosegrass

Goosegrass (*Eleusine indica)* is a native perennial in tropical areas, but also can behave as an annual due to colder temperatures (McElroy et al. 2007). This characteristic of climate adaptation allows goosegrass to persist in many different areas of the world. In the Eastern portion of the United States, goosegrass is a problem in all areas except for the Northern most areas of Maine. Goosegrass is often mistaken for crabgrass because of the "silvery appearance" of its lower sheaths. There are a few key differences between goosegrass and crabgrass: The individual seed of goosegrass is larger than crabgrass seed, goosegrass has a folded vernation whereas crabgrass has a rolled vernation, and goosegrass germination is generally two to three weeks later than crabgrass in the spring (Christians 2007).

Goosegrass, also called wiregrass or crowfoot grass, is considered one of the five most troublesome weeds in the world. In previous reports, goosegrass has been listed as problematic for 46 different crop species in more than 60 countries (Holm 1991). Surveys carried out in the 14 adjacent southern states to determine the most disturbing weeds in agronomic crops ranked goosegrass as the following: 15^{th} in corn and cotton, and 34^{th} in soybean in 2009 (Webster and Nichols 2012).

Goosegrass is commonly found in high traffic areas, high stress areas and areas where the desired turf is thin and vulnerable for weed populations (McCarty 1991). It is called a "gap colonist", this means that seeds from the plant germinate rapidly in sunlit holes in the turf caused by injury from insects, wear from equipment or people and divots from golfers (Busey 1999). Goosegrass thrives in areas that are poorly drained, compacted soils that are usually found in high foot traffic areas such as golf greens and athletic fields (McCarty 1991). In pasture situations, goosegrass thrives in areas where animal traffic is constant. Areas around gates,

watering areas, shade, are some common areas where goosegrass can be found (Bryson and Defelice 2009). Although some believed that goosegrass could serve as a forage for cattle, goosegrass has been documented in Asia to contain enough hydrogen cyanide in the 1-2 tiller growth stages to kill calves and sheep (Holm 1991).

Goosegrass Biology

Goosegrass can be identified by its folded leaf vernation along the midvein, no auricles, a flattened leaf sheaths, and a short, membranous ligule. Few hairs may be found near the leaf collar and near the base of the leaves, especially when the plant is mature. The center of the plant appears silver because of the white almost translucent leaf sheaths (Bryson and DeFelice et al. 2009).

Goosegrass reproduces by seed that is formed in two flattened rows on spikelets on various amounts of spikes (Bryson and DeFelice et al. 2009). Goosegrass can produce as many as 3 to 6 seedheads per spikelet and can produce up to 50,000 total seeds per plant (Uva, 1997). The seed of goosegrass is considered to remain viable in the soil for as long as five years (Kranz et al. 1977). Germination of goosegrass occurs in the late spring to early summer, a few weeks after crabgrass germination (Callahan 1986). There have been many studies conducted on the control of goosegrass but studies on germination of goosegrass has been insignificant (Nishimoto and McCarty 1997). Light, alternating temperatures, and comparatively high daytime temperatures result in successful germination of goosegrass seed (Engel 1959). Goosegrass seed germinated well with alternating temperatures of 35 C daytime temperature and 25 C night whereas temperatures of 25 C daytime and 15 C nighttime resulted in less seed germinated

(Johnson, 2008). When outdoor temperatures were at a constant of 20, 25 and 35 C goosegrass germination was less than 10%. However, with the addition of light and fluctuating the temperature in cycles of 8 hours the germination increased to 99. Goosegrass germination is best when temperatures are fluctuating, this is typical of conditions of bare ground and thinned or scalped turf. Maintaining a healthy and dense growing turf will keep germination of goosegrass to a minimum. Past research has shown that the seeds of goosegrass show considerable survival over several years and viability can remain high even after 3 years (Nishimoto and McCarty 1997).

Planting depth also has an effect of goosegrass germination. Holm (1991) reported that germination of goosegrass seedling decreased as the burial depth increased. Two populations were planted, R and S, both population planted at the 0cm depth emerged 5 days after planting and for seeds planted at the 2cm depth the seeds began to germinate at 6 days after planting. Research by Hawton and Drennan (1980) showed that goosegrass seed emergence declined at depths greater than 4 cm and no emergence was reported at depths greater than 8 cm. In hot climates where the soil surface is always moist and warm, much of goosegrass seed germinate continuously (Wiecko 2000).

Goosegrass Control

Weed control consists of: the reduction of the competitive ability of an existing population of weeds in a crop, the foundation of a barrier to the evolution of future significant weeds within that crop or turf stand; and the deterrence of weed problems in future crops, either from the existing weed reservoir or from additions to that weed flora (Cobb and Reade 2011).

The use of copper sulphate was first introduced to control charlock (*Sinapis arvensis)* in oats. It was the discovery of the phenoxyacetic herbicides in Britain and the United States from 1942 to 1944 that marked the beginning of the herbicide phase of the "Chemical Era of Agriculture" (Timmons 1970). Herbicides may be classified by several standards. These standards include movement and selectivity in the plant, chemistry, application, timing, and mode of action. When a herbicide is classified by application timing it is listed as a preemergent or postemergent chemical.

Monosodium methanearsonate (MSMA) plus metribuzin (Sencor) as two to three application treatment 7-10 days apart has provided effect for postemergence goosegrass control for many years. MSMA is an organic arsenal that binds tightly to soil particles only in the organic form, which is notably less toxic than the inorganic form (Ross and Lembi et al. 2009). Registered for use in cotton, turfgrass, and forestry, MSMA was a relied upon herbicide for farmers and turf managers which made goosegrass control in turfgrass difficult when restrictions were placed on the herbicide (EPA 2013). Total arsenic concentrations surpassed acceptable thresholds in groundwater in Florida which resulted in MSMA to be no longer an option in most turfgrass sites (EPA 2006). With these limitations on MSMA, overall goosegrass control has been difficult for turf managers (Wiecko 2000).

Protoporphyrinogen Oxidase Inhibitors

There are several herbicides such as oxadiazon (Ronstar), oxyfluorfen (Goal), sulfentrazone (Dismiss), and flumioxazin (Sureguard) that inhibit protoporphyrinogen IX oxidase (Protox), the enzyme that converts protoporphyrinogen IX to protoporphyrin IX (Proto). A concise explanation is provided by Duke (1991): "This results in uncontrollable autoxidation of the substrate and results in accumulation of Proto. Blockage of the porphyrin pathway at this site inhibits synthesis of both chlorophylls and heme. Heme is a feedback regulator of the porphyrin pathway. Thus, inhibition of Protox also deregulates the pathway, causing increased carbon flow to the accumulating pool of Proto. Proto is a potent photosensitizer that generates high levels of singlet oxygen in the presence of molecular oxygen and light. In plants treated with these herbicides, damage is light dependent and closely correlated with the level of Proto that accumulates. Proto accumulation is apparently largely extraplastidic, resulting in rapid photodynamic damage to the plasmalemma and tonoplast. After high levels of Proto accumulate in response to these herbicides, protochlorophyllide (PChlide) levels can increase also; however, Proto appears to be the primary photodynamic pigment responsible for the herbicidal activity".

Preemergence Herbicides

Preemergence herbicides are important to preventing major goosegrass contamination. Several herbicide families and modes-of-action have been used to control goosegrass. The earliest herbicides used for preemergence goosegrass control were the dinitroanilines such as pendimethalin, prodiamine, oryzalin, and benefin (Busey 1999). With restrictions to MSMA (monosodium methanearsonate) and the challenge of controlling mature goosegrass plants, the use

of preemergence herbicides has become an important part of the control of goosegrass (Busey 1999). Other herbicides that have proven to provide quality control include oxadiazon, indaziflam and dithiopyr (Johnson 2008).

Oxadiazon is an organic, selective preemergence herbicide in the oxadiazole chemical class. The trade name is Ronstar, it is registered for use on many warm season turfgrasses and is listed to control goosegrass (Beard 2005). Oxadiazon is persistant and long lasting in the soil, providing season long control. The field half-life is 60 days. However, some minor issues include temporary discoloration of desired turf including bentgrass and bermudagrass. Also the reseeding of desired grass should be delayed four months after application of herbicide (Ross and Lembi 2009). The recommended application rate for oxadiazion is 2-4 Kg/ha (Beard 2005).

Dithiopyr is a preemergence herbicide that controls annual grasses in warm and cool season turfgrass. Dithiopyr is a mitosis inhibitor in the pyridine chemical class, the trade name is Dimension. Past research showed that a single application of dithiopyr EC applied at rates < 1.1 kg/ha did not provide adequate control, however sequential applications of dithiopyr EC provided superior control of goosegrass (Murphy et al. 1986).

Indaziflam (Specticle) is a preemergence herbicide released in 2012. This herbicide belongs to the alkylazine chemical class, a chemical class that stops the growth of weed seedlings through the inhibition of cellulose biosynthesis (EPA 2013). Indaziflam is applied to the soil (Breeden and Brosnan 2009) and has a long residual with a half-life of 150 days (EPA 2013), this is significantly longer than other preemergence herbicides such as oxadiazon with an estimated half-time of 60 days. Indaziflam can impact surface water quality and enter streams and lake through runoff for several months after application (EPA 2013).

Postemergence Herbicides

Postemergence herbicides are applied to the weed foliage. Several on the market report goosegrass control. However, only a few will control mature goosegrass (2-3 tiller stage) other than MSMA plus metribuzin. Because of preemergent herbicides and their efficacy because of resistance, poor residual activity and environmental conditions turf managers rely heavily on postemergence control of goosegrass (Dernoeden et al. 1984).

Diclofop (Illoxan 3 EC) is a restricted use herbicide that has a maximum use rate of 1.0 kg ai ha. The Illoxan 3 EC herbicide label states that control can be only achieved with goosegrass with less than 2 tillers, and is only safe on established bermudagrass (10 cm long stolons) (Anonymous, 2004). Diclofop can control mature goosegrass growing on putting greens but failed to control control at fairway heights (McCarty et al. 1991). Foramsulfuron (Revolver), a sulfonylurea herbicide, has been reported to control goosegrass early post-emergence but fails to adequately control mature (McCullough et al. 2012). Previous research by Busey (1999) reported that foramsulfuron alone resulted in only moderate control of goosegrass (< 50**%**)**,** but with the addition of metribuzin control of goosegrass was effective (>85 %) (Busey 1999). The effective rate of metribuzin can cause injury to bermudagrass (0.21 kg/ha) even rates as low as (0.07kg/ha) (Murdoch and Nishimoto 1982) however metribuzin is the most important factor when using a foramsulfuron mixture (McCarty 1991).

Sulfentrazone, a triazolinone herbicide, is labeled for postemergence goosegrass control. Past research reported that a single application of sulfentrazone applied alone achieved less than 35 % control of goosegrass but with the addition of nicosulfuron, which is not labeled for use on

turfgrass, improved the control to around 60 %. Sequential applications of sulfentrazone alone improved the control to 65 % but sequential applications with the addition of nicosulfuron improved goosegrass control to 84 % at 9 weeks after treatment (McCullough et al. 2012).

Herbicide Resistance

Herbicide use has simplified the management of weeds in many crops. This method of weed control is much simpler than tillage, burning cover crops, and crop rotations but chemical weed control has some serious risks and drawbacks. The appearance of herbicide resistant weeds is threatening the usefulness of herbicides. It is reported that there are resistant populations of goosegrass in eight countries and nine different states across the US, including Eight herbicide families and six modes of action, and in over eleven different cropping systems. In 1973 the first case of herbicide resistance was reported in goosegrass by Dr. Harold Coble in North Carolina. The population of goosegrass was reported to be unaffected to trifluralin in cotton fields in the southern area of North Carolina and northeastern South Carolina. Field trials were repeated showed that trifluralin and other dinitroaniline herbicides were used on cotton were inefficient on goosegrass at this location (Heap 2013). Goosegrass poses a problem in warm and cool season turf because of the continued use of herbicides with the same mode of action allowing the weed to evolve and become resistant to the active ingredient such as prodiamine. The first reported case of herbicide resistance in turf was in 2003 on the island of Kaudai, Hawaii with triazines and in eastern Georgia on a golf course with dinitroanilines (Heap 2013). The two populations

of goosegrass displayed a level of resistance 100 to 200 times that of the susceptible populations when treated with metribuzin at 0.28 kg ai ha plus MSMA at 2.20 kg ai ha (Brosnan et al. 2008).

Resistance to PPO Inhibitors

The first report of resistance to PPO inhibitors was recorded in common waterhemp [*Amaranthus tuberculus* (Moq.) J.D. Sauer] in 2001 in soybean fields in the Midwestern United States. The herbicide was acifluorfen (Shoup et al. 2003). The first reported case of an annual grass being resistant to a PPO inhibitor occurred on a golf course in Richmond, Virginia. Oxadiazon (Ronstar) had been used as a preemergence herbicide for the control of goosegrass since the early 1990's. Lack of goosegrass control was noted when oxadiazon was applied at rates of 3.4 to 4.5 kg ai/ha. It is unknown whether this biotype was an isolated incident or if there are other resistant populations of goosegrass in the area (Askew 2013).

Current Herbicidal Control and Future Research.

With the recent restrictions put on the use of MSMA, goosegrass control has become more problematic for turf managers. The use of preemergence herbicides has become important for control of goosegrass because of the lack of options with postemergence control and the difficulty of eradicating a mature goosegrass plant. Overall choices of herbicides are limited, both pre and post emergence control when it comes to eradicating goosegrass.

Oxadiazon is a popular preemergence option for turf managers. However, with only a few options to choose from to achieve quality control, one must be worried about developing a resistant goosegrass population. With a resistant population of goosegrass reported in Virginia it is necessary to research other options for control of goosegrass in the preemergence stage.

Indaziflam is an option for preemergence control of goosegrass. However, with the threat of movement in the soil and herbicide entering runoff, proper measures should be taken to ensure the application is safe to the environment. Future research should be conducted to determine the optimal growth stage to treat a goosegrass plant, to determine if all goosegrass populations have the same growth habits and if germination of all populations happens at the same time of year (Cox 2014).

Literature Cited

Beard JB (2005) Beard's Turfgrass Encyclopedia for Golf Courses, Grounds, Lawns, Sports Fields. 192 p

Breeden G and Brosnan JT (2009) W170-Goosegrass (*Eleusine indica*). Knoxville, TN: The University of Tennessee Agricultural Extension Service. 5 p

Brosnan JT, Nishimoto RK, and DeFrank J (2008) Metribuzin-resistant goosegrass (*Eleusine indica*) in bermudagrass turf. Weed Technol 22: 675-678

Bryson CT and DeFelice MS, ed (2009) Weeds of the South. Athens, GA:University of Georgia Press. 374 p

Busey P (1999) Goosegrass and golf don't mix. GCM September 63-66

Busey P (2001) Goosegrass most difficult weed in South Florida. Fla. Turf Digest 18: 24-27

Callahan LM (1986) Crabgrass and Goosegrass Control in a Bentgrass Green in the Transition Zone Agron Journal 78:625-628

Cox MC (2014) Characterizing oxadiazon resistance and improving postemergence control programs for goosegrass (Eleusine indica) in bermudagrass (Cynodon spp.). PhD Dissertation, Virginia Tech Polytechnic and State University. 125 pp

Dernoeden PH, Watschke TL, and Mathias JK (1984) Goosegrass (*Eleusine indica*) control in turf in the transition zone. Weed Sci 32:4-7

Duke SO, Lydon J, Becerril TD, and Sherman R (1991) Protoporphyrinogen Oxidase-Inhibiting Herbicides. Weed Sci. 39: 465-473

Engel FA (1959) The Effect of Temperature and Light on Germination of Seed of Goosegrass (*Eleusine indica*). Weed Sci. 7: 359-361

[EPA] Environmental Protection Agency-Office of Prevention, Pesticides, and Toxic Substances (2006) Reregistration Eligibility Decision for MSMA, DSMA, CAMA, and Cacodylic Acid. Washington, DC: U.S. Environmental Protection Agency. Accessed: August 2016

[EPA] Environmental Protection Agency (2013) Organic Arsenals. Washington DC: U.S. Environmental Protection Agency

http://www.epa.gov/oppsrrd1/reregistration/organic_arsenals_fs.html Accessed: June 2016

Fryer JD (1979) Key factors affecting important weed problems and their control. Proc EWRS Symp. Mainz, Germ: 13-23

Hawton D, and Drennan DSH (1980) Studies on the longevity and germination of seed of goosegrass (*Eleusine indica*) and *Crotalaria goreensis* Weed Res. 20: 217-223

Heap IM (1997) The occurrence of herbicide-resistant weeds worldwide. Pesticide Sci. 51: 235-243

Heap IM (2013) International Survey of Herbicide Resistant Weeds http://www.weedscience.org. Accessed July 15, 2016

Heap IM (2017) International Survey of Herbicide Resistant Weeds http://www.weedscience.org Accessed January 11, 2017

Holm LG, Plucknett DL, Pancho JV, and Herberger JP (1977) The World's Worst Weeds: Distribution and Biology. Honolulu: University of Hawaii Press. Pp 609

Johnson BSC (2008) Germination Ecology of Goosegrass (*Eleusine indica*): An Important Grass Weed of Rainfed Rice Weed Sci 56: 699-706

Kranz JH, Schmutterer, and Koch W (1977) Diseases, Pests, and Weeds in Tropical Crops New York: J. Wiley 585 p

McCarty LB (1991) Goosegrass (*Eleusine indica*) control in bermudagrass (*Cynodon spp.*) turf with diclofop. Weed Sci.: 255-261

McCullough P., De Barreda DG, and Raymer P (2012) Nicosulfuron use with foramsulfuron and sulfentrazone for late summer goosegrass (*Eleusine indica*) control in bermudagrass and seashore paspalum. Weed Technol. 26: 376-381

McElroy JS, Robinson D, Samples T, Sorochan J, and Breeden G (2007) Weed Management Recommendations for Professional Turfgrass Managers: Athletic Fields, Golf Courses, Commercial Lawns and Turfgrass Sod. Knoxville, TN: University of Tennessee Agricultural Extension Service. Pp 54

Murdoch CL and Ikeda D (1974) Goosegrass (*Eleusine indica*) control in bermudagrass turf with combinations of MSMA and s-triazines. Agron Journal 66: 712-714

Nishimoto RK and McCarty LB (1997) Fluctuating temperature and light influence seed germination of goosegrass (*Eleusine indica*). Weed Sci.: 426-429

Radosevich SR, Holt JS and Ghersa C (1997). Weed Ecology: Implications for Management:Wiley.Senseman SA (2007) Herbicide Handbook 9th edition. Champaign IL: Weed Sci: 212-213

Shoup DE, Al-Khatib K, and Peterson DE (2003). Common waterhemp (*Amaranthus rudis*) resistance to protoporphyrinogen oxidase-inhibiting herbicides. Weed Sci. 51: 145-150

Timmons F (1970) A history of weed control in the United States and Canada. Weed Sci.: 294-307

Webster, T.M. and R.L. Nichols. 2012. Changes in the prevalence of weed species in the major agronomic crops of the Southern United States: 1994/1995 to 2008/2009. Weed Sci. 60: 145-157

Wiecko, G. 2000. Sequential Herbicide Treatments for Goosegrass (*Eleusine indica*) Control in Bermudagrass (*Cynodon dactylon*) Turf 1. Weed Tech. 14: 686-691

Identification of Goosegrass Biotypes Resistant to Preemergence-Applied Oxadiazon

ABSTRACT

Goosegrass biotypes from golf courses in Richmond, VA (CCV) and New Bern, NC (RB) historically treated with oxadiazon were identified as resistant compared to susceptible standard (PBU) based on comparisons of oxadiazon applied preemergence at increasing rates (0.03 to 2.24 kg ha^{-1}). Oxadiazon at rates $\leq$ 2.24 kg ha^{-1} rate did not prevent emergence of suspected resistant CCV and RB seedlings. PBU emergence was completely prevented at 0.14 kg ha^{-1}. Based on percent seedling emergence relative to non-treated and percent above-ground biomass reduction relative to non-treated, the oxadiazon rate at which emergence would be reduced 50% (I_{50}) or 90% (I_{90}) ranged from 0.12 to 0.18 kg ha^{-1} or 10.83 to 85.57 kg ha^{-1}, respectively for suspected resistant CCV and RB, compared to 0.03 to 0.4 kg ha^{-1} or 0.12 to 0.19 kg ha^{-1}, respectively for susceptible standard PBU. Seedling emergence data predicted 7.9 and 3.0 times greater I_{90} values for CCV and RB, respectively compared to biomass data. All three biotypes were completely controlled by preemergence applied labeled rates of prodiamine and indaziflam. This is the first peer-reviewed report of evolved weed resistance to oxadiazon.

Nomenclature: oxadiazon; goosegrass, *Eleusine indica* (L.) Gaertn. ELEIN

Key words: Turfgrass, herbicide resistance, weed evolution

INTRODUCTION

Oxadiazon is a protoporphyrinogen oxidase (PPO) inhibiting herbicide in the oxadiazole chemical family (Duke et al. 1997; Senseman 2007). Oxadiazon is utilized for control of annual grasses such as goosegrass (*Eleusine indica* (L.) Gaertn.) and crabgrass (*Digitaria* spp Heist. Ex Fabr.), and also broadleaves such as carpetweed (*Mollugo verticillata* L.) and Florida pusley (*Richardia scabra* L.) (Derr 1994; Johnson 1976). Due to the possibility of oxadiazon-induced turfgrass phytotoxicity, oxadiazon is typically applied either on a granular carrier to dry green turfgrass or as a liquid foliar application to dormant turfgrass (Kelly and Coates 1999). As a PPO-inhibiting herbicide, oxadiazon does not inhibit turfgrass root development similar to that of mitotic-inhibiting dinitroaniline herbicides or dithiopyr (McCarty et al. 1995). Oxadiazon can even be utilized during sprigging or stolonizing establishment of bermudagrass (*Cynodon* spp. Rich.) due to root development safety (Bingham and Hall III 1985; Bingham and Shaver 1981). Oxadiazon is generally perceived more effective in controlling goosegrass compared to dinitroaniline herbicides which is exacerbated by the fact that dinitroaniline resistant goosegrass has been reported (Johnson 1976, Bingham and Shaver 1981, Dernoeden et al. 1984, Mudge et al. 1984).

Goosegrass has C4 carboxylation and is typically considered to be an annual, but can perennialize in tropical environments (Buker et al. 2002). Goosegrass has a white to almost silver appearance at the center of the plant, giving rise to the alternative common name “silver crabgrass” (Holm et al. 1977). Goosegrass is considered one of the five worst weeds in the world and is commonly found growing in areas of high traffic with soil compaction or in areas of poor drainage (Holm et al. 1977, McCarty 1991). On athletic fields, sidelines goal mouths, and between hash marks are common areas that goosegrass can be found infesting. On golf courses,

areas around cart paths, tee boxes and putting greens are common places goosegrass infestations can occur. Not only is goosegrass unsightly, it can cause a putting green to be uneven and compete with the desirable turf stand. Furthermore, goosegrass presents a problem because of a lack of control options and resistance threats. Goosegrass ranks fourth out of all the herbicide resistant weeds with a total number of sites of action with seven and a total of 45 cases reported (Heap 2017).

Herbicides that provide selective goosegrass control are limited. When bermudagrass (*Cynodon* spp.) turf is considered, the number of safe yet effective herbicides is fewer. For example, dinitroanilines, dithiopyr, indaziflam, foramsulfuron, metribuzin, and oxadiazon can be used to control goosegrass in bermudagrass turf, however each presents specific limitations. Dinitroanilines and dithiopyr are generally viewed as inferior for goosegrass control compared to oxadiazon and indaziflam (Mudge et al. 1984, McCullough et al. 2013). Indaziflam, however, can injure bermudagrass on high sand soils such as constructed rootzones and can hamper bermudagrass development (Brosnan et al. 2014, Jones et al. 2013). Foramsulfuron can control goosegrass with acceptable bermudagrass safety, however multiple applications are normally required (Busey 2004). Metribuzin can control goosegrass postemergence but unacceptable bermudagrass injury can occur and resistance has been reported (Brosnan et al. 2008).

As of early 2017, The International Survey of Herbicide Resistant Weeds reports 22 resistance cases to PPO inhibitors (Heap 2017). Further, despite almost three decades of PPO-inhibiting herbicide usage (beginning in the late 1980's), the first reported of resistance did not occur until 2001; and 13 of the 22 cases occurring since 2011 (Heap 2017; Note: Not all cases presented in scientific literature have been reported to the International Survey of Herbicide Resistant Weeds). A biotype of common waterhemp (*Amaranthus rudis* J.D. Sauer) in Kansas

treated for consecutive years with acifluorfen was identified with 4 to 34 times more resistant to acifluorofen, lactofen, fomesafen, and sulfentrazone than non-resistant biotypes (Shoup et al. 2003). Researchers in Brazil have identified cross-resistance to PPO- and ALS-inhibiting herbicides in populations of wild poinsettia (*Euphorbia heterophylla* L.; Trezzi et al 2005). A separate population of common waterhemp collected in Illinois with confirmed resistance to PPO, acetolactate synthase, and photosystem II inhibiting herbicides was determined to be caused by a third encoded protox encoding gene, PP2XL, that contained a glycine deletion at position 210 (Patzoldt et al. 2006). Normally, PPO is encoded by two separate nuclear genes in plants, PPX1 and PPX2 (Patzoldt et al. 2006; Lee et al. 2008). PPX1 is a nuclear encoded plastid utilized form, whereas PPX2 is nuclear encoded mitochondrial utilized (Lee et al. 2008). It was predicted that common waterhemp plastid and mitochondrial PPO is encoded by a single gene—PPX2L (Patzoldt et al. 2006). To date, no weed species have evolved resistance in any turfgrass management system to PPO-inhibiting herbicides such as oxadiazon nor has any weed species evolved resistance to oxadiazon.

Goosegrass plants uncontrolled by oxadiazon treatment were collected in 2014 from golf course fairways in Virginia and North Carolina. Both facilities had greater than 10 years of continuous oxadiazon use. Objectives of this study were to 1) determine if the suspected-resistant goosegrass biotypes are resistant to oxadiazon, and 2) evaluate oxadiazon rate response to develop rate response models.

MATERIALS AND METHODS

Greenhouse experiments were conducted from January 2015 to June 2015 to determine effectiveness of oxadiazon applied preemergence on suspected oxadiazon-resistant goosegrass biotypes. Research was conducted at Auburn University Weed Science Greenhouse in Auburn, Alabama. Suspected resistant goosegrass plants were collected from golf course fairways at the Country Club of Virginia, Richmond, Virginia (CCV) and River Bend Golf and Country Club, New Bern, NC (RB). A biotype collected from Alabama Agricultural Experiment Station, Plant Breeding Unit, Tallassee, AL (PBU) was included as a susceptible standard with no known history of exposure to PPO-inhibiting herbicides. All the biotypes were propagated separately in a greenhouse environment to increase seed lots for experiments. Seed was air dried and stored at 4 C until needed. Plastic pots (10 cm diameter) were filled with the surface horizon of Marvyn Sandy Loam (fine-loamy, kaolinitic, thermic Typic Kanhapludults) with a pH of 6.5 and 1.1% organic matter. Twenty seeds were planted per pot at a 0.5-cm depth per pot. Plants were overhead irrigated three times daily (~0.2 cm each irrigation).

Treatments included oxadiazon (Ronstar FLO, Bayer Environmental Sciences, Research Triangle Park, NC 27709) applied at rates of 0.035, 0.07, 0.14, 0.28, 0.56, 1.12, and 2.24 kg ha^{-1}. Additional comparison treatments included indaziflam (Specticle FLO, Bayer Environmental Sciences, Research Triangle Park, NC 27709) at 0.03 kg ha^{-1}; and prodiamine (Barricade, Syngenta Crop Protection, LLC, Greensboro, NC 27419-8300) at 0.56 kg ha^{-1} and a non-treated check. Treatments were applied using an enclosed spray chamber at 280 L ha^{-1} with a single 8002E nozzle at 32 PSI (TeeJet Spray Systems Co., Wheaton, IL 60189). Treatments were applied preemergence approximately one-hour after seeding each pot. Pots were lightly mist irrigated prior to and immediately after herbicide applications. Normal irrigation was resumed

24-h after application. Experiments were conducted as a completely random design, replicated three times, and were repeated in time. Pots were randomized weekly to account for unexplained discrepancies in the greenhouse environment. Data collected were number of emerged seedlings per pot at 42 days after treatment (DAT) and above-ground biomass collected 42 DAT. To determine above ground biomass, shoots were clipped at the soil surface and fresh weights recorded. At the time of harvest, no plants were observed to be producing seedheads thus the weights can be considered foliar weight only.

Data were subjected to ANOVA using the PROC GLM procedure using SAS 9.4 (SAS Institute Inc., Cary, NC). Fisher's Protected LSD (P = 0.05) was used to compare the labeled rate of oxadiazon (1.12 kg ha^{-1}) to industry standard treated checks and non-treated. To facilitate regression analysis, oxadiazon rates were log-transformed to create equal spacing between treatments. The non-treated treatment (0 kg ha^{-1}) was transformed to -1.76 to maintain the equal spacing among log treatment rates. Seedling emergence counts were converted to percent reduction relative to counts in non-treated pots and above-ground biomass weight was converted to percent above-ground biomass reduction relative to the weight in non-treated pots. The non-treated mean for seedling emergence and above-ground biomass was used for conversion calculations to relative measures. Percent reduction relative to non-treated data were used for regression model fitting rather than count or biomass data due to differences between biotypes with respect to germination in non-treated. Data were fitted to a linear or exponential decay model using Sigma Plot 10.2 (Systat Software Inc., London, UK) using an exponential decay function:

$$y = a * e^{-b*x} \quad [1]$$

where y represents goosegrass seedling biotype reduction (%), x represents log-transformed oxadiazon rate (kg ha^{-1}), and b represents the y-intercept. Regression equations were used to calculate inhibition values at 50 and 90% relative to non-treated of each biotype, commonly referred to as I_{50} and I_{90} values, respectively.

RESULTS AND DISCUSSION

Experimental run by herbicide treatment was not significant ($P > 0.05$) for both seedling emergence and above-ground biomass; thus, these data were pooled over experimental run. A herbicide treatment by goosegrass ecotype interaction was observed for seedling emergence and above-ground biomass ($P < 0.05$).

Comparison of registered control options. Results presented will focus on ecotype comparison of oxadiazon, prodiamine, and indaziflam labeled rate response and regression analysis of oxadiazon rates. Indaziflam at 0.03 kg ha^{-1}, prodiamine at 0.56 kg ha^{-1}, and oxadiazon at 1.12 kg ha^{-1} completely prevented seedling emergence of the susceptible goosegrass biotype PBU (Table 1) resulting in no biomass accumulation. Indaziflam and prodiamine applied at 0.03 kg ha^{-1} and 0.56 kg ha^{-1}, respectively completely prevented seedling emergence of suspected resistant biotypes CCV and RB resulting in no above-ground biomass accumulation 42 DAT. Oxadiazon at 1.12 kg ha^{-1} allowed six to nine suspected resistant seedlings of CCV or RB to emerge resulting in 0.5 or 0.4 g biomass accumulation per pot, respectively, 42 DAT. While this is the first report of oxadiazon resistant goosegrass in peer-reviewed literature, it is not the first reported in non-peer reviewed sources (Cox, 2014; Askew et al., 2013). Cox (2014) and Askew et al. (2013) reported reduced control of a goosegrass biotype treated with labeled rates of oxadiazon, however indaziflam (0.035 or 0.062 kg ha^{-1}), prodiamine (0.84 kg ha^{-1}), and dithiopyr (0.56 kg ha^{-1}) resulted in acceptable control.

Response to oxadiazon rate progression. Oxadiazon at rates $\geq$ 0.14 kg ha^{-1} completely prevented seedling emergence of PBU seedlings resulting in no above-ground biomass except for unexplainable minor emergence that occurred at 0.56 kg ha^{-1} (Figure 1). No oxadiazon rate completely prevented either emergence or biomass of CCV or RB, i.e. the two suspected resistant biotypes. Increasing rates of oxadiazon decreased emergence and biomass, but little to no difference was observed for rates from 0.28 to 2.24 kg ha^{-1}. In non-treated pots, susceptible goosegrass PBU had lower emergence of ~8 seedlings per pot in non-treated compared to 16-18 seedlings per pot in non-treated for suspected resistant CCV and RB goosegrass. To overcome this discrepancy in non-treated germination, data were adjusted to percent relative to the non-treated within each biotype prior to regression analysis.

Percent emergence relative to the non-treated and percent above-ground biomass response to oxadiazon rate was modeled for all biotypes using linear or exponential decay functions to allow for calculation of I_{50} and I_{90} values (Table 2). Prior to regression analysis, oxadiazon rates were converted to log-scale to create equal spacing among oxadiazon rates (Figure 2). Suspected oxadiazon-resistant CCV and RB emergence and biomass response to increasing rates of oxadiazon were modeled using a linear function. Susceptible PBU emergence and biomass response to increasing rates of oxadiazon were modeled using exponential decay functions. R^2 values for all models were from 0.81 to 0.97.

I_{50} and I_{90} values were calculated based on the developed models (Table 3). Oxadiazon I_{50} and I_{90} values for PBU based on seedling emergence relative to the non-treated were 0.04 and 0.19, respectively. Oxadiazon I_{50} and I_{90} values for PBU based on above-ground biomass relative to the non-treated were 0.03 and 0.12 kg ha^{-1}, respectively. Suspected-resistant CCV and RB were calculated to have higher I_{50} and I_{90} values of 0.12 to 0.18 and 10.83 to 85.57 kg ha^{-1},

respectively, based on emergence and biomass for both biotypes. Seedling emergence data predicted 7.9 and 3.0 times greater I_{90} values for CCV and RB, respectively compared to biomass data. One explanation for this discrepancy based on data and observations is while CCV and RB emergence occurred as oxadiazon rate increased, plant size and concomitant weight were decreased compared to the non-treated and lower rates. Regardless of possible differences among models, predicted I_{90} values for CCV and RB are greater than the standard labeled rate of 1.12 kg ha^{-1}. Further, such predicted values exceed the upper tested limit and therefore should be taken as theoretical.

Research Implications. Resistance to PPO-inhibiting herbicides is relatively rare. The International Survey of Herbicide Resistant Weed currently reports 10 species resistant to PPO inhibiting herbicides, compared to 48 species for ACCase inhibiting herbicides, 159 species for ALS inhibiting herbicides, and 74 species for photosystem II inhibiting herbicides (Heap 2017). Further, nine of the species reported resistant to PPO-inhibiting herbicides are eudicots and only one is a monocot- wild oat (*Avena fatua* L.). The lack of development of PPO-inhibiting herbicide resistant weeds is theorized to be due to a polygenic site of action compared to a monogenic site of action for ACCase, ALS, and PSII inhibiting herbicides (Patzoldt et al. 2006). Herbicides that target monogenic sites of action often result in target-site resistance caused by single nucleotide changes (Powles and Yu 2010). Such changes generally yield complete resistance with no rate inducing herbicidal activity. Polygenic target site of action resistance, or resistance to herbicides that act on more than one enzyme, would conceivably require changes in all target sites (Patzoldt et al. 2006). Common waterhemp has avoided the necessity of dual mutation through duplication of the PPX2 to PPX2L which contains a codon deletion and is also utilized in both mitochondria and plastid following translation (Patzoldt et al. 2006). PPO-

resistant common ragweed (*Ambrosia artemisiifolia* L.) was discovered to contain a single nucleotide polymorphism in only one of the two PPO target sites, PPX2 (Rousonelos et al. 2012).

We have yet to decipher the resistant mechanism evolved by CCV and RB goosegrass. The observed resistance of RB and CCV goosegrass could be described as incomplete (recessive) resistance that does not completely eliminate herbicidal activity thus resulting in the death of some seedlings within a population. Similar incomplete PPO-inhibiting herbicide resistance response has been observed with tall waterhamp (*Amaranthus tuberculatus* (Moq.) J.D. Sauer, Shoup et al. 2003). It is also possible that the resistance mechanism is still undergoing segregation within each population yielding the decline in germination with increasing herbicide rates. We propose these as hypotheses for future research.

Literature Cited

Askew SD, Cox M, Spak DR (2013) A suspected oxadiazon-resistant goosegrass population in Virginia. Proceedings, South. Weed Sci. Soc. 66:229

Bingham SW, Hall III JR (1985) Effects of herbicides on bermudagrass (*Cynodon* spp.) sprig establishment. Weed Sci. 33:253-257

Bingham SW, Shaver RL (1981) Goosegrass (*Eleusine indica*) control during bermudagrass (*Cynodon dactylon*) establishment. Weed Sci. 29:11-16

Brosnan JT, Breeden GK, Thoms AW, Sorochan JC (2014) Effects of preemergence herbicides on the establishment rate and tensile strength of hybrid bermudagrass sod. Weed Technol. 28:206-212

Brosnan JT, Nishimoto RK, DeFrank J (2008) Metribuzin-resistant goosegrass (*Eleusine indica*) in bermudagrass turf. Weed Technol. 22: 675-678

Buker RS, Steed ST, Stall WM (2002) Confirmation and control of a paraquat-tolerant goosegrass (*Eleusine indica*) biotype. Weed Technol. Pp. 309-313

Busey P (2004) Goosegrass (*Eleusine indica*) control with foramsulfuron in bermudagrass (*Cynodon* spp.) turf. Weed Technol. 18:634-640

Cox MC (2014) Characterizing oxadiazon resistance and improving postemergence control programs for goosegrass (Eleusine indica) in bermudagrass (Cynodon spp.). PhD Dissertation, Virginia Tech Polytechnic and State University. 125 p

Dernoeden PH, Watschke TL, Mathias JK (1984) Goosegrass (*Eleusine indica*) control in turf in the transition zone. Weed Sci. 32:4-7

Derr JF. (1994) Weed control in container-grown herbaceous perennials. Hortsci. 29:95-97

Duke SO, Lee HJ, Duke MV, Reddy KN, Sherman TD, Becerril JM, Nandihalli UB, Matsumoto H, Jacobs NJ, Jacobs JM. (1997) Mechanisms of resistance to protoporphyrinogen oxidase-inhibiting herbicides. Pp. 155-160. Published in Weed and crop resistance to herbicides. De Prado R, Jorrin J, and Garcia-Torres L (eds). Kluwer Academic Publishers: Dordrecht, Netherlands

Heap I (2017) International Survey of Herbicide Resistant Weeds. http://www.weedscience.org [Last confirmed: 13 February 2017]

Holm LG, Plucknett DL, Pancho JV, Herberger JP (1977) The world's worst weeds. University Press: Honolulu, Hawaii. 609 p

Johnson BJ (1976) Dates of herbicide application for summer weed control in turf. Weed Sci. 24:422-424

Jones PA, Brosnan JT, Kopsell DA, Breeden GK (2013) Effect of reed-sedge peat moss on hybrid bermudagrass injury with indaziflam and prodiamine in sand-based root zones. Weed Technol. 27:547-551

Kelly ST, Coats GE (1999) Influence of fertilizer particle size on efficacy of dithiopyr, prodiamine, oryzalin, and oxadiazon for southern crabgrass (*Digitaria ciliaris*) control. Weed Technol. 13:385-389.

Lee RM, AG Hager, PJ Tranel (2008) Prevalence of a novel resistance mechanism to PPO-inhibiting herbicides in waterhemp (*Amaranthus tuberculatus*). Weed Sci. 56:371-375

McCarty LB (1991) Goosegrass (*Eleusine indica*) control in bermudagrass (*Cynodon* spp.) turf with diclofop. Weed Sci. 39:255-261

McCarty LB, Porter DW, Colvin DL (1995) Sod regrowth of St. Augustinegrass after preemergence herbicide applications. Agron. J. 87:503-507

McCullough PE, Yu J, de Barreda DG (2013) Efficacy of preemergence herbicides for controlling a dinitroaniline-resistant goosegrass (*Eleusine indica*) in Georgia. Weed Technol. 27:639-644

Mudge LC, Gossett BJ, Murphy TR (1984) Resistance of goosegrass (*Eleusine indica*) to dinitroaniline herbicides. Weed Sci. 32:591-594

Patzoldt WL, Hager AG, McCormick JS, Tranel PJ (2006) A codon deletion confers resistance to herbicides inhibiting protoporphyrinogen oxidase. PNAS 103:12329-12334

Powles SB, Yu Q (2010) Evolution in action: Plants resistant to herbicides. Ann. Rev. Plant Biol. 61:317-347

Rousonelos SL, Lee RM, Moreira MS, VanGessel MJ, Tranel PJ (2012) Characterization of a common ragweed (*Ambrosia artemisiifolia*) population resistant to ALS- and PPO-inhibiting herbicides. Weed Sci. 60:335-344

Senseman SA (2007) Herbicide Handbook. 9th ed. Champaign, IL: Weed Science Soc.: Pp. 212-213

Shoup DE, Al-Katib K, Peterson DE. (2003) Common waterhemp (*Amaranthus rudis*) resistance to protoporphyrinogen oxidase-inhibiting herbicides. Weed Sci. 51:145-150

Trezzi MM, Felippi CL, Mattei D, Silva HL, Nunes AL, Debastiani, Vidal RA, Marques A (2005) Multiple resistance of acetolactate synthase and protoporphyrinogen oxidase inhibitors in *Euphorbia heterophylla* biotypes. J. Environ. Sci. Health, Part B 40:101-109.

Figure Legends

Figure 2.1. Seedling emergence (A) and above-ground biomass (B) per pot response of four goosegrass biotypes to oxadiazon at increasing rates. Oxadiazon rates have not been log transformed to create equal spacing among treatments. Vertical bars represent standard errors (P = 0.05).

Figure 2.2. Seedling emergence response (A) and above-ground biomass response (B) of three goosegrass species 42 DAT with increasing rates of oxadiazon. Response was modeled based on the log rate of oxadiazon to create equal spacing between rates using either exponential decay or linear regression analysis of emergence number or weight (g) per pot relative to the non-treated. Non-log transformed oxadiazon rates are presented for reference. Vertical bars represent standard errors (P = 0.05).

Figure 2.1.

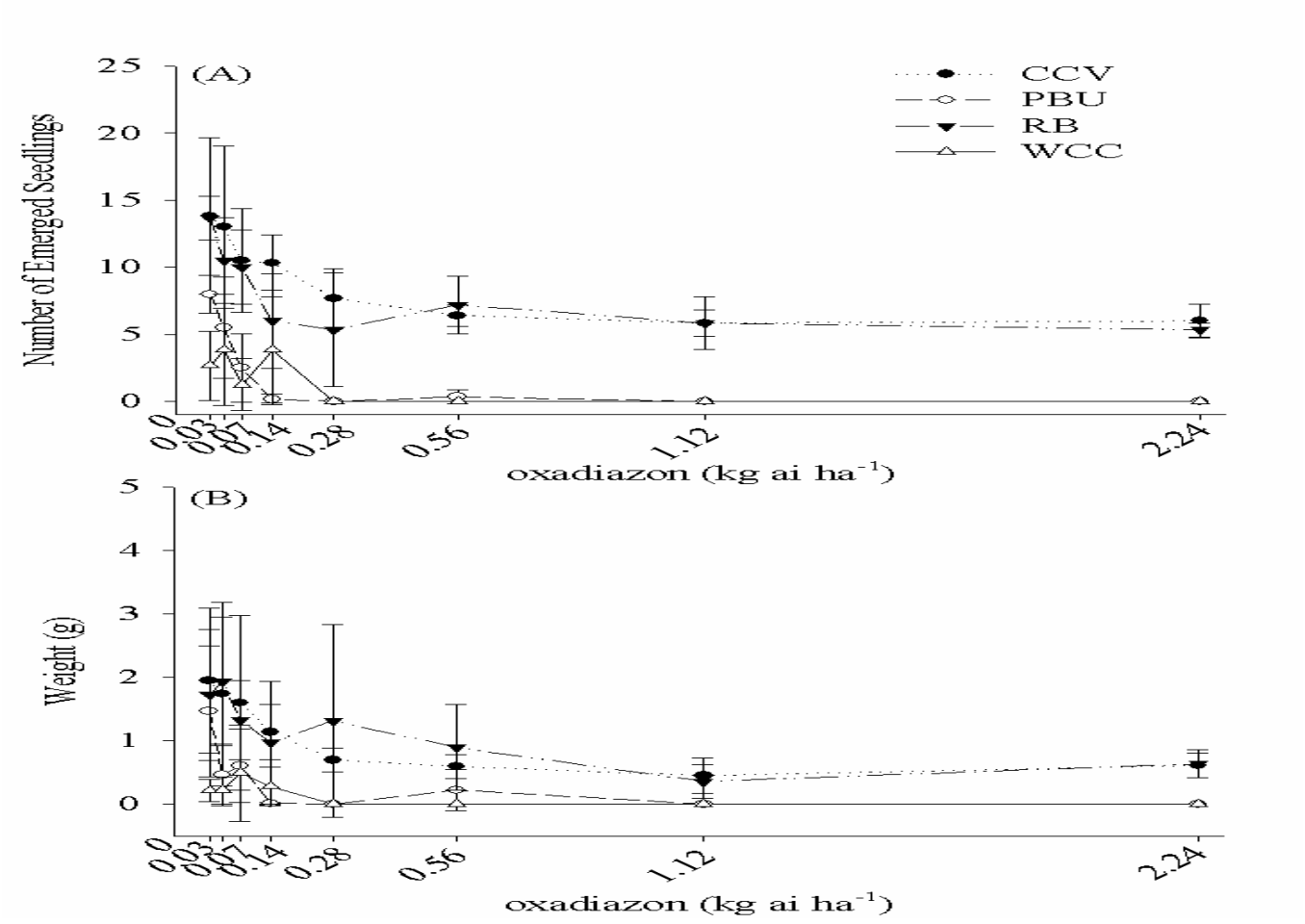
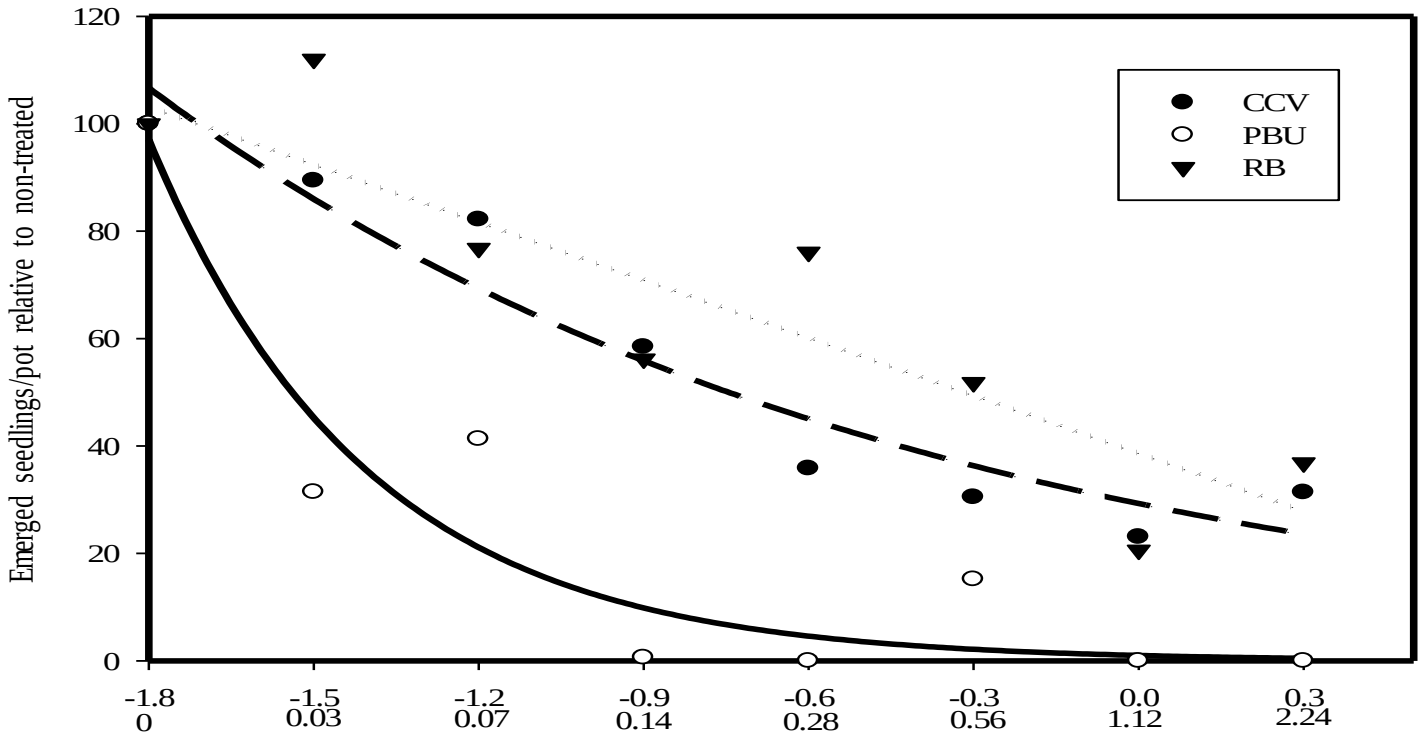

(A)
CCV
PBU
RB
WCC
Number of Emerged Seedlings
25
20
15
10
5
0
0
0.03
0.07
0.14
0.28
0.56
1.12
2.24
oxadiazon (kg ai ha^{-1})
(B)
Weight (g)
5
4
3
2
1
0
0
0.03
0.07
0.14
0.28
0.56
1.12
2.24
oxadiazon (kg ai ha^{-1})

Figure 2.2.

A)


Emerged seedlings/pot relative to non-treated
120
100
80
60
40
20
0
CCV
PBU
RB
-1.8
0
-1.5
0.03
-1.2
0.07
-0.9
0.14
-0.6
0.28
-0.3
0.56
0.0
1.12
0.3
2.24
oxadiazon rate; top number=log of rate
bottom number= kg ai ha-1

B)

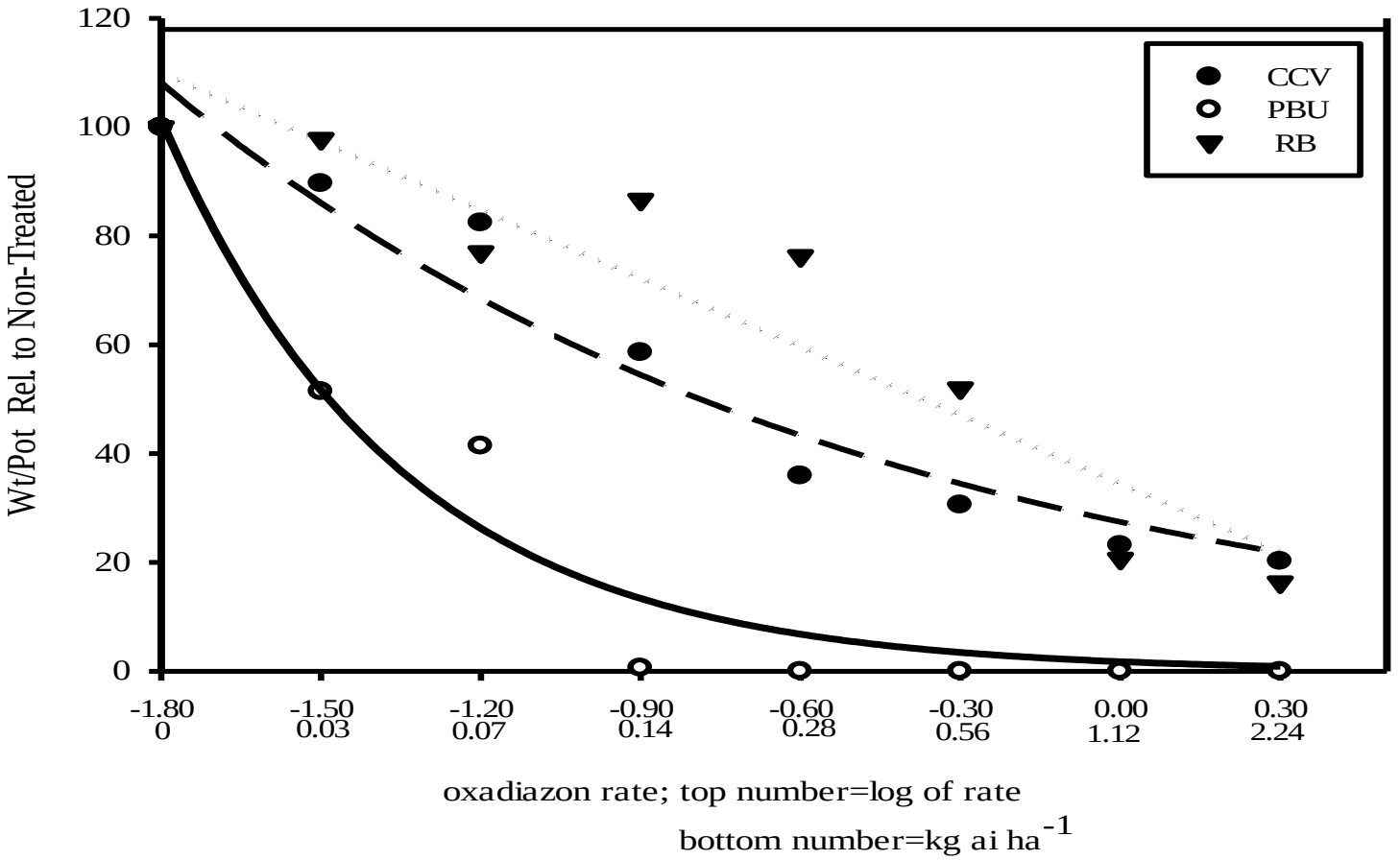

120
100
80
60
40
20
0
Wt/Pot Rel. to Non-Treated
CCV
PBU
RB
-1.80
0
-1.50
0.03
-1.20
0.07
-0.90
0.14
-0.60
0.28
-0.30
0.56
0.00
1.12
0.30
2.24
oxadiazon rate; top number=log of rate
bottom number=kg ai ha-1

Table 2.1. Goosegrass seedling emergence (top) and above-ground biomass (bottom) in response to registered-rate, preemergence-applied treatments with different modes of action. Greenhouse experiment; data collected 42 DAT.[a]

Treatment		Seedling emergence		
Herbicide		CCV	RB	PBU
	kg ha^{-1}	———number seedlings/pot———		
Non-treated	-	18.2	16.0	8.0
Indaziflam	0.03	0	0	0
Oxadiazon	1.12	5.8	5.9	0
Prodiamine	0.56	0	0	0
LSD (0.05)[b]		2.7	1.3	2.6
		Above-ground biomass		
		——————g/pot——————		
Non-treated	-	2.3	2.3	1.5
Indaziflam	0.03	0	0	0
Oxadiazon	1.12	0.5	0.4	0
Prodiamine	0.56	0	0	0
LSD (0.05)[b]		0.3	0.3	1.6

[a] Goosegrass biotype abbreviations: CCV, Country Club of Virginia, Richmond, VA, suspected resistant; RB, River Bend Golf Course, New Bern, NC, suspected resistant; PBU, Plant Breeding Unit, Tallassee, AL, known susceptible.

[b] LSD ($P<0.05$) value for comparison of means within a common biotype.

Table 2.2. Predictive model with exponential decay and linear regression equations and R^2 values for number of emerged seedlings and fresh weight[1] for increasing oxadiazon rate.

Goosegrass biotype[a]	Seedling Rating	Equation[b]	R^2
CCV	Emerged Seedlings	$y=31.88exp^{-0.60x}$	0.94
	Above-ground biomass	$y=23.36exp^{-0.82x}$	0.96
RB	Emerged Seedlings	$y=30.92exp^{-0.59x}$	0.81
	Above-ground biomass	$y=27.72exp^{-0.71x}$	0.90
PBU	Emerged Seedlings	$y= 2.03exp^{-2.24x}$	0.97
	Above-ground biomass	$y=0.79exp^{-2.72x}$	0.86

[a] Goosegrass biotype abbreviations: CCV, Country Club of Virginia, Richmond, VA, suspected resistant; RB, River Bend Golf Course, New Bern, NC, suspected resistant; PBU, Plant Breeding Unit, Tallassee, AL, known susceptible.

[b] Where x being log rate of oxadiazon, and y being the response variable of percent emerged seedlings or percent above-ground biomass relative to the non-treated.

Table 2.3. Estimated oxadiazon rate required to reduce goosegrass biotype by 50% (I_{50}) and 90% (I_{90}) based on number of emerged seedlings and above-ground biomass harvested 42 DAT.

Goosegrass biotype[a]	Emerged seedlings		Above-ground biomass	
	I_{50}	I_{90}	I_{50}	I_{90}
	——————————kg ha^{-1}——————————			
CCV	0.18	85.57	0.12	10.83
RB	0.15	81.89	0.15	27.2
PBU	0.04	0.19	0.03	0.12

[a] I_{50} and I_{90} values were calculated based on linear to exponential decay models presented in Table 2.2.

[b] Abbreviations: CCV, Country Club of Virginia; RB, River Bend Golf Course; PBU, Plant Breeding Unit.

Table 2.4. Predictive model with exponential decay equations for percent of emerged seedlings and fresh weight relative to the non-treated control response to increasing oxadiazon rate. Parameter estimate and parameter estimate 95% confidence intervals (CI) are presented as a means of model comparision.

Goosegrass population[a]	Rating	Equation	R2
		$y=a*exp^{-bx}$	
CCV	Emerged Seedlings	y=31.88exp-0.60x	0.94
	Above-ground biomass	y=23.36exp-0.82x	0.96
RB	Emerged Seedlings	y=30.92exp-0.59x	0.81
	Above-ground biomass	y=27.72exp-0.71x	0.9
PBU	Emerged Seedlings	y=2.03exp-2.04x	0.97

[a] Goosegrass population abbreviations: (CCV) Country Club of Virginia, (RB) River Bend Golf Course, North Carolina, and (PBU) Plant Breeding Unit, Alabama.

Table 2.5. Estimated oxadiazon rate required to reduce goosegrass biotype by 50% (I50) and 90% (I90) based on number of emerged seedlings and above-ground biomass harvested 42 DAT. 95% confidence intervals (CI) at I50 and I90 values are provided as a means of comparison[a].

Goosegrass biotype[b]	Emerged seedlings		Above-ground biomass	
	I50	I90	I50	I90
	kg ha-1 (95% CI)			
CCV	0.18 (0.02, 0.38	85.57 (62.95, 117.17)	0.12 (0.02, 0.28)	10.83 (7.69, 11.98)
RB	0.15 (0.00, 0.59)	81.89 (48.77, 130.92)	0.15 (0.02, 0.42)	27.2 (14.18, 31.99)
PBU	0.04 (0.00, 0.13)	0.19 (0.00, 0.48)	0.03 (0.00, 0.16)	0.12 (0.00, 0.37)

[a] I50 and I90 values were calculated based on exponential decay functions.

[b] Abbreviations: CCV, Country Club of Virginia; RB, River Bend Golf Course; PBU, Plant Breeding Unit.

Response of Oxadiazon- Resistant Goosegrass to Postemergence Applications of Protoporphyrinogen Oxidase Inhibitors

ABSTRACT

Goosegrass (*Eleusine indica)* is a problematic weed in both warm and cool season turf. Postemergence control is difficult because of plant size and herbicide resistant populations. Incidences of goosegrass resistance to mitosis inhibitors, EPSP synthase inhibitors, photosystem I inhibitors, and ALS inhibitors have been reported. To prevent further incidences of resistance, alternative herbicidal control options need to be evaluated. Until recently, no PPO-resistance cases have been reported from grass species. Oxadiazon resistant goosegrass populations from Virginia have prompted additional studies with various PPO-inhibiting herbicides. Research was conducted to determine response of five goosegrass ecotypes ('Craft Farms', 'CCV', 'RB' 'Alabama Row Crop', and 'Texas') to postemergence-applied oxadiazon, flumioxazin, and sulfentrazone. CCV and RB were previously confirmed as resistant to preemergence-applied oxadiazon. A dose response curve was developed to predict at which rate a goosegrass population will be inhibited. The rate at which a given herbicide inhibits growth 50 or 90%, also referred to as I_{50} and I_{90} values are commonly presented in dose response research. Estimated *i* values from susceptible (S) seed were several orders of magnitude higher than *i* values from oxadiazon resistant (OR) plants. The OR populations in Auburn University trials required an estimated 103.53 and 5922 kg ai $ha^{-1,}$ respectively, of the population at 42 DAT while the Craft Farms (S) plants required only 2.83 and 5.64 kg ai ha^{-1}, respectively. These data indicate that oxadiazon rates required to achieve commercially acceptable control (90%) were significantly higher for the OR than the S successions.

Nomenclature: PPO inhibiting herbicides; goosegrass, *Eleusine indica* (L.) Gaertn. ELEIN

Key words: Turfgrass, herbicide resistance, oxadiazon

INTRODUCTION

Goosegrass is considered one of the five most troublesome weeds in the world (Holm 1991). Goosegrass is an issue in lawns, golf courses, row crop environments and sports fields, however, goosegrass tends to be more problematic on golf course greens and tee boxes where it is rare for a pre-emergence herbicide to be used because of reseeding. (Leibhart 2014). Goosegrass has a rough leaf texture, unsightly seedheads, and minimizes the quality and aesthetics of turfgrass (McCullough et al. 2012). Goosegrass has a prostrate growth habit, ability to produce thousands of seeds, and tolerate mowing at heights less than 0.25 cm (McCarty 1991). In turfgrass, goosegrass can fill worn or bare areas rapidly during peak germination periods in early summer (Bingham and Shaver 1981), especially areas that are subject to heavy traffic, wear, and compaction (Wiecko 2000).

Dinitroaniline herbicides, such as prodiamine, are widely used for preemergence weed control in turf. These herbicides attach to tubulin, a microtubule protein, resulting in a loss of microtubule function for spindle apparatus formation during mitosis (Parka and Soper 1977). Thus, cell division is stopped and the weed fails to germinate. The dinitroaniline herbicides are widely used in turfgrass. However, years of continued use has led to evolution of resistant goosegrass populations. Since the 1970s, a dinitroaniline resistant goosegrass population has been suspected in a cotton field in Marlboro county, South Carolina (Mudge 1984). Trifluralin had been used repeatedly on turf for 10 years and control began to become ineffective. Developing resistance issues with the dinitroaniline herbicides, alternative modes of action such as the PPO inhibitors had to be offered to turf managers for goosegrass control (McCullough et al. 2012).

Oxadiazon is a selective pre-emergent herbicide in the oxadiazole chemical family. Oxadiazon is registered for use on select turfgrasses for goosegrass control. Oxadiazon has a field half-life of sixty days, which makes it persistent and long lasting in the soil, providing season long control (Beard 2005). Oxadiazon does not hinder root establishment and can serve as an alternative for the dinitroaniline herbicides (Ferrell et al. 2003). Bingham and Shaver (1981) conducted preemergence herbicide research on an area with variable bermudagrass density. Their findings suggested that oxadiazon controlled goosegrass acceptably in areas of low or high bermudagrass density, while pendimethalin results varied.

Sulfentrazone, a triazolinone herbicide, is labeled for postemergence goosegrass control. Past research stated that a single application of sulfentrazone applied alone achieved less than 35% control of goosegrass. With the addition of nicosulfuron, which is not labeled for use on turfgrass, improved the control to around 60%. Sequential applications of sulfentrazone alone improved the control to 65%, but sequential applications with the addition of nicosulfuron improved goosegrass control to 84% at 9 weeks after treatment (WAT)(McCullough et al. 2012).

Flumioxazin inhibits protoporphyrinogen oxidase enzyme and can be used for pre and postemergence weed control (Senseman 2007). However, flumioxazin is not registered for application on actively growing bermudagrass. Flumioxazin applied at 6 rates between 0.03 and 0.25 resulted in bermudagrass injury between 25 and 45%. At 2 WAT, bermudagrass had recovered and injury was no more than 10% (Askew and Beam 2002). No known research has been conducted for post-emergence goosegrass control at reduced rates.Past research confirms that two goosegrass ecotypes collected from Virginia (CCV and RB) are resistant to oxadiazon (Askew 2013).

The objectives of this study were to evaluate: a) the effectiveness of PPO inhibitors for post emergence goosegrass control (b) to rapidly screen goosegrass plants, or retrieve a plant from the field, to quickly test for resistance and to determine if the oxadiazon resistant ecotypes are resistant to postemergence flumioxazin and sulfentrazone. It would take 6 to 8 months to complete an assessment of oxadiazon resistance from preemergence applications. Obtaining ripened seed, harvesting and weighing emerged plants after application, harvest seed, and allow seed to ripen to repeat the process. Being able to repeat this process multiple times allows us to gain a conclusion about oxadiazon resistance rapidly.

MATERIALS AND METHODS

Greenhouse experiments were conducted at Auburn University at Auburn, Al from June 2015 to April 2016 to evaluate postemergence goosegrass control to increasing rates of three PPO-inhibiting herbicides. Five goosegrass populations were included (Table 3.1). The first two populations (CCV and RB) were endemic to golf courses and have been confirmed as PPO resistant (Askew, Cox, Spak et al. 2013). The next two populations (CF and TX) were also endemic to golf courses but not known to be PPO resistant. The fifth population (AL) was from a row crop environment and assumed to be PPO sensitive.

All of the populations were propagated separately in a greenhouse environment to increase seed lots for experiments. Collected seed was air dried and stored at 4 C until needed. Greenhouse temperature conditions were 30/25(± 2C) at day/ night temperature. Plastic pots (10 cm diameter) were filled with Marvyn sandy loam (fine-loamy, kaolinitic, thermic Typic Kanhapludults) soil with a pH of 6.5 and 0.5% humic matter.

Seeds were planted at a 2-cm depth in 28 x 20-cm flats and were hand watered as needed daily to ensure germination. Once the seedlings emerged they were separated and planted in individual 4-cm pots, one seedling per pot approximately 4 cm in height. The plants were irrigated three times daily for 2 min with overhead irrigation and treated 7 days after final potting. Plants were not irrigated 24 hours after treatment, then, normal irrigation was resumed with careful attention not to over-irrigate and cause leaching.

Herbicide treatments included oxadiazon (Ronstar FLO, Bayer Environmental Science, Research Triangle Park, NC 27709) applied at seven rates ranging from 0.14 to 8.96 kg ai ha^{-1}; sulfentrazone (Dismiss, FMC Corporation, 1735 Market Street, Philadelphia, PA 19103) applied

at seven rates ranging from 0.07 to 4.50 kg ai ha^{-1} and flumioxazin (Sureguard, Valent U.S.A. Corporation, P.O. Box 8025 Walnut Creek, CA 94596) applied at seven rates ranging from 0.08 to 5.70 kg ai ha^{-1}. A non-treated control was also included. Foliar applications were made with a CO_2- powered handheld spray boom calibrated to deliver 280 L/ha with three 8002 TeeJet flat fan nozzles.

Experiments were conducted as a randomized complete block design, with three replicates repeated in time. The pots were randomized weekly to account for unexplained discrepancies in the greenhouse environment.

Goosegrass control ratings were taken weekly beginning at 7 DAT, yielding 6 total rating. However, only the final rating at 42 DAT will be included in this study. Control ratings were based on 0 to 100 where 0= no control, >80% = acceptable control, and 100 = complete control. At 42 DAT plants were clipped at the soil surface and fresh weights were recorded.

Herbicide response data was also expressed as percent reduction relative to the appropriate non-treated control. This data as well as the plant weight data were subject to ANOVA at a significance level of P=0.05 using the PROC GLM procedure of SAS 9.4 (SAS Institute Inc., Cary, NC). Significant (P<0.05) main effects and interactions are presented per ANOVA with importance given to higher-order interactions in the factorial arrangement (Steele et al. 1997). Control based upon weight data were fitted to either an exponential decay, linear, or sigmoidal models using Sigma Plot 10.2 (Systat Software Inc, London, UK) based on best fit of each population. Regression equations for these three possible modes of action are as follows:

Linear: $y = y_{0+} ax$ [1]

Exponential decay: $y = a\, e^{-bx}$ [2]

Sigmoidal: $y= a1+e^{(-(x-x0)-1b)^{-1}}$ [3]

where *y* represents goosegrass seedling weight (%) and X represents oxadiazon rate (kg ai ha^{-1}). Estimated parameters, were subjected to ANOVA and were used to calculate I_{50} and I_{90} values. From the regression analysis: LD_{50} and LD_{90} values were calculated for each population-herbicide combination for both the weight data and the control based upon weight data.

RESULTS AND DISCUSSION

Per ANOVA, experimental run by rate was not significant ($P>0.05$); so, the data was pooled over experimental run. Significant interactions were observed between herbicide rate and population; warranting presentation of interactions over main effects. Results presented will focus on ecotype comparison of flumioxazin, oxadiazon, and sulfentrazone labeled rate postemergence response and regression analysis of increasing rates of herbicides listed above. Applications of flumioxazin and sulfentrazone (1.43 kg ha^{-1} and 1.12 kg ha^{-1}) provided greater postemergence goosegrass control of resistant populations when compared to oxadiazon at 1.12 kg ha^{-1}(Table 3.2 and 3.3). Oxadiazon averaged 15% control of resistant populations and 75% control of susceptible populations when applied at 1.12 kg ha^{-1}. Flumioxazin and sulfentrazone averaged 100% control across all populations in the study, indicating that oxadiazon resistant populations were susceptible to other PPO inhibiting herbicides used in the study.

A goal of this research was to rapidly screen confirmed oxadiazon resistant plants with postemergence applications of oxadiazon and to evaluate the effectiveness of two other PPO inhibiting herbicides on both oxadiazon resistant and susceptible goosegrass plants. A dose

response curve was developed to predict at which rate a goosegrass population will be inhibited. The rate at which a given herbicide inhibits growth 50 or 90%, also referred to as I_{50} and I_{90} values as described by Seefeldt (1995), are commonly presented in dose response research. Bartley et al. (1993) state that the selectivity and efficacy of herbicides can be analyzed by comparing their dose response curves with the main advantage being that we can predict and compare doses. Culpepper (2006) confirmed an herbicide resistant palmer amaranth population in Georgia, a log-logistic dose-response curve was used to determine an I_{50} and I_{90} prediction value. Flumioxazin, oxadiazon, and sulfentrazone rates were log-transformed to provide equal spacing among rates prior to model development. By using the curve that provided the best fit, it was possible to predict an I_{50} and I_{90} value for each goosegrass population.

Estimated i values from susceptible (S) seed were several orders of magnitude higher than i values from oxadiazon resistant (OR) plants. The OR populations in Auburn University trials required an estimated 700.25 and 3500.024 kg ha^{-1}, respectively, while the Craft Farms (S) plants required only 0.17 and 0.52 kg ha^{-1}, respectively based upon goosegrass control. These data indicate that oxadiazon rates required to achieve commercially acceptable control (90%) (Cox 2014) were significantly higher for the SR than the S successions. Results based upon goosegrass weights were similar to the goosegrass control data collected in the study. The OR populations required an estimated 15.58 and 3000.71 kg ha^{-1}, respectively, while the Craft Farms (S) population required 0.23 and 2.52 kg ha^{-1}.

Utilizing the rapid postemergence screening methods resulted in ecotypes RB and CCV showing lack of control from all rates of oxadiazon used in the study. Based on our results, rapid postemergence screening of goosegrass plants with oxadiazon, collecting plants from the field and applying postemergence, is a viable replacement for preemergence testing. This allows a

researcher to gather more data by conducting more tests that can be completed rapidly when compared to preemergence testing. Additionally, RB and CCV ecotypes are not resistant to flumioxazin and sulfentrazone based on our results. Flumioxazin provided superior goosegrass control when compared with sulfentrazone and oxadiazon.

Flumioxazin applied at 0.08 kg ha^{-1} controlled 1-2 tiller goosegrass plants 60% 6 WAT. Flumioxazin applied at 0.35 kg ha^{-1} controlled goosegrass plants susceptible to oxadiazon 100% 6 WAT. Control was consistent across all ecotypes and across all runs. Overall, all flumioxazin treatments used in this study appear to have potential for postemergence goosegrass control, however flumioxazin causes unacceptable injury to bermudagrass. Reed (2013) reported similar results with flumioxazin for postemergence annual bluegrass control. He noted that flumioxazin caused greater injury to annual bluegrass when temperatures were near 30 C, which were similar temperatures recorded for the study we conducted at Auburn University.

In conclusion, rapid postemergence screening of goosegrass with oxadiazon, yielded similar results as the preemergence oxadiazon study conducted in 2015 at Auburn University. Based on our results, sulfentrazone and flumioxazin showed adequate control of oxadiazon resistant goosegrass populations collected from Virginia and North Carolina indicating that a mutation could have occurred in the plant that is site specific to oxadiazon.

LITERATURE CITED

Askew SD, Beam JB (2002) Turfgrass response to flumioxazin, mesotrione, and rimsulfuron. Proc. Northeast. Weed Sci. 56:127-128

Askew SD, Cox M, Spak DR (2013) A suspected oxadiazon-resistant goosegrass population in Virginia. Proceedings, South. Weed Sci. 66:229

Bartley MR (1993) Assessment of herbicide selectivity. Herbicide Bioassays. Boca Raton, FL: CRC Press. Pp 57–73

Beard JB (2005) Beard's Turfgrass Encyclopedia for Golf Courses, Grounds, Lawns, Sports Fields. Michigan State University Press. 286 p

Bingham SW, Shaver RL (1981) Goosegrass (*Eleusine indica*) control during bermudagrass (*Cynodon dactylon*) establishment. Weed Sci.:11-16

Cox MC (2014) Characterizing oxadiazon resistance and improving postemergence control programs for goosegrass (Eleusine indica) in bermudagrass (Cynodon spp.). Ph.D dissertation, Blacksburg, VA: Virginia Polytechnic and State University. 125 p

Culpepper AS, Grey TL, Vencill WK, Kichler JM, Webster TM, Brown SM, York AC, Davis JW, Hanna WW (2006) Glyphosate-resistant Palmer amaranth (Amaranthus palmeri) confirmed in Georgia. Weed Sci. 54.4:620-626

Ferrell JA, Murphy TR, Vencill WK (2003) Tolerance of Winter-Installed Tall Fescue (Festuca arundinacea) and Hybrid Bermudagrass (*Cynodon transvaalensis*× *C. dactylon*) Sod to Herbicides 1. Weed Tech. 17.3:521-525

Leibhart LJ (2014) Speedzone has Potential for Postemergence Goosegrass Control in Perennial Ryegrass and Creeping Bentgrass. Applied Turfgrass Sci. 11.1

McCarty LB (1991) Goosegrass (*Eleusine indica*) control in bermudagrass (*Cynodon spp*.) turf with diclofop. Weed Sci.: 24: 255-261

McCullough PE, De Barreda DG, Raymer P (2012) Nicosulfuron use with foramsulfuron and sulfentrazone for late summer goosegrass (*Eleusine indica*) control in bermudagrass and seashore paspalum. Weed Tech. 26.2:376-381

Mudge LC, Gossett BJ, Murphy TR (1984) Resistance of goosegrass (Eleusine indica) to dinitroaniline herbicides. Weed Sci. 1.59:1-4

Parka SJ, Soper OF (1977) The Physiology and mode of action of the dinitroaniline herbicides. Weed Sci. 25.1:79-87

Reed T (2013) Evaluation of flumioxazin efficacy for annual bluegrass control in warm-season turfgrasses. Master's Thesis, University of Georgia at Athens. 101 p

Seefeldt SS, Jensen JE, Fuerst EP (1995) Log-logistic analysis of herbicide dose-response relationships. Weed Tech. 9:218-227

Senseman SA (2007) Herbicide Handbook. 9th ed. Champaign, IL: Weed Science Soc.: Pp 212-213

Wiecko G (2000) Sequential Herbicide Treatments for Goosegrass (*Eleusine indica*) Control in Bermudagrass (*Cynodon dactylon*). Weed Tech. 14.4:686-691

Table 3.1. The five goosegrass populations included for evaluation of their response to 3 PPO inhibiting herbicides.

Goosegrass population, abbreviation and collection location	Reason collected
Country Club of Virginia (CCV), golf course in Richmond, VA	Oxadiazon resistant[a]
River Bend Golf Course (RB), golf course in New Bern, VA	Oxadiazon resistant
Soybean Field (AL) near Town Creek, AL	Endemic to an agronomic row crop field, but with no known herbicide resistance.
Craft Farms Golf course (CF), golf course in Gulf Shores, AL	Endemic to golf course; no known herbicide resistance.
Willow Creek Golf course (TX), golf course in Dallas, TX	Endemic to golf course putting green with dwarf growth characteristics, no known herbicide resistance.

[a] Known resistance based on previous research conducted by Cox (2014) at Virginia Tech University.

Table 3.2. Goosegrass above-ground fresh weight as influenced by three PPO - inhibiting herbicides applied at their labeled rate, plants were evaluated 6 weeks after a single postemergence application.[a]

Treatment		Goosegrass[c] population				
Herbicide	Rate	CCV	CF	TX	AL	RB
	kg ai ha^{-1}	g pot^{-1}				
Non-treated	-	7.83	5.24	3.45	7.35	8.02
Flumioxazin	1.43	0	0	0	0	0
Oxadiazon	1.12	4.66	0.25	0.55	0.5	4.89
Sulfentrazone	1.12	0	0	0.31	0	0
LSD[b]		1.22	1.24	1.11	1.23	1.06

[a]All treatments were applied once. Experiments were conducted and repeated in 2015; data pooled for analysis and presentation.

[b] LSD ($P<0.05$) values for comparison of means within a common goosegrass population.

[c] Refer to Table 3.1 for description of goosegrass populations.

Table 3.3. Goosegrass visual control as influenced by three PPO - inhibiting herbicides applied at their labeled rate; evaluated 6 weeks after a single postemergence application.[a]

Treatment		Goosegrass populations[c]				
Herbicide	Rate	CCV	RB	CF	TX	AL
	kg ai ha^{-1}	% control				
Non-treated	0	0	0	0	0	0
Flumioxazin	1.43	100	100	100	100	100
Oxadiazon	1.12	16	20	80	76	75
Sulfentrazone	1.12	100	100	100	100	100
LSD[b]	-	2	3	19	31	14

[a]Greenhouse experiment using a Marvyn sandy loam soil. Control ratings were taken 42 DAT. All treatments were applied once. Experiments were conducted and repeated in 2015; data pooled for analysis and presentation.

[b] LSD ($P<0.05$) value for comparison of means within a common population.

[c] Refer to Table 3.1 for goosegrass population descriptions.

Table 3.4. Best fit equation as determined from either linear or nonlinear regression analysis of goosegrass foliar fresh weight in response to a rate progression of three, PPO - inhibiting herbicides.[a]

Goosegrass Populations[b]	Herbicide	Best fit regression equation[c]	R^2
AL	oxadiazon	$y= 28.73\exp^{-1.00x}$	0.82
RB	oxadiazon	$y= 80.28 - 22.54x$	0.92
CCV	oxadiazon	$y= 59.31\exp^{-0.49x}$	0.95
CF	oxadiazon	$y= 18.42\exp^{-1.55x}$	0.94
TX	oxadiazon	$y= 35.81\exp^{-0.89x}$	0.86
AL	sulfentrazone	$y= 92.51/(1+\exp^{(-(x-0.78)/-0.04))}$	0.99
RB	sulfentrazone	$y= 99.18/(1+\exp^{(-(x-0.84)/-0.06))}$	0.99
CCV	sulfentrazone	$y=101.25/(1+\exp^{(-(x-0.93)/-0.15))}$	0.99
CF	sulfentrazone	$y=96.99/(1+\exp^{(-(x-0.60)/-0.19))}$	0.96
TX	sulfentrazone	$y=97.51/(1+\exp^{(-(x-0.80)/-0.04))}$	0.99
AL	flumioxazin	$y= 106.02/(1+\exp^{(-(x-0.56)/-0.36))}$	0.98
RB	flumioxazin	$y= 104.67/(1+\exp^{(-(x-0.56)/-0.34))}$	0.97
CCV	flumioxazin	$y= 97.85/(1+\exp^{(-(x-0.34)/-0.22))}$	0.98
CF	flumioxazin	$y= 34.17/(1+\exp^{(-(x-0.92)/-0.46))}$	0.95
TX	flumioxazin	$y= 89.67/(1+\exp^{(-(x-0.49)/-0.17))}$	0.96

[a] Greenhouse experiment using a Marvyn sandy loam soil. All treatments were a single postemergence application. Experiments conducted and repeated in 2015; data pooled for analysis and presentation.

[b] Refer to Table 3.1 for goosegrass population descriptions.

[c] Equations: exp. decay: $y = a*\exp^{(-bx)}$, linear: $y= y0+ax$, and sigmoidal: $y= a/(1+\exp^{(-(x-x0)/b)})$; where x = herbicide rate, y = response variable (weight).

Table 3.5. Best fit equation as determined from either linear or nonlinear regression analysis of goosegrass visual control in response to a rate progression of three, PPO inhibiting herbicides.[a]

Goosegrass Populations[b]	Herbicide	Best fit regression equation[c]	R^2
AL	oxadiazon	$y=102.69/(1+\exp^{(-(x-0.47)/0.29))}$	0.98
RB	oxadiazon	y= 13.05+11.71x	0.95
CCV	oxadiazon	y= 16.91+12.90*x	0.94
CF	oxadiazon	$y= 94.63/(1+\exp^{(-(x-0.77)/0.21))}$	0.94
TX	oxadiazon	$y= 107.69/(1+\exp^{(-(x-0.04)/0.23))}$	0.95
AL	sulfentrazone	$y= 101.10/(1+\exp^{(-(x-0.83)/0.14))}$	0.99
RB	sulfentrazone	$y= 99.92/(1+\exp^{(-(x-0.87)/0.13))}$	0.99
CCV	sulfentrazone	$y= 101.36/(1+\exp^{(-(x-0.77)/0.18))}$	0.99
CF	sulfentrazone	$y= 97.56/(1+\exp^{(-(x-0.90)/0.16))}$	0.99
TX	sulfentrazone	$y= 100.10/(1+\exp^{(-(x-0.99)/0.08))}$	0.99
AL	flumioxazin	$y= 101.71/(1+\exp^{(-(x-0.80)/0.19))}$	0.98
RB	flumioxazin	$y= 100.36/(1+\exp^{(-(x-0.62)/0.19))}$	0.99
CCV	flumioxazin	$y= 99.95/(1+\exp^{(-(x-0.81)/0.18))}$	0.99
CF	flumioxazin	$y= 98.47/(1+\exp^{(-(x-0.85)/0.24))}$	0.97
TX	flumioxazin	$y= 101.04/(1+\exp^{(-(x-0.89)/0.20))}$	0.98

[a] Greenhouse experiment using a Marvyn sandy loam soil. All treatments were a single application. Experiments conducted and repeated in 2015; Data pooled for analysis and presentation. Visual control = fresh weight suppression relative to non-treated control.

[b] Refer to Table 3.1 for goosegrass population descriptions.[c] Equations: linear: y = y0+a*x, and sigmoidal: y= a/(1+exp(-(x-x0)/b)); where x being oxadiazon rate, y being response variable (weight).

Table 3.6. Estimated rates of three PPO inhibiting herbicides required for 50 and 90 % (I_{50} and I_{90}, respectively) foliar fresh weight reduction of goosegrass populations as determined from best fit regression equations.[a]

Goosegrass populations[b]	Herbicide	Rate	
		I_{50}	I_{90}
		kg ai ha^{-1}	
AL	oxadiazon	0.28	11.52
RB	oxadiazon	21.88	1288.24
CCV	oxadiazon	2.29	5495.41
CF	oxadiazon	0.23	2.52
TX	oxadiazon	0.43	29.51
AL	sulfentrazone	0.17	0.20
RB	sulfentrazone	0.15	0.20
CCV	sulfentrazone	0.12	0.26
CF	sulfentrazone	0.25	0.66
TX	sulfentrazone	0.16	0.20
AL	flumioxazin	0.31	1.78
RB	flumioxazin	0.30	1.58
CCV	flumioxazin	0.46	1.35
CF	flumioxazin	0.21	1.70
TX	flumioxazin	0.30	0.74

[a] Best fit regression equations identified in Table 3.4.

[b] Refer to Table 3.1 For goosegrass population descriptions.

Table 3.7. Estimated rates from three PPO-inhibiting herbicides required for 50 and 90% visual control (I_{50} and I_{90} respectively) of goosegrass populations determined from best fit regression equations.[a]

Goosegrass populations[b]	Herbicide	Rate	
		I_{50}	I_{90}
		kg ai ha^{-1}	
AL	oxadiazon	0.33	1.26
RB	oxadiazon	1412.54	3612277.64
CCV	oxadiazon	363.08	457088.11
CF	oxadiazon	0.17	0.52
TX	oxadiazon	0.85	2.14
AL	sulfentrazone	0.15	0.30
RB	sulfentrazone	0.14	0.26
CCV	sulfentrazone	0.17	0.40
CF	sulfentrazone	0.12	0.32
TX	sulfentrazone	0.10	0.15
AL	flumioxazin	0.16	0.39
RB	flumioxazin	0.24	0.62
CCV	flumioxazin	0.15	0.39
CF	flumioxazin	0.14	0.54
TX	flumioxazin	0.13	0.34

[a] Best fit regression equations defined in Table 3.5.

[b] Refer to Table 3.1 for goosegrass population descriptions.

Figure Legends

Figure 3.1. Sigmoidal regression analysis of flumioxazin for control of goosegrass populations. Refer to Table 3.1 for goosegrass population descriptions. Refer to Table 3.4 for regression equations and R^2 values.

Figure 3.2. Exponential decay and linear transformed regression analysis of oxadiazon for control of goosegrass populations. Refer to Table 3.1 for goosegrass population descriptions. Refer to Table 3.4 for regression equations and R^2 values.

Figure 3.3. Sigmoidal regression analysis of sulfentrazone for control of goosegrass populations. Refer to Table 3.1 for goosegrass population descriptions. Refer to Table 3.4 for regression equations and R^2 values.

Figure 3.4. Sigmoidal regression analysis of flumioxazin applications for control of goosegrass populations. Refer to Table 3.1 for goosegrass population descriptions. Refer to Table 3.5 for regression equations and R^2 values.

Figure 3.5. Exponential decay and linear transformed regression analysis of oxadiazon applications for control of goosegrass populations. Refer to Table 3.1 for goosegrass population descriptions. Refer to Table 3.5 for regression equations and R^2 values.

Figure 3.6. Sigmoidal regression analysis of sulfentrazone applications for control of goosegrass populations. Refer to Table 3.1 for goosegrass population descriptions. Refer to Table 3.5 for regression equations and R^2 values.

Figure 3.1.

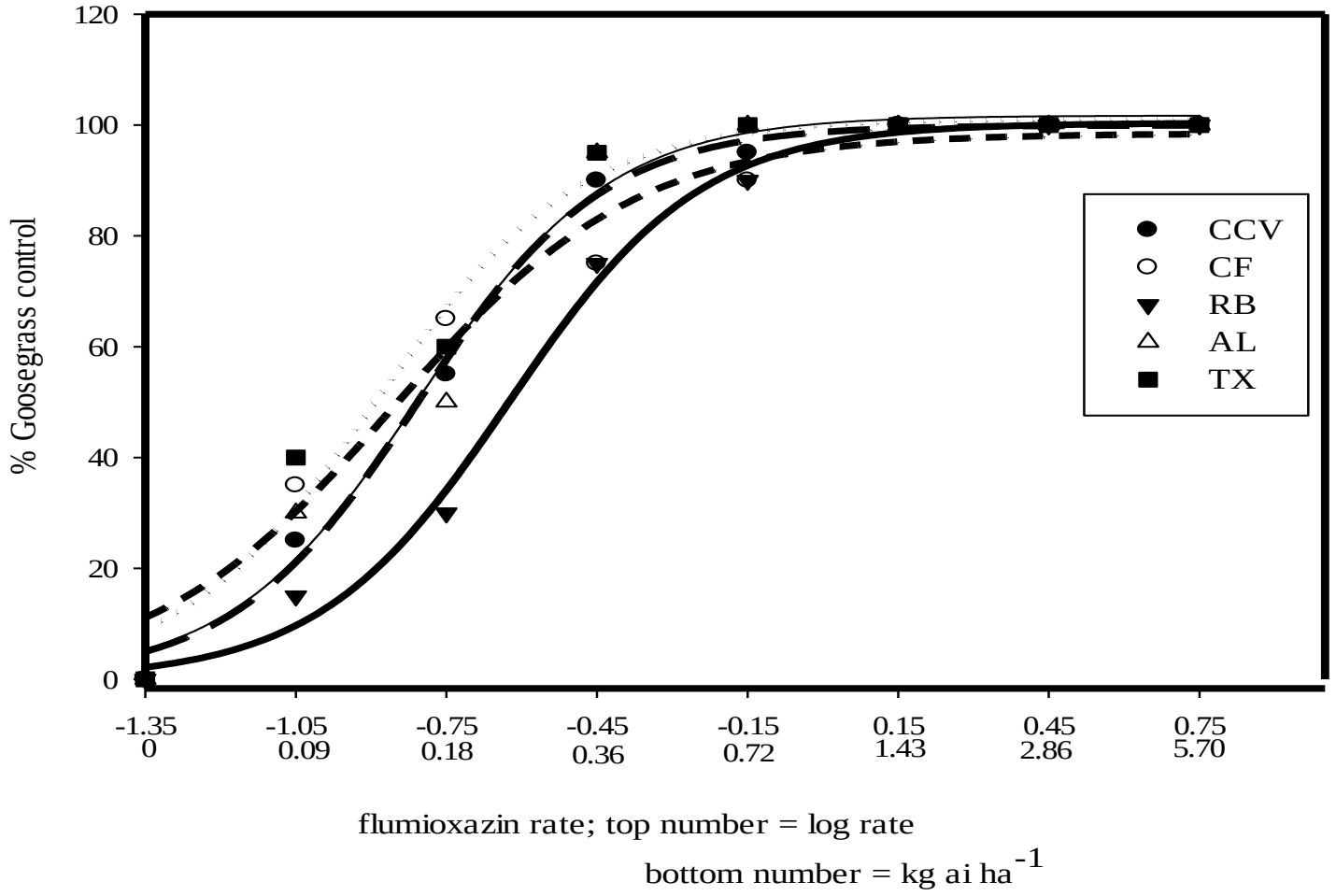

120
100
80
60
40
20
0
% Goosegrass control
CCV
CF
RB
AL
TX
-1.35
0
-1.05
0.09
-0.75
0.18
-0.45
0.36
-0.15
0.72
0.15
1.43
0.45
2.86
0.75
5.70
flumioxazin rate; top number = log rate
bottom number = kg ai ha-1

Figure 3.2

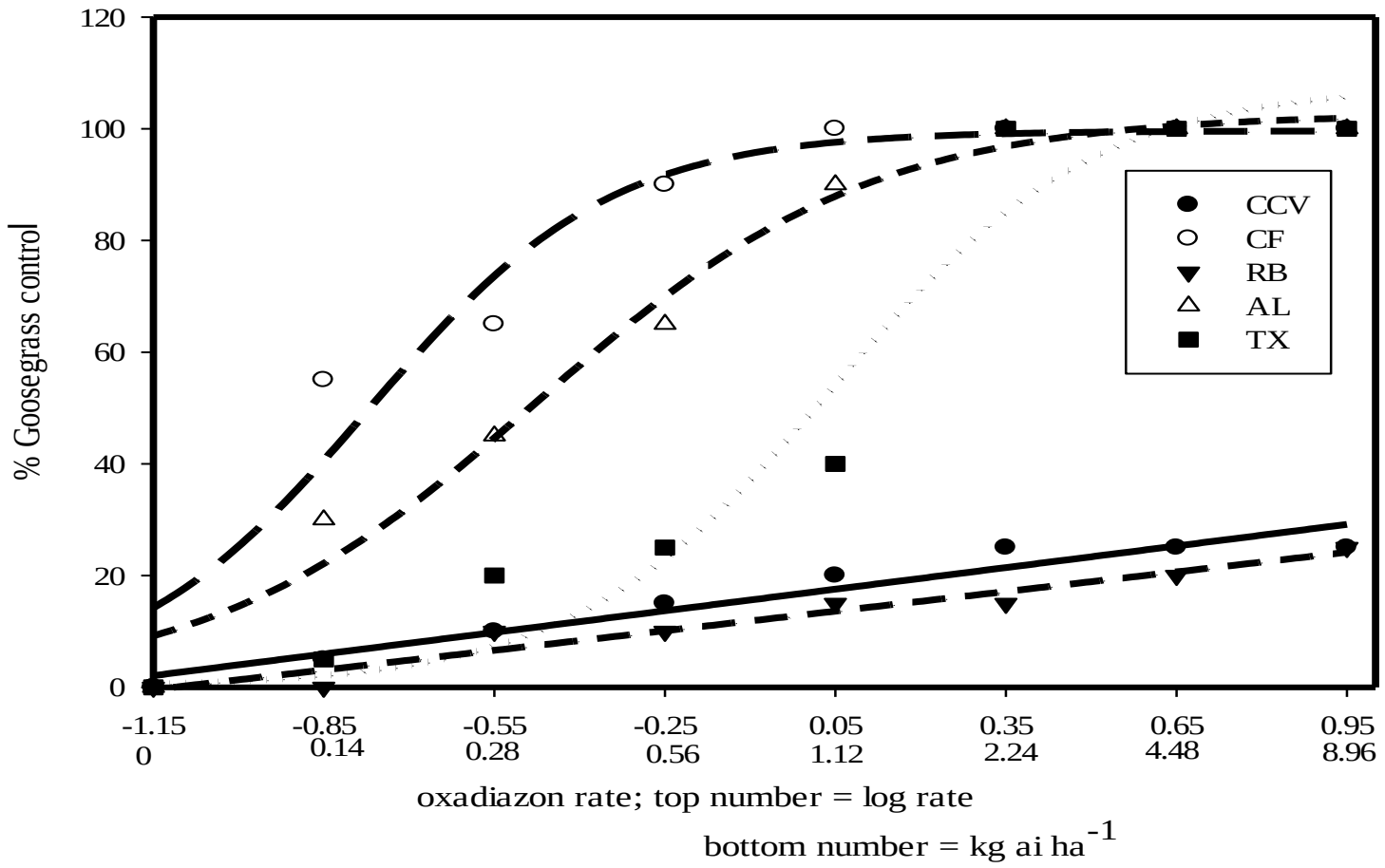

120
100
80
60
40
20
0
% Goosegrass control
CCV
CF
RB
AL
TX
-1.15
0
-0.85
0.14
-0.55
0.28
-0.25
0.56
0.05
1.12
0.35
2.24
0.65
4.48
0.95
8.96
oxadiazon rate; top number = log rate
bottom number = kg ai ha-1

Figure 3.3.

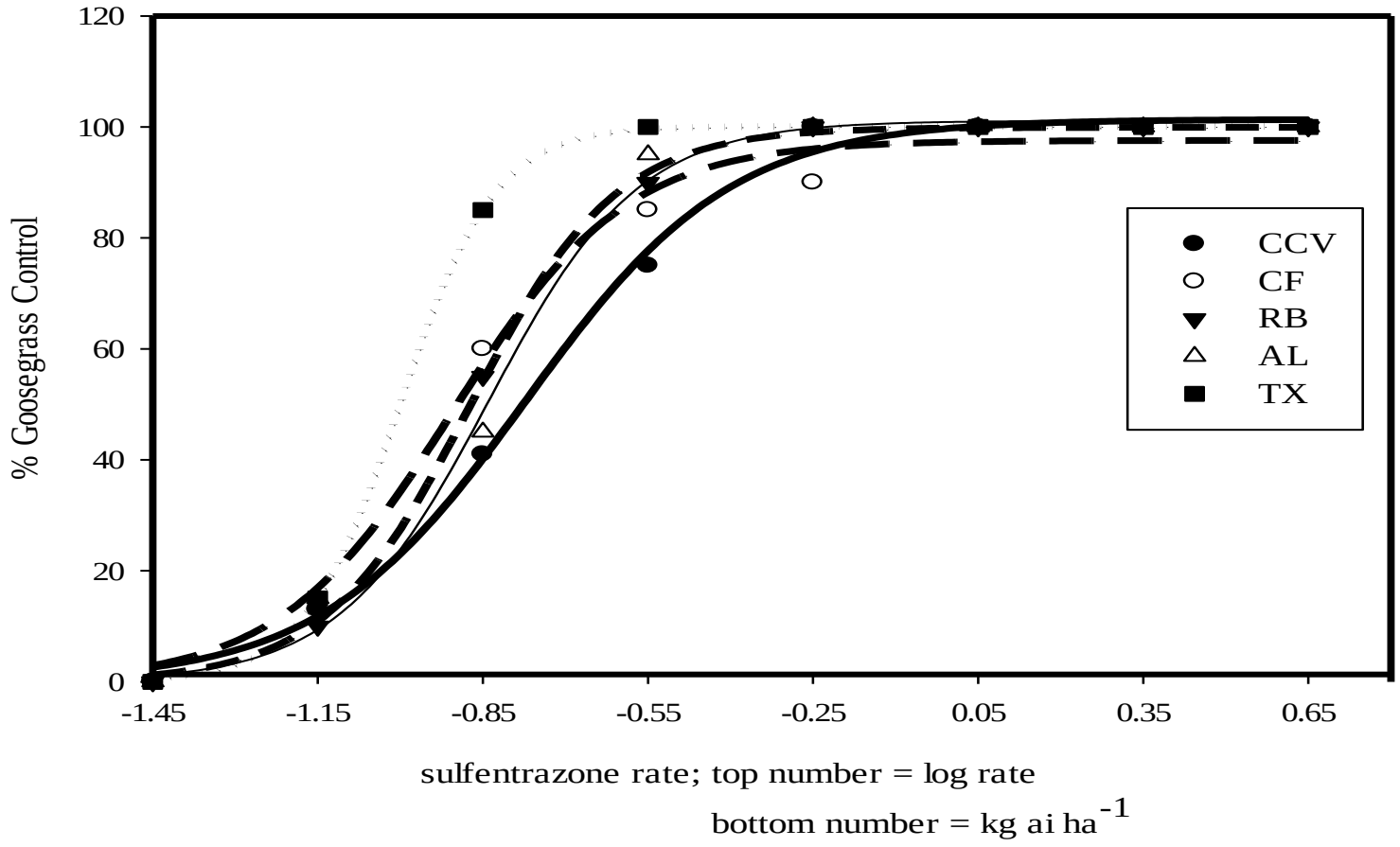

120
100
80
60
40
20
0
% Goosegrass Control
-1.45
-1.15
-0.85
-0.55
-0.25
0.05
0.35
0.65
CCV
CF
RB
AL
TX
sulfentrazone rate; top number = log rate
bottom number = kg ai ha-1

Figure 3.4.

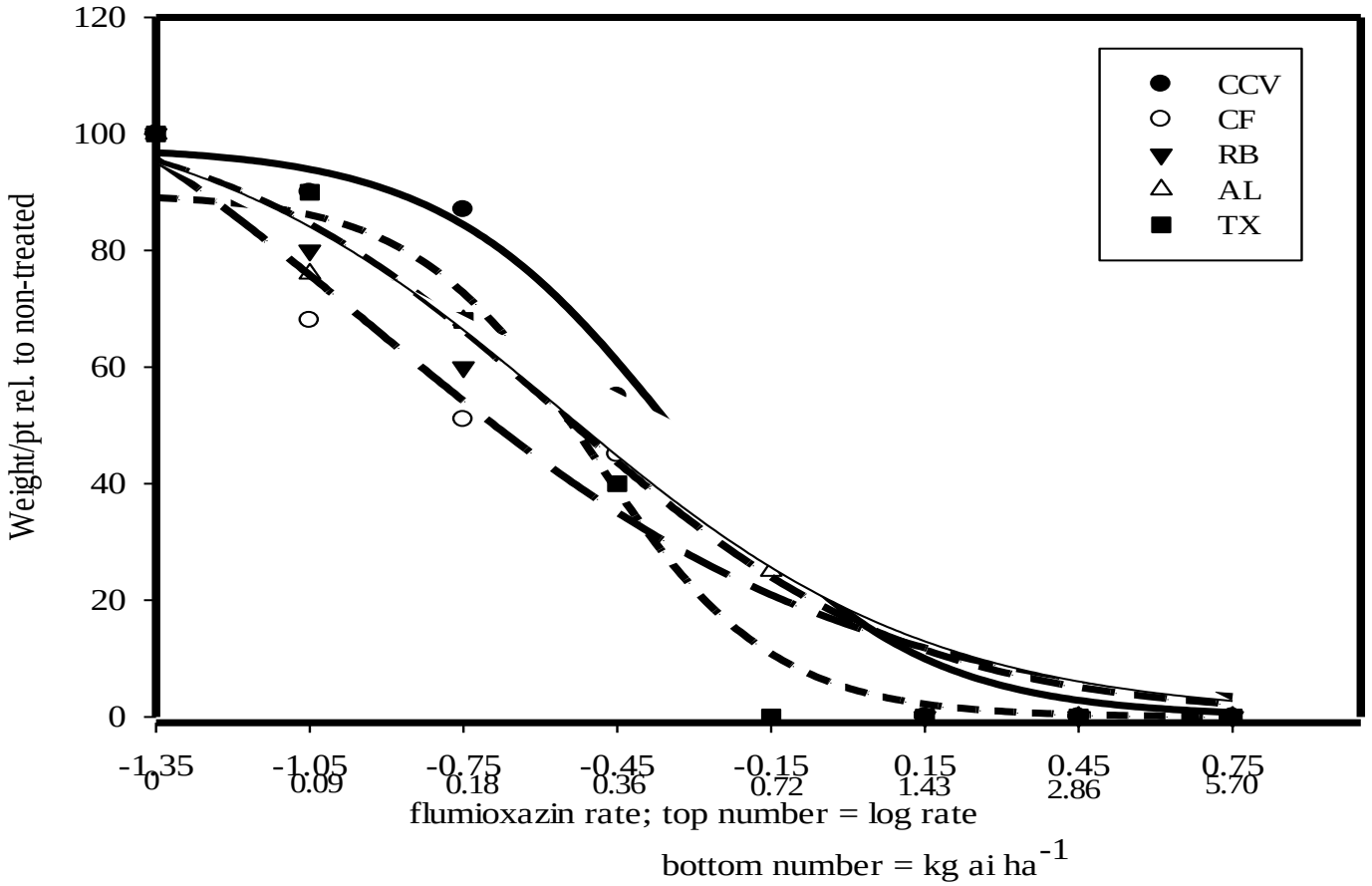

120
100
80
60
40
20
0
Weight/pt rel. to non-treated
CCV
CF
RB
AL
TX
-1.35
-1.05
-0.75
-0.45
-0.15
0.15
0.45
0.75
0
0.09
0.18
0.36
0.72
1.43
2.86
5.70
flumioxazin rate; top number = log rate
bottom number = kg ai ha-1

Figure 3.5.

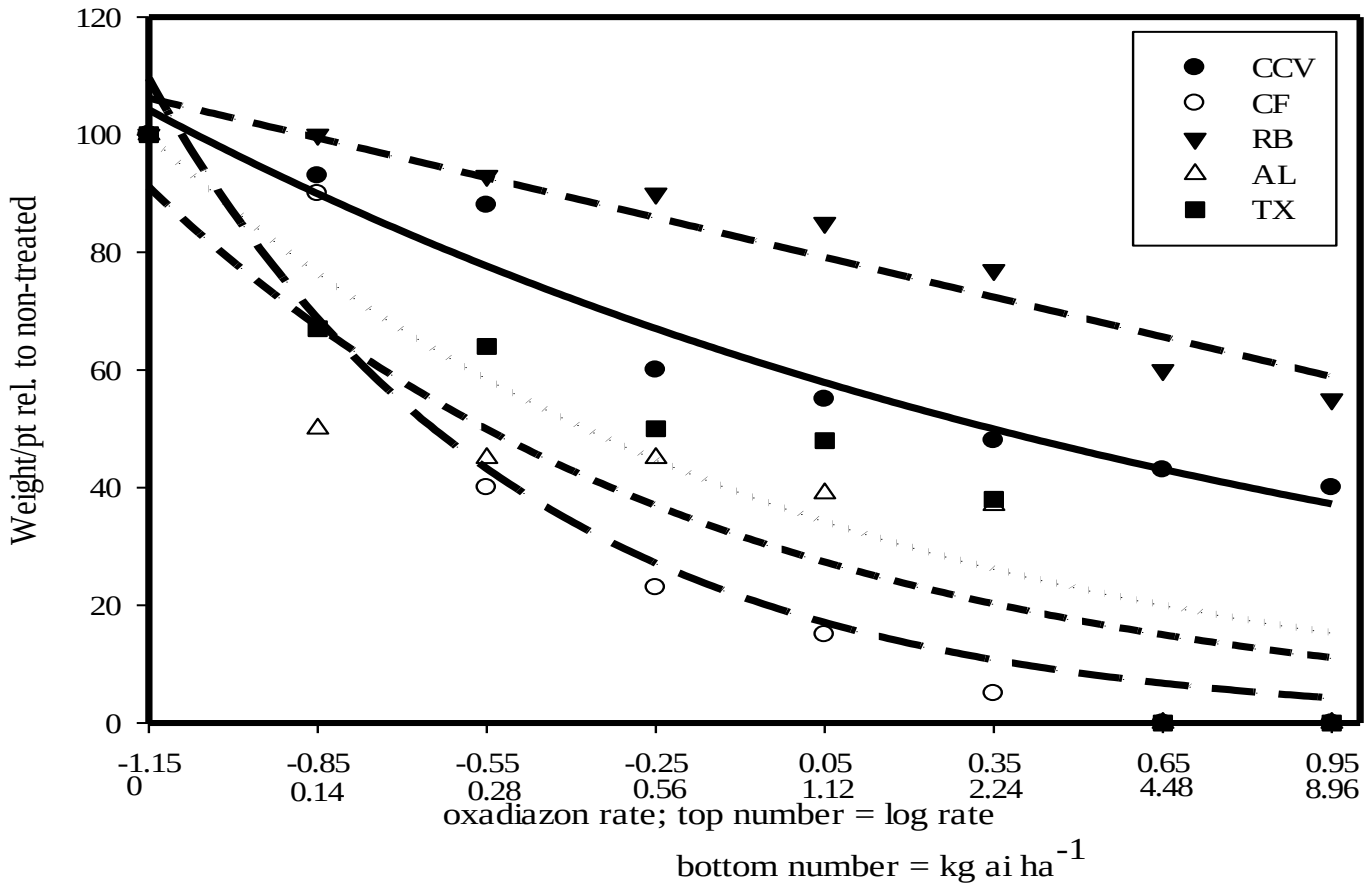
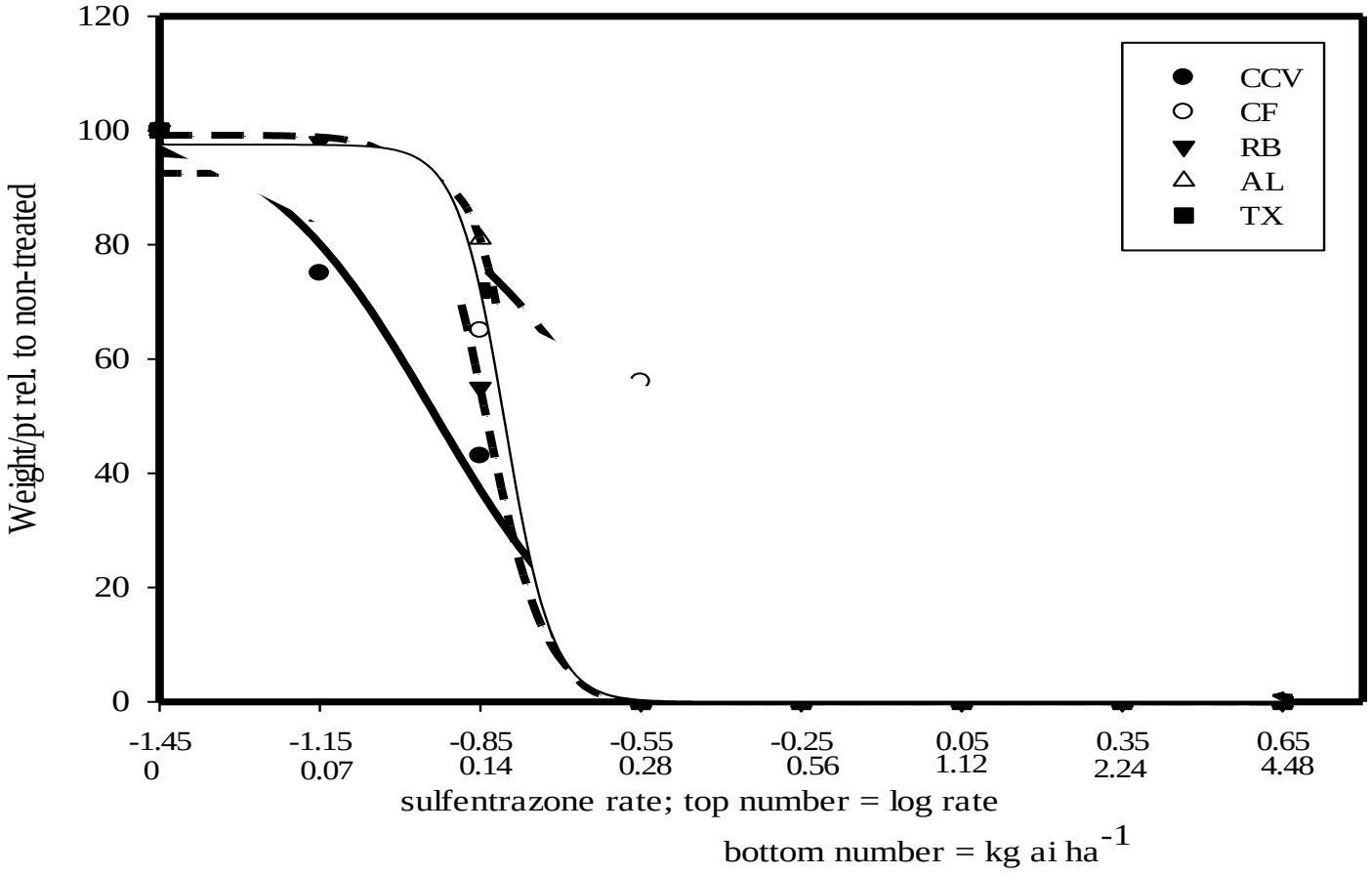

120
100
80
60
40
20
0
Weight/pt rel. to non-treated
CCV
CF
RB
AL
TX
-1.15
0
-0.85
0.14
-0.55
0.28
-0.25
0.56
0.05
1.12
0.35
2.24
0.65
4.48
0.95
8.96
oxadiazon rate; top number = log rate
bottom number = kg ai ha-1

Figure 3.6.


120
100
80
60
40
20
0
Weight/pt rel. to non-treated
CCV
CF
RB
AL
TX
-1.45
0
-1.15
0.07
-0.85
0.14
-0.55
0.28
-0.25
0.56
0.05
1.12
0.35
2.24
0.65
4.48
sulfentrazone rate; top number = log rate
bottom number = kg ai ha-1