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SOYBEAN [*Glycine max* (L.) Merr] GROWTH AND DEVELOPMENT, WHITE MOLD [*Sclerotinia sclerotiorum* (Lib.) de Bary] INCIDENCE, AND YELLOW NUTSEDGE (*Cyperus esculentus* L.) CONTROL AS AFFECTED BY GLYPHOSATE AND OTHER HERBICIDES

By

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A DISSERTATION

Submitted to
Michigan State University
in partial fulfillment of the requirements
for the degree of

DOCTOR OF PHILOSOPHY

Department of Crop and Soil Sciences

2000

ABSTRACT

SOYBEAN [*Glycine max* (L.) Merr] GROWTH AND DEVELOPMENT, WHITE MOLD [*Sclerotinia sclerotiorum* (Lib.) de Bary] INCIDENCE, AND YELLOW NUTSEDGE (*Cyperus esculentus* L.) CONTROL AS AFFECTED BY GLYPHOSATE AND OTHER HERBICIDES

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Field and greenhouse research was conducted to determine the effect of glyphosate and other herbicides on soybean [*Glycine max* (L.) Merr] growth and development, the incidence of white mold [*Sclerotinia sclerotiorum* (Lib.) de Bary] in glyphosate-resistant and non-resistant soybean, and the influence of glyphosate and acetolactate synthase (ALS)-inhibiting herbicides on yellow nutsedge control and tuber production. In the absence of white mold, soybean canopy development was reduced, reproductive development was delayed, and yield was reduced from 130 to 270 kg ha^{-1} with tank mixture treatments of bentazon/acifluorfen plus thifensulfuron plus sethoxydim and lactofen plus bentazon plus clethodim applied to soybean at V5. Soybean tolerance to white mold was not related to glyphosate-resistance in the cultivars evaluated. 'S 12-49', 'S14-M7' Roundup Ready® (RR), 'S 19-90', and 'S20-B9' (RR) had a lower incidence of white mold compared to 'GL2415', 'GL2600' (RR), 'P9281', and 'P93B01'(RR). Glyphosate did not affect soybean growth and development or the incidence of white mold in glyphosate-resistant soybean. Thifensulfuron reduced soybean leaf area and delayed reproductive development, but did not affect the incidence of white mold. Lactofen reduced soybean leaf area, delayed reproductive development, increased phytoalexin

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production 2 to 26 d after treatment, and reduced the incidence of white mold compared to untreated soybean. However, in the presence of white mold, neither lactofen nor thifensulfuron affected soybean yield. Other protoporphyrinogen-inhibiting herbicides such as sulfentrazone, oxyfluorfen, and oxadiazon reduced *Sclerotinia sclerotiorum* lesion growth similar to lactofen. Lactofen and sulfentrazone postemergence increased leaf phytoalexin production, but did not influence phytoalexin production in soybean stems. Sulfentrazone preemergence also increased soybean phytoalexin production compared to untreated soybean. Glyphosate at 840 g ae ha^{-1} suppressed yellow nutsedge 13 to 34% greater than glufosinate at 400 g ae ha^{-1}. Additional adjuvant did not increase or decrease yellow nutsedge control with glyphosate. When glyphosate was tank mixed with acetolactate synthase (ALS)-inhibiting herbicides aboveground visual control increased, but tuber yield was similar to the ALS-inhibiting herbicides applied alone. Yellow nutsedge control by the herbicides evaluated and applied at recommended use rates was ranked halosulfuron and chlorimuron (> 80% control and reduction in tuber density); imazethapyr/imazapyr, imazethapyr, and glyphosate (50 to 80% control and reduction in tuber density); and cloransulam, rimsulfuron, and imazamox (20 to 50% control and reduction in tuber density). However, pyrithiobac controlled yellow nutsedge 48% and reduced tuber density 60%.

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ACKNOWLEDGMENTS

A true friend is someone who shares our hopes and dreams. I want to thank everyone who has been involved in this research for being wonderful friends. My family and extended family deserve a special thanks for their encouragement, enthusiasm, and assistance. A distinctive thanks goes out to my lovely wife, Teak, for her love, confidence, help, support, and devotion throughout the last three years. This degree would not have been possible without our family. My parents, Lloyd and Betty Nelson, for providing me with a life time of wonderful experiences. Ken and Barbara Nelsen for their support throughout the process. Chris, Kathy, and Zachary Long have always been there for us no matter what the occasion and kept us accountable for the important things in life.

A special thank you is extended to Dr. Karen Renner for her encouragement, example as a teacher and parent, editing expertise, and for helping make this degree a reality. I would like to thank Dr. Donald Penner for all of his help and wisdom. I will never forget all of the late night visits. I am also grateful for the motivation and friendship of Dr. Ray Hammerschmidt and Dr. Irvin Widders. Amy Guza, Pat O'Boyle, Mike Partika, Rob Herron, and Gary Powell deserve a huge thanks for their technical assistance. I would also like to thank Chad Lee, Kyle Poling, Nate Kemp, Sherry White, Jason Fausey, Luis Velasquez, Tiffiny Murrell, Brent Tharp, Christy Sprague, Joe Simmons, Caleb Dalley, Matt Rinella, Andy Chomas, John Boyse, Dr. Jim Kells, Kyle Fiebig, Julie Root, and Stephanie Eickholt for their assistance and support through the various aspects of graduate school.

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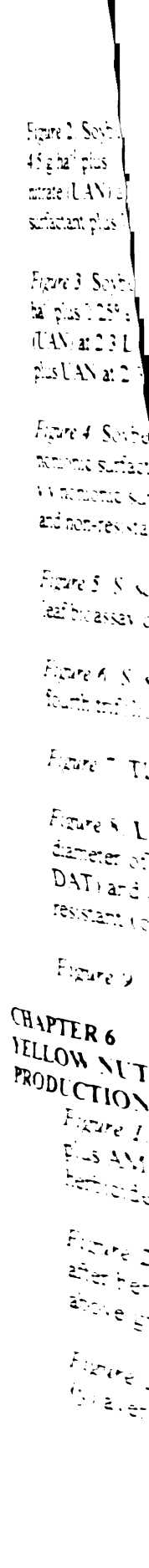

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INTRODUCTION

The use of effective, broad spectrum postemergence herbicide treatments has encouraged the adoption of reduced tillage practices and the transformation of crop production from wide to narrow row culture. The utilization of glyphosate-resistant soybean technology has rapidly increased in the past few years. This technology allows a postemergence application of glyphosate, a non-selective herbicide, to soybean with no observed phytotoxicity.

White mold, caused by the fungus [*Sclerotinia sclerotiorum* (Lib.) de Bary], is a disease on the rise and is a prominent problem in Michigan. A majority of the glyphosate-resistant soybean have been grown in narrow-rows for early canopy closure and weed control. However, white mold is prevalent in narrow-row soybean cultures. In 1997, white mold was wide-spread and questions regarding the susceptibility of glyphosate-resistant soybean and the effect of glyphosate (formulated as Roundup Ultra®) on the incidence of white mold in soybean were asked by producers, agribusiness personnel, and extension specialists. However, other postemergence herbicides may change soybean growth and development and reduce the incidence of white mold [*Sclerotinia sclerotiorum* (Lib.) de Bary] in soybean. An interaction between herbicide treatments and the incidence of white mold in soybean could affect production practices and weed control recommendations. A weed control timing that targets white mold suppression could be attractive and profitable for producers.

Glyphosate-resistant crops have provided new opportunities for cost-effective perennial weed management for Michigan producers. However, yellow nutsedge is a

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perennial weed that is difficult to control with glyphosate applied at a rate typically used for annual weed management. As glyphosate-resistant crops become more prevalent, producers may select for weeds that are not completely controlled by glyphosate like yellow nutsedge. Tank mixture treatments of glyphosate with residual ALS-inhibiting herbicides could increase control and reduce below ground tuber production which could affect recommendations and the management of this weed in glyphosate-resistant crops. In addition, treatments that reduce tuber production could also help eradicate this weed species over time or manage this weed in a glyphosate-resistant rotational crop production system.

Field and greenhouse research was conducted to determine the effect of glyphosate and other herbicides on soybean growth and development, the incidence of white mold in glyphosate-resistant and non-resistant soybean, and yellow nutsedge control and tuber production.

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CHAPTER 1

GLYPHOSATE-RESISTANT AND NON-RESISTANT SOYBEAN (*Glycine max*) GROWTH AND DEVELOPMENT AS AFFECTED BY GLYPHOSATE AND POSTEMERGENCE HERBICIDE TANK MISTURES

Abstract: Field research was conducted to evaluate the effects of glyphosate and postemergence herbicide tank mixtures on soybean development, canopy development, and yield of glyphosate-resistant and non-resistant cultivars. Herbicide treatments were applied to V5 soybean. Glyphosate did not affect growth, development, or yield of resistant soybean compared to the untreated control. Soybean injury was 14 to 18% 21 d after treatment (DAT) from postemergence tank mixture treatments of bentazon/acifluorfen + thifensulfuron + sethoxydim and lactofen + bentazon + clethodim. Red:far red light that reached the soil surface 28 DAT was dependent on the soybean cultivar and herbicide treatment. Soybean injury caused by postemergence herbicide tank mixtures reduced vegetative development 7 DAT, reproductive development 20 and 80 DAT, height, and dry weight. The leaf area index was reduced by the postemergence tank mixtures up to 52 DAT, and canopy development was delayed 70 and 80 DAT depending on the cultivar. Soybean yield in plots treated with herbicides other than glyphosate was reduced 130 to 270 kg ha^{-1} compared to the untreated control. Yield of soybean cultivars varied by year with a ranking of A1900 = A2704 = AG2701 > AG1901 in 1997, and A2704 > AG2701 > A1900 > AG1901 in 1998.

Abbreviations: AMS, ammonium sulfate ($(NH_4)_2SO_4$); 'Asgrow 1900', A1900; 'Asgrow 1901', AG1901; 'Asgrow 2701', AG2701; 'Asgrow 2704', A2704; COC, crop oil concentrate; DAT, days after treatment; LAI, leaf area index; UAN, 28% urea ammonium nitrate (NH_4NO_3).

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INTRODUCTION

Tank mixtures of postemergence herbicides were usually necessary for broadspectrum weed control prior to the introduction of glyphosate-resistant soybean cultivars (Fielding and Stoller 1990, Green 1991, Hart and Roskamp 1998, Monks et al 1993). Glyphosate applications to glyphosate-resistant soybean did not injure soybean (Lich et al. 1997, Nelson and Renner 1999) which allowed for rapid canopy closure (Nelson and Renner 1999). Other postemergence herbicides, such as thifensulfuron, lactofen, and acifluorfen cause chlorosis, necrosis, or stunting of soybean (Hart and Roskamp 1998, Kapusta et al. 1986, Wichert and Talbert 1993). Such herbicide injury may persist in weed-free conditions up to 21 d after treatment yet result in no yield loss (Kapusta et al. 1986). However, the use of postemergence herbicides may alter the canopy and delay canopy closure such that late germinating weeds or difficult-to-control weeds may escape control (Mickelson and Renner 1997, Nelson and Renner 1999).

A change in canopy development may also influence development of white mold [*Sclerotinia sclerotiorum* (Lib.) deBary], a common disease problem for producers in the north central region. White mold is common in narrow-row soybean culture (Grau and Radkey 1984) and crop rotations that include hosts like dry edible bean (*Phaseolus vulgaris* L.) (Schwartz et al. 1978). Postemergence herbicides such as lactofen may suppress white mold in soybean (Dann et al. 1999) and affect soybean cyst nematode reproduction (Levene et al. 1998). The interaction between herbicide treatments and soybean canopy closure could also affect soybean production practices and weed control recommendations. A weed control timing that would also target white mold suppression

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Soybean restricts light penetration to the soil surface by forming a dense leaf canopy near the upper portion of a mature canopy. The upper 20% of the canopy intercepts 90% of the total photosynthetically active radiation and produces a majority of the plant's photosynthate, yet has only 30% of the total LAI (leaf area index) (Hatfield and Carlson 1978, Sakamoto and Shaw 1967). Light quality may stimulate weed seed germination or stem elongation of a crop or weed. Far-red light penetrates soybean canopies more readily than red light. Singh et al. (1968) reported a sharp peak of absorption with approximately 12 to 24% intensity in the infrared radiation region once the soybean canopy had closed. Phytochrome conversion from Pr (red) to Pfr (far-red) is necessary for the germination of some weed seeds. For instance, red light was related to redroot pigweed seed germination (Gallagher and Cardina 1998). A low level of red light (3 μmol m^{-2}) stimulated buried redroot pigweed seed germination in the laboratory (Gallagher and Cardina 1998).

Soybean cultivars vary in their competitiveness with weeds (Burnside 1972, Bussan et al. 1997). Bussan et al. (1997) reported that several high yielding cultivars in weed-free conditions were also high yielding in weed-infested conditions. Soybean canopy area and height were evaluated 30 to 45 days after planting, but there was no correlation between these parameters and the competitive ability of the cultivars evaluated (Bussan et al. 1997). Predicting the outcome of an interaction between a weed and crop species is difficult due to the numerous yield limiting factors involved in crop production and the weed species evaluated. Soybean cultivars may also differ in their photosynthetic

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rates (Johnston et al. 1969), leaf orientation (Blad and Baker 1972), nodulation efficiency (Hunt et al. 1990), and growth habits (Huang et al. 1993) as a result of differential light interception with respect to the quantity and quality of light intercepted. These differences may ultimately increase or decrease soybean yield and help the plant gain a competitive advantage over weeds.

Several producers have expressed concerns regarding the susceptibility of genetically modified soybean to postemergence herbicides other than glyphosate. Other studies have reported differential sensitivity between soybean cultivars to herbicides, but herbicide sensitivity was not linked to other herbicide resistance in the cultivar (Burnside 1972, Connelly et al. 1988, Dayan et al. 1996 and 1997, Griffin and Habetz 1989, Wax et al. 1974). Other research has evaluated acifluorfen and bentazon applied at the V3 (Levene et al. 1998) and V6 (Browde et al. 1994) stage of development and the interaction with nematodes. However, no research has evaluated how the soybean canopy would be affected when a herbicide treatment for weed control and white mold suppression was applied and the implications on late weed seed germination. The objective of this research was to evaluate the effects of glyphosate and postemergence herbicide tank mixtures, timed for weed control and white mold suppresion, on soybean vegetative and reproductive development, canopy development, and yield of glyphosate-resistant and non-resistant cultivars in a weed- and disease-free environment.

MATERIALS AND METHODS

Field research was conducted in 1997 and 1998 at the Bean and Beet Research Farm near Saginaw, MI (43° N, 83° W). The field was fall plowed and spring field

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cultivated in 1997. The soil was a Misteguay clay (Aeric Haplaquept, fine, mixed, mesic) with pH 7.9 and 3.7% organic matter. In 1998, the field was fall plowed and spring field cultivated twice. The soil was a Misteguay silty clay with pH 8 and 1.9% organic matter. The study was arranged in a split-plot design with four replications. Soybean cultivar was the main plot and the sub-plot was herbicide treatment. 'Asgrow 1900' (A1900), 'Asgrow 1901' (AG1901 Roundup Ready®), 'Asgrow 2701' (AG2701 Roundup Ready®), and 'Asgrow 2704' (A2704 sulfonylurea-tolerant) soybean were planted with tool-bar mounted International 185 (International Harvester Co., Chicago, IL) planter units in 38 cm rows at 350,000 seeds ha^{-1} on May 23, 1997 and May 12, 1998 in plots 2.7 by 12.2 m that were maintained weed-free.

Herbicide treatments included an untreated control, glyphosate (*N*-(phosphonomethyl)glycine) (formulated as Roundup Ultra®, Monsanto Co., St. Louis, MO) at 840 g ha^{-1} plus ammonium sulfate (AMS) at 20 g L^{-1}, bentazon/acifluorfen (3-(1-methylethyl)-(1*H*)-2,1,3-benzothiadiazin-4(3*H*)-one 2,2-dioxide)/(5-[2-chloro-4-(trifluoromethyl)phenoxy]-2-nitrobenzoic acid) (formulated as Galaxy®, BASF, Research Triangle Park, NC) at 1030 g ha^{-1} plus thifensulfuron (3-[[[[(4-methoxy-6-methyl-1,3,5-triazin-2-yl)amino]carbonyl]amino]sulfonyl]-2-thiophenecarboxylic acid) at 2.2 g ha^{-1} plus sethoxydim (2-[1-(ethoxyimino)butyl]-5-[2-(ethylthio)propyl]-3-hydroxy-2-cyclohexen-1-one) at 240 g ha^{-1} plus UAN (28% urea ammonium nitrate) and COC (crop oil concentrate) (Herbimax, paraffinic oil plus emulsifiers plus surfactants, Loveland Industries Inc., Greeley, CO) at 1.5% v/v, and lactofen ((±)-2-ethoxy-1-methyl-2-oxoethyl 5-[2-chloro-4-(trifluoromethyl)phenoxy]-2-nitrobenzoate) at 105 g ha^{-1} plus bentazon at

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1120 g ha^{-1} plus clethodim ((*E*,*E*)-(±)-2-[1-[[(3-chloro-2-propenyl)oxy]imino]propyl]-5-[2-(ethylthio)propyl]-3-hydroxy-2-cyclohexen-1-one) at 140 g ha^{-1} plus UAN and COC at 1.5% v/v. Herbicide treatments were selected that may affect leaf area and soybean height and had limited or no soil residual to allow weed seed germination. Herbicide treatments were applied with a tractor-mounted compressed-air plot sprayer equipped with 8003 flat-fan tips (Spraying Systems Co., Wheaton, IL) delivering 178 L ha^{-1} at 207 kPa and 6.3 km h^{-1}. Soybean were 23 cm tall and at the V5 growth stage (Fehr and Caviness 1977) at the time of application. Air temperature was 28 and 22 C, and relative humidity was 40 and 78% in 1997 and 1998, respectively.

Visual injury was estimated 7, 21, and 28 DAT. Injury symptoms included leaf necrosis, chlorosis, and soybean stunting which were rated on a scale of 0 (no effect) to 100% (crop death). Five photosynthetically active radiation light measurements were recorded in each plot with a one-m SunScan Canopy Analysis System (Dynamax Inc., Houston TX) perpendicular to the soybean row from the time of herbicide application until maturity to estimate soybean LAI. Incident and diffused light measurements have been utilized as an effective non-destructive method to measure soybean LAI (Walker et al. 1988). Three red:far red light (Skye-Probetech, Perkasie, PA) readings were measured between soybean rows with a single photocell 14 and 28 DAT. All light measurements were recorded at the soil surface at approximately 1230 h. Vegetative and reproductive stages were recorded according to Fehr and Caviness (1977) for three randomly sampled plants in each plot. Dry weights were measured for one m of soybean row prior to herbicide application, 35, 56, and 77 DAT according to Hunt et al. (1987). Soybean were

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Data were subjected to analysis of variance and means separated using Fisher's Protected LSD ($p \leq 0.05$). Data were combined over years and main effects presented where interactions were not observed.

RESULTS

Glyphosate did not injure glyphosate-resistant soybean (Table 1). Soybean treated with glyphosate had similar vegetative development, reproductive development, leaf area index, dry weight, height, and yield compared with the untreated control; therefore, glyphosate data is not presented in Table 2 or Figures 1, 2, and 3.

Soybean injury was 17% from lactofen + bentazon + clethodim and 14% from bentazon/acifluorfen + thifensulfuron + sethoxydim 7 DAT (data not presented). By 21 DAT, soybean injury was 14 to 18% from bentazon/acifluorfen + thifensulfuron + sethoxydim and lactofen + bentazon + clethodim (Table 1). AG1901 and AG2701 injury from bentazon/acifluorfen + thifensulfuron + sethoxydim and lactofen + bentazon + clethodim was greater than injury to A2704. This could be due to increased tolerance of A2704 to components of the postemergence herbicide treatments (Sebastian et al. 1989, Simpson and Stoller 1996).

The red:far red light was greatest at the soil surface in the absence of a crop (glyphosate treatment of non-resistant cultivars A1900 and A2704) (Table 1). By 28 DAT, the red:far red light at the soil surface beneath the canopy of A1900, AG1901 and A2704 treated with bentazon/acifluorfen + thifensulfuron + sethoxydim and A2704 treated

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with lactofen + bentazon + clethodim was similar to the untreated control. There was no difference in the red:far red light at the soil surface 14 and 28 DAT in the untreated control of each cultivar. Such effects on light quality below the canopy were found at high plant populations where the far red:red light was greater compared to the ratio below the canopy of low populations (Burkey and Wells 1991). The red:far red light was similar for all cultivars treated with bentazon/acifluorfen + thifensulfuron + sethoxydim 14 and 28 DAT. An application of lactofen + bentazon + clethodim to A1900 and AG1901 may create an environment that is favorable for weed seed germination since the red:far red light was greater in these treatments 28 DAT compared to the untreated control.

Soybean response to herbicides, as measured by LAI, was influenced by cultivar (Figure 1). At 40 and 52 DAT, LAI was greatest in the untreated control followed by bentazon/acifluorfen + thifensulfuron + sethoxydim and lactofen + bentazon + clethodim (data not presented). Canopy development was delayed by these herbicide treatments for A1900 and A1901 70 DAT and for A1900, AG2701, and A2704 80 DAT. Delayed development resulted in slower maturity which had characteristically later leaf abscission. The untreated control of A1900 and AG1901 reached a LAI equal to the peak LAI of soybean treated with the postemergence tank mixture treatments approximately two weeks prior to these cultivars treated with postemergence herbicide tank mixtures. The untreated control of AG2701 and A2704 reached a LAI equal to the peak LAI of soybean treated with the postemergence tank mixture treatments approximately one week prior to these cultivars treated with postemergence tank mixtures. AG2701 and A2704 had a larger LAI compared to A1900 and AG1901 from 24 DAT until harvest. In previous

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research, light interception by soybean cultivars was affected by postemergence herbicides (Ralston and Witt 1998).

Several researchers have evaluated light interception in the soybean canopy (Board and Harville 1992, Burkey and Wells 1991, Egli 1994, Neeser et al 1997, Wells 1991, Wells et al 1993). Much of the research has evaluated the utilization of narrow-row spacings to capture more light and thus reach the highest yield potential of soybean. However, weeds compete with soybean for light. Rapid soybean canopy development is important for soybean to be competitive with weed species. Rapid canopy closure reduces the reproductive potential of weed species through shading (Neeser et al. 1997, Keeley and Thullen 1978, Santos et al. 1997).

Soybean height was AG2701 = A2704 > AG1901 > A1900 48 DAT until harvest in the untreated control (Figure 2). Bentazon/acifluorfen + thifensulfuron + sethoxydim and lactofen + bentazon + clethodim applied postemergence to A1900 and AG1901 caused season-long stunting compared with the untreated control. AG2701 was also stunted until 35 DAT.

Bentazon/acifluorfen + thifensulfuron + sethoxydim and lactofen + bentazon + clethodim reduced soybean dry weight compared to the untreated control 35 and 56 DAT (Figure 3). By 77 DAT, there were no longer differences in dry weights between treatments due to earlier senescence in the untreated control plots. In other research, dry matter production was related to the intercepted radiation (Sinclair and Horie 1989, Shibles and Weber 1966). A linear relationship between the percent light interception and dry matter increase per day was positively correlated (Shibles and Weber 1965). Canopy

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development and light interception may be affected by the morphological traits of a soybean cultivar; however, increased yield was not always reported (Huang et al. 1993, Wells et al. 1993).

Bentazon/acifluorfen + thifensulfuron + sethoxydim and lactofen + bentazon + clethodim reduced the vegetative growth stage 7 DAT (Table 2), but by 14 DAT vegetative stage was not affected by herbicide treatment (data not shown). Herbicide treatments had limited effects on vegetative development. However, reproductive development was delayed from an application of bentazon/acifluorfen + thifensulfuron + sethoxydim or lactofen + bentazon + clethodim when compared to the untreated control during the transition between full flower and early pod (20 DAT) and physiological maturity (80 DAT).

The yield of each soybean cultivar differed each year and data is therefore presented for each year separately. Bentazon/acifluorfen + thifensulfuron + sethoxydim and lactofen + bentazon + clethodim reduced soybean yield from 130 to 270 kg ha^{-1} compared to the untreated control in 1997 and 1998. Soybean cultivar yield was A1900 = A2704 = AG2701 > AG1901 in 1997, and A2704 > AG2701 > A1900 > AG1901 in 1998. Rainfall in July and August totaled 9.5 inches in 1997 and 2.7 inches in 1998, and may be the primary factor that caused lower soybean yield in 1998.

DISCUSSION

Factors that affect canopy development and light interception may also reduce soybean yield. Light interception is influenced by the crop species, population (Shibles and Weber 1966), fertility (Flénet and Kiniry 1995), planting date (Board and Harville 1992),

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maturity group (Board and Harville 1992), growth stage (Luxmoore et al. 1971), insects (Board et al. 1997), row spacing and leaf orientation (Keeley and Thullen 1978, Baker and Meyer 1966), and leaf morphology (Egli et al. 1970, Wells et al. 1993). The effect of these factors on canopy development depends on the soybean growth stage. For instance, soybean that were 50% defoliated at R3 recovered 20 days later for maturity group IV soybean (Board and Harville 1993). Light interception before R5 was considered essential for soybean yield (Board and Harville 1993). Controversy regarding light interception during R1 to R5 or R5 to R7 and the effect on yield has been argued. Increased soybean yield in narrow-row soybean has been related to increased light interception in the early reproductive growth stages compared to wide-row cultures (Shibles and Weber 1965 and 1966, Board and Harville 1992, Hicks et al. 1969). However, Egli (1994) reported that soybean yield did not rely on increased light interception in the early reproduction stages. In our research, postemergence herbicides applied at the V5 stage of development, prior to flowering, affected season long canopy development and reduced yield. The degree of this effect may depend on the row-spacing, plant population, environmental conditions at the time of application, or the cultivar selection.

Soybean height was reduced and canopy development was reduced and delayed from bentazon/acifluorfen + thifensulfuron + sethoxydim and lactofen + bentazon + clethodim. Shorter beans could contribute to increased light penetration thus resulting in a smaller LAI as indicated in this research. The reduction and delay in soybean development was more evident for early maturing soybean (A1900 and AG1901) than late group 2 cultivars (AG2701 and A2704). Delayed soybean development may increase the risk of

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yield loss due to frost when producers grow late group 2 soybean in the northern latitudes. A1900 indicated quick canopy development in the untreated control which may make it very competitive with weeds early in the season when injury is not incurred. An altered canopy from herbicide injury also affected the red:far red light reaching the soil surface which may affect weed seed germination. An altered canopy could also affect the canopy microclimate and influence white mold development. Lactofen applied postemergence is currently labeled for white mold suppression (Anonymous 1998). A change in canopy development, in addition to the physiological effects (Dann et al. 1999, Levene et al. 1998), as a result of postemergence herbicides may be a factor in reducing white mold in a non-irrigated soybean culture.

Soybean treated with bentazon/acifluorfen + thifensulfuron + sethoxydim and lactofen + bentazon + clethodim had a more branched growth habit compared to the untreated control (personal visual observation). Soybean appeared to counteract herbicide injury and stunting by branching, but this did not completely compensate for the injury. The effect of light on canopy development may be observed in the branching characteristics of the soybean plant. For example, soybean planted in wide-row spacings branched more which resulted in increased light interception per plant (Shibles and Weber 1966). The percent main and branched reproductive dry matter in optimal and late planted soybean depended on the cultivar (Board et al. 1990). The lower portion of the soybean plant, below the dense cover of the soybean canopy, receives limited light. Shaded leaves may continue to photosynthesize when supplemental light is provided (Johnston et al. 1969). An open canopy could reduce the loss of abscised pods and flowers which could

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Soybeans have an incredible ability to recover from injury caused by postemergence herbicides. Several soybean growth characteristics are altered by a late application of postemergence herbicides for rescue weed control or timing for weed control and white mold suppression. A reduction in yield may occur when tank mixture treatments of bentazon/acifluorfen + thifensulfuron + sethoxydim or lactofen + bentazon + clethodim are applied at the V5 stage of development. However, at the benefit of white mold suppression and reduced weed seed production may justify such treatments.

IMPLICATIONS AND CONCLUSIONS

Soybean reproductive development was delayed by postemergence herbicide treatments, but vegetative development was not affected 7 DAT. LAI up to 52 DAT, height, and dry weight up to 56 DAT were reduced by postemergence herbicide tank mixture treatments and the degree of reduction was related to cultivar. The red:far-red light was lower in the untreated control compared to the postemergence herbicide treatments 28 DAT for certain treatments and cultivars. Soybean treated at the V5 stage of soybean development with bentazon/acifluorfen + thifensulfuron + sethoxydim or bentazon + lactofen + clethodim yielded less than the untreated control. Soybean that are not injured have more rapid canopy closure which reduces light quality and quantity at the soil surface. This is important to reduce late germinating weeds and potential weed seed production, and to maximize soybean yield potential. Changes in the time of maximum leaf area and canopy development may affect the microclimate in the canopy and the potential

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for white mold development and infection. Future research should evaluate the effects of postemergence herbicides on canopy development and the incidence of white mold.

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Table 2. Soybean injury 21 d after treatment, and red fir red light at the soil surface 14 and 28 d after treatment in 1997 and 1998.[a]

		Injury 21 DAT				Red Far red 14 DAT				Red Far red 28 DAT			
Treatment[b]	Rate	A1900	AG1901	AG2701	A2704	A1900	AG1901	AG2701	A2704	A1900	AG1901	AG2701	A2704
	(g ai ha^{-1})	%											

Table 1. Soybean injury 21 d after treatment, and red:far red light at the soil surface 14 and 28 d after treatment in 1997 and 1998.[a]

		Injury 21 DAT				Red:Far red 14 DAT				Red:Far red 28 DAT			
Treatment[b]	Rate	A1900	AG1901	AG2701	A2704	A1900	AG1901	AG2701	A2704	A1900	AG1901	AG2701	A2704
	(g ai ha^{-1})	%											
Untreated		0	0	0	0	0.35	0.33	0.36	0.34	0.09	0.10	0.08	0.09
Glyphosate + AMS	840 + 20 g L^{-1}	—	0	0	—	1.13[c]	0.30	0.40	1.08[c]	1.06[c]	0.11	0.09	1.07[c]
Bentazon/ acifluorfen + thifensulfuron + sethoxydim + UAN + COC	1030 + 2.2 + 240 + 1.5% v/v + 1.5% v/v	15	16	17	14	0.40	0.43	0.38	0.43	0.16	0.16	0.15	0.12
Bentazon + lactofen + clethodim + UAN + COC	1120 + 105 + 140 + 1.5% v/v + 1.5% v/v	17	18	18	16	0.51	0.58	0.54	0.44	0.22	0.21	0.16	0.15
$LSD_{(p\leq 0.05)}$		2				0.38				0.07			

[a]AG1901 and AG2701 were glyphosate-resistant soybean and A2704 was sulfonylurea tolerant soybean. Comparisons between cultivars are valid.

[b]Abbreviations: AMS, ammonium sulfate; COC, crop oil concentrate; UAN, 28% urea ammonium nitrate.

[c]These treatments were representative of no canopy present since these cultivars were not glyphosate-resistant.

Table 2. Soybean vegetative stage 7 d after treatment, reproductive stage 20 and 80 d after treatment, and yield in 1997 and 1998

Herbicide treatment[b]	*Rate*	Vegetative	Reproductive		Yield	
			20 DAT	80 DAT	1997	1998
	(g ai ha^{-1})		stage			

Table 2. Soybean vegetative stage 7 d after treatment, reproductive stage 20 and 80 d after treatment, and yield in 1997 and 1998.

			Reproductive		Yield	
Herbicide treatment[b]	Rate	Vegetative	20 DAT	80 DAT	1997	1998
	(g ai ha^{-1})	stage			kg ha^{-1}	
Untreated		7.4	2.4	7.3	3630	2960
Bentazon/acifluorfen + thifensulfuron + sethoxydim + UAN[c] + COC[d]	1030 + 2.2 + 240 + 1.5% v/v + 1.5% v/v	7.1	2.2	6.8	3430	2760
Bentazon + lactofen + clethodim + UAN + COC	1120 + 105 + 140 + 1.5% v/v + 1.5% v/v	6.9	2.1	6.7	3360	2830
$LSD_{(p\leq0.05)}$		0.3	0.2	0.2	130	130
Cultivar[c]						
A1900		7.1	2.5	7.3	3630	2690
AG1901		6.9	2.4	7.6	3160	2490
AG2701		7.5	2.3	7.0	3430	2960
A2704		7.0	2.0	6.4	3630	3100
$LSD_{(p\leq0.05)}$		0.4	0.2	0.6	270	130

[a]Abbreviations: AMS, ammonium sulfate; COC, crop oil concentrate; UAN, 28% urea ammonium nitrate.
[b]Herbicide treatment data was averaged over cultivar.
[c]Cultivar data was averaged over herbicide treatment. AG1901 and AG2701 were glyphosate-resistant soybean and A2704 was sulfonylurea tolerant soybean.

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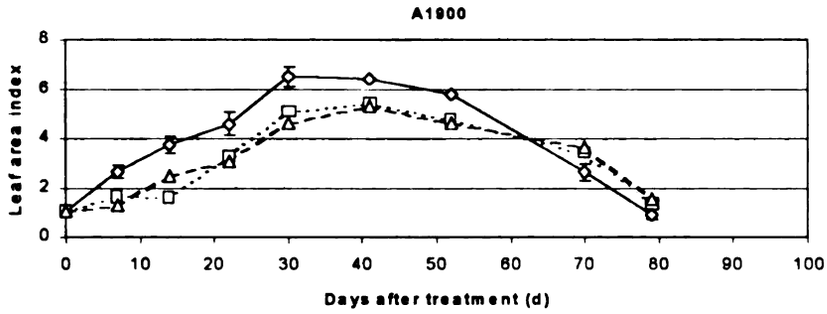

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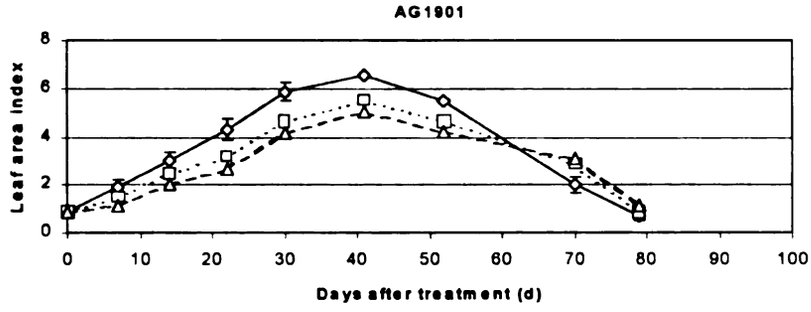

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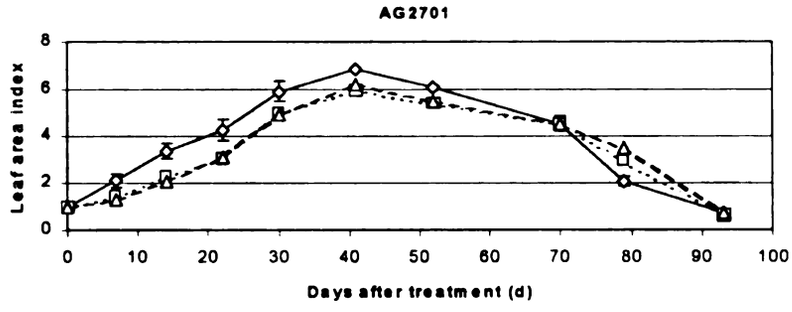

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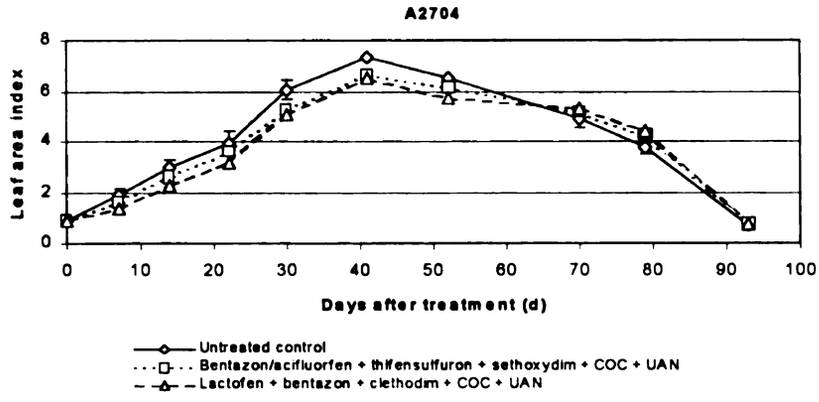

Figure 1. Leaf area index for each soybean cultivar in the untreated control, bentazon/acifluorfen at 1030 g ha^{-1} plus thifensulfuron at 2.2 g ha^{-1} plus sethoxydim at 240 g ha^{-1} plus UAN and COC at 1.5% v/v, and lactofen at 105 g ha^{-1} plus bentazon at 1120 g ha^{-1} plus clethodim at 140 g ha^{-1} plus UAN and COC at 1.5% v/v treatments combined over 1997 and 1998. Vertical lines indicate the LSD ($p \leq 0.05$).

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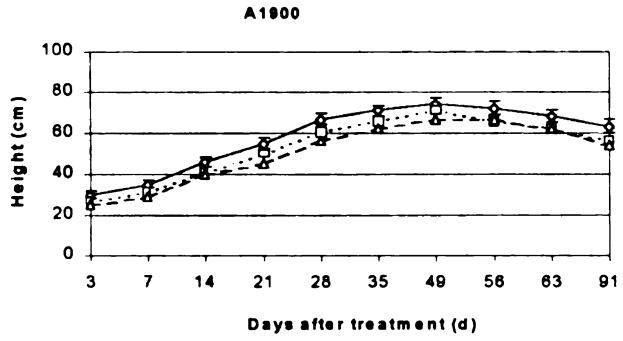

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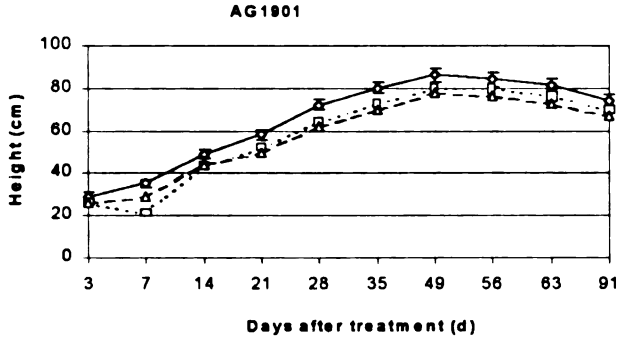

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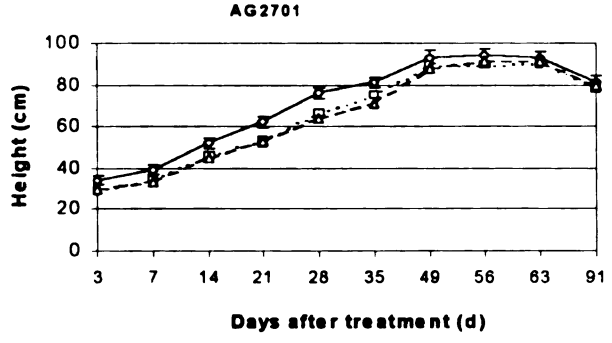

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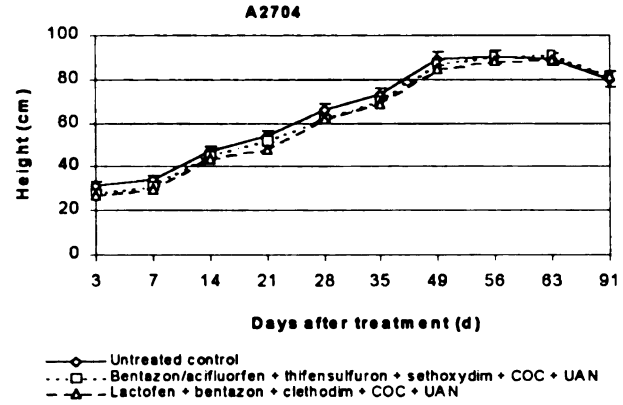

Figure 2. Height of each soybean cultivar in the untreated control, bentazon/acifluorfen at 1030 g ha^{-1} plus thifensulfuron at 2.2 g ha^{-1} plus sethoxydim at 240 g ha^{-1} plus UAN and COC at 1.5% v/v, and lactofen at 105 g ha^{-1} plus bentazon at 1120 g ha^{-1} plus clethodim at 140 g ha^{-1} plus UAN and COC at 1.5% v/v treatments combined over 1997 and 1998. Vertical lines indicate the LSD ($p \leq 0.05$).

Biomass (grams/m of row)

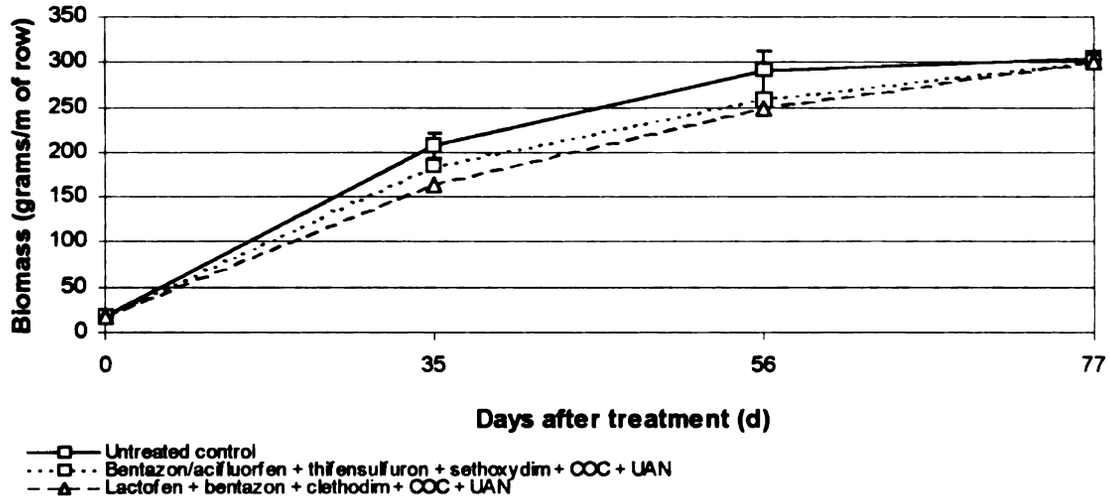

Figure 3. Soybean above ground biomass of the untreated control, bentazon/acifluorfen at 1030 g ha^{-1} plus thifensulfuron at 2.2 g ha^{-1} plus sethoxydim at 240 g ha^{-1} plus UAN and COC at 1.5% v/v and lactofen at 105 g ha^{-1} plus bentazon at 1120 g ha^{-1} plus clethodim at 140 g ha^{-1} plus UAN and COC at 1.5% v/v combined over cultivars and years. Vertical lines indicate the LSD ($p \leq 0.05$).

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CHAPTER 2

WHITE MOLD IN SOYBEAN LITERATURE REVIEW

Introduction and Disease Cycle of White Mold. A pathogen causes the physiological processes of the infected plant to deviate from normal. The interaction between a pathogen, compatible host plant, and suitable environment is essential for the infection, growth, survival, and persistence of the disease. The disease will not be expressed if there is an absence of or variation in one of these factors. In other instances, a vector is necessary for a disease to infect a plant, and without the vector the disease will not persist. The interaction between the host plant and the environment are necessary to cause stress in the plant or provide growing conditions that facilitate the infection and subsequent colonization of the disease. The environmental conditions must be conducive for inoculation, infection, and the development of the pathogen in the plant. Entry of a pathogen into a plant may take place through direct penetration through the cuticle, natural openings like stomata or abscised plant parts, vectors, or wounds. Finally, the pathogen must be genetically compatible to infect a plant. Particular methods to control diseases include protection, elimination, eradication, exclusion, and sanitation. A combination of cultural, genetic, and chemical methods may effectively suppress diseases like white mold caused by *Sclerotinia sclerotiorum* (Lib.) de Bary and thus reduce yield losses in crops like soybean [*Glycine max* (L.) Merr.].

White mold was first reported in the United States in 1946 (Chamberlain 1951) and is common in the North Central United States (OH, IA, IN, IL, NE, ND, MI, WI, and MN). World-wide, white mold has been reported in China, South America (Marinelli et al.

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1998), and Africa (Thompson and Van der Westhuizen 1979). Boland and Hall reported in 1982 that there was an increase of white mold occurrence in soybean in Ontario. The popular agriculture press has also reported that white mold was increasing in the United States. An Illinois survey in 1998 reported that 45% of the fields evaluated had white mold present (Hartman et al. 1998).

S. sclerotiorum is a soil-borne ascomycete fungus that causes wilting and premature death of Group 0 to Group III soybean in the regions mentioned above. *S. sclerotiorum* does not cause soybean seedling blight (Chamberlain 1951); however soybean death usually occurs prior to or during pod fill. *S. sclerotiorum* undergoes a simple life cycle (Figure 1 in color). The effects and importance of this disease depend heavily on the environmental conditions, adopted cultural methods, and cultivar selection.

The mycelium and sclerotia of *S. sclerotiorum* over-winter in the soil or on decaying organic matter. Sclerotia are found in fields with a history of white mold, surrounding fence rows, orchards, in nearby fields, and around low growing plants like dandelion (*Taraxacum officinale* Weber in Wiggers), white clover (*Trifolium repens* L.) (Abawi and Grogan 1975), or Canada thistle [*Cirsium arvense* (L.) Scop] (Bronsten and Sands 1986). Sclerotia are black aggregates of tightly packed mycelium covered by a rind for protection (Merriman 1976). Sclerotia are similar in appearance to mouse or rat feces and may survive several years in the soil (Coley-Smith and Cooke 1971) even after fumigation treatment (Merriman 1976). Sclerotia have contaminated soybean seed (Hartman et al. 1998, Hoffman et al. 1998) and can be distributed to other locations on machinery, by animals, or in the soil.

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Carpogenic germination of sclerotia results in the production of tan to brown apothecia that are 1 to 7 mm in diameter and resemble a small, frail golf-tee. A single sclerotia may produce over two apothecia in a growing season (Schwartz and Steadman 1978). The apothecia produce several million ascospores that are released from the asci for approximately nine d (Schwartz and Steadman 1978) and are carried by air currents to nearby plants. Ascospores germinate and form mycelium which produces oxalic acid (Godoy et al. 1990). Oxalic acid allows the growth and spread of mycelium in soybean tissue (Cline and Jacobsen 1983, Radke and Grau 1986). Most inoculum originates from the apothecia since no secondary inoculum has been reported which is unlike other important crop diseases (Boland and Hall 1982). In general, *S. sclerotiorum* ascospores infect soybean through and grow on senescent tissues like flower petals which provide a rich nutrient source and a location for easy penetration into the plant (Boland and Hall 1982, Boland and Hall 1987, Boland and Hall 1988a, Cline and Jacobsen 1983). Early research indicated the role and importance of oxalic acid in pathogenicity. Maxwell and Lumsden (1970) demonstrated that oxalic acid was produced by *S. sclerotiorum* and increased in bean (*Phaseolus vulgaris* L.) after infection depending on the isolate and culture media. Later, Marciano et al. (1983) compared strong and weakly virulent *S. sclerotiorum* isolates. High oxalic acid production by the strongly virulent isolate indicated an important role in virulence. However, the role of oxalic acid as a pathogenicity factor was confirmed with mutants of the fungus (Godoy et al. 1990). Mutants that did not produce oxalic acid, but were capable of producing pectinase and

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cellulase, were not pathogenic (Godoy et al. 1990). Oxalic acid kills plant tissue and allows the pathogen to penetrate and overcome bean tissue.

Field variability of disease incidence has been related to the number and location of apothecia in the field and environmental conditions that affect apothecia production and development. There has been a strong relationship between the apothecia number and the incidence of white mold in the field (Boland and Hall 1982, Boland and Hall 1988b, Phipps 1983). Apothecia at a New York location were produced from April 23 to June 15, and in a controlled environment apothecia formation was greatest at 11 to 15 C (Abawi and Grogan 1975). However, dried sclerotia produced fewer apothecia (Abawi and Grogan 1975). Ascospore germination produced by apothecia was 95% at 5 to 30 C, but the fastest germination, mycelial growth, germ tube growth, greatest sclerotia production, and best lesion development with bean was at 25 C (Abawi and Grogan 1975). These factors may help explain the variability of white mold development in the field.

Infection by *S. sclerotiorum* usually occurs after the soybean canopy has closed, during flowering (Cline and Jacobsen 1983), and when the leaf or plant surface was wet for 40 to 112 h (Abawi and Grogan 1975, Boland and Hall 1982, 1988a). In a growth chamber at 20 C, a leaf wetness of 70 to 120 h was adequate for white mold infection and growth (Boland and Hall 1988a). Infection of bean took place where an old blossom was attached, where a cast blossom laid on the leaf, or where mechanical injury to the plant was observed (Abawi and Grogan 1975, Boland and Hall 1982). In addition, the microclimate in the canopy was considered an important factor influencing disease

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susceptibility of bean (Blad et al. 1978). Other conditions that were favorable for infection included fields where corn surrounded the crop or irrigation was utilized for growing soybean (Fuller et al.1984).

A typical sign of *S. sclerotiorum* disease develops on the lower portion of the soybean stem which becomes covered with a "white mold" that is cottony in appearance. The fungus girdles the stem and may infect the petioles, branches, pods, or leaves under wet conditions. Lesions that originate from the leaf axils at nodes 3 and 4 turned from a grey to tannish color, and develop up and down the stem (Cline and Jacobsen 1983, Boland and Hall 1982, Grau et al. 1982). Later, sclerotia, black and cylindrical in appearance, are produced in the white mycelium both in and on the stem. White mold symptoms generally develop approximately 15 to 40 cm above the soil surface in July or August during the R1 to R2 stage of development, and from R3 to R5 the disease has made significant growth on the plant (Boland and Hall 1988a, Grau et al. 1982, Phipps 1983). Infected soybean plants first appear grey-green and wilted, and then turn necrotic. Disease incidence is visible by mid-August to mid-September (Boland and Hall 1988a). In the fall, the stems appear bleached where the mycelium was present which is characteristic of a plant infected with white mold and sclerotia pepper the surface of the stem. Table 1 provides a summary of the environmental and soybean growth characteristics that influence the infection and incidence of white mold in the field and greenhouse.

The white mold pathogen may infect soybean seed (Hartman et al. 1998, Hoffman et al. 1998, Thompson and Van der Westhuizen 1979, Yang et al. 1998) or over-winter as sclerotia in the soil which may survive for several seasons. Fields with white mold may

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contaminate seed with sclerotia since combines may not completely separate all of the sclerotia from the grain. Infected seed may produce mycelium from shriveled seed (Thompson and Van der Westhuizen 1979) and in normal appearing seed (Hartman et al. 1998). Infected seed may transfer white mold to an uninfected field (Hoffman et al. 1998, Yang et al. 1998). It is important to plant seed that is not contaminated with sclerotia, clean machinery between fields where white mold is known to exist, and to be aware of the spread by animals or other environmental methods.

Evaluating White Mold: Disease Severity Index. Several researchers have developed and defined indexes to rate the disease severity of white mold in soybean. The DSI (disease severity index) rating characterizes the extent or type of symptom exhibited by the plant (Table 2). DSI's have been determined during the R6 to R8 stage of soybean development (Boland and Hall 1988a). The classes evaluated are defined by the researcher and may vary depending on the researcher (Table 2). A scale from 0 to 3 where 0 = no disease, 1 = lesions on lateral branches only, 2= lesions on the main stem and no effect on pod fill, and 3 = lesions on the main stem with plant death and poor pod fill has been utilized in several field studies to determine DSI (Dann et al. 1998, Dann et al. 1999, Grau et al. 1982, Kim et al. 1999). The following formula was used to calculate the DSI:

$$DSI = \left[\frac{\sum(\text{ratings of each plant})}{3 \times \text{total number of rated plants}} \right] * 100$$

Several greenhouse assays have been developed to evaluate and screen soybean cultivar sensitivity to white mold. Cline and Jacobsen (1983) evaluated carrot, ascospore,

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and limited term inoculation using celery petiole sections for inoculation. Limited term inoculation was the most successful method for identifying differences in soybean susceptibility (Cline and Jacobsen 1983). The measurement of lesion length by an oxalic acid treatment and soluble stem pigment levels may be additional method to evaluate soybean tolerance to white mold (Wegulo et al. 1998). Limitations in the greenhouse prevent researchers from evaluating and factoring in the effects of lodging, canopy architecture, height, flowering characteristics, and maturity which may be simple avoidance mechanisms that help reduce the incidence of white mold. Since light is less intense in a greenhouse, soybean stems do not grow as large which could affect cultivar sensitivity to white mold; therefore, there were limited correlations between field and laboratory experiments (Nelson et al. 1991b). Nonetheless, Pennypacker and Risius (1999) argued that PAR levels may have an effect on correlations between the greenhouse and field. Care is advised when using these methods since greenhouse and laboratory assays don't always indicate field resistance.

Effect of White Mold on Soybean Yield. Numerous factors, like disease, affect soybean yield. Diseases may completely destroy a crop or reduce yield to an unprofitable margin. *S. sclerotiorum* causes premature soybean death which results in the production of shriveled pods with little or no seed. Soybean cultivars may differ in white mold tolerance, but yield losses may not be realized under low levels of infestation due to yield compensation by nearby soybean plants.

Several researchers have determined the relationship between white mold disease severity or incidence and soybean yield. Grau and Radkey (1984) reported that there was

a negative correlation between the incidence of white mold or disease severity and soybean yield. Chun et al. (1987) reported that for every 10% increase in disease incidence from 0 to 52%, soybean yield was reduced 7.8% (235 kg ha^{-1} or 3.5 bu A^{-1}) in Michigan. At a maximum level of disease infestation (52%), soybean yield was reduced more than 40% (Chun et al. 1987). Hoffman et al. (1998) determined that for every 10% increase in disease incidence in group III soybean in Illinois, yield was reduced 147 to 263 kg ha^{-1} (2 to 4 bu A^{-1}) depending on the cultivar. Similarly, Yang et al. (1999) reported a 170 to 335 kg ha^{-1} (2.5 to 5 bu A^{-1}) yield loss for every 10% increase in disease incidence in Northern Iowa (r^2 = 0.59 to 0.83). Finally, for every 10% increase in disease severity, yield loss totaled 370 kg ha^{-1} (5.5 bu A^{-1}) in Michigan (Kim et al. 1999). We can conclude that yield reductions by white mold may range from 147 to 370 kg ha^{-1} (2 to 5.5 bu A^{-1}) for every 10% increase in disease severity depending on the environment and cultivar.

Narrow-row soybean usually yield more than or equal to wide-row soybean due to greater light interception in the absence of disease (Shibles and Weber 1965 and 1966, Board and Harville 1992, Hicks et al. 1969). Narrow-row soybean canopy closure was also reported earlier than wide-row soybean in other research which benefits weed control (Burnside and Colville 1964; Mickelson and Renner 1997; Wax and Pendleton 1968). However, greater yield reductions by white mold may occur in narrow- compared to wide-row soybean due to an environment that encourages white mold development. For instance, soybean yield was lower in narrow-row (25 to 38 cm) compared to wide-rows (76 cm) due to increased disease incidence in the narrow-row soybean (Grau and Radkey 1984). Therefore, the environment in narrow-row soybean may be more conducive to the

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carpogenic germination, infection, and growth of *S. sclerotiorum*. White mold disease is promoted by a cool, wet environment that is typical of narrow-row soybean since air movement and sunlight penetration is limited. However, narrow-row soybean don't always yield less than wide-row soybean in the presence of white mold. In a study conducted by Buzzell et al. (1993), soybean yields were greater in 23 compared to 45 cm soybean rows and there was no difference in disease severity between 23 and 69 cm rows. 'Essex' and 'Forrest' soybean cultivars infested with white mold planted in 25 cm rows yielded more compared to soybean planted in 76 cm rows (Phipps 1983). Similarly, narrow-rows (25 cm) had higher yields than wide-rows (50, 75, or 100 cm) two of three years when infested with *Rhizoctonia solani* (Joye et al. 1990). Soybean cultivars may have a higher incidence of disease in narrow-rows, but a reduction in yield is not always observed due to the yield compensation of nearby soybean plants. The effect of row spacing may depend on the adaptability of the cultivar to a wide- or narrow-row culture.

Several studies have reported the contamination of seed by mycelium and sclerotia as mentioned earlier, but it is important to reiterate as contaminated seed may encourage the spread of this disease. Contaminated soybean seed may produce white mold mycelium from shriveled seed of infected plants (Thompson and Van der Westhuizen 1979, Hartman et al. 1998, Hoffman et al. 1998, Nicholson et al. 1978). In addition, sclerotia were reported in 88% of the lots evaluated by Hartman et al. (1998). Seed-borne white mold incidence ranged from 0.3 to 0.7% depending on the cultivar (Hoffman et al. 1998). Seed quality, oil, and germination were usually reduced as the incidence of disease increased (Hoffman et al. 1998). White mold may not only reduce yield, but also soybean quality

and could spread the disease to non-contaminated fields via contaminated seed. This is important for producers to consider when bin-run seed is planted the following year.

Soybean Response to *S. sclerotiorum* and Phytoalexin Production. A plant must detect the presence of a pathogen before it can initiate an active defense response. A plant may undergo a hypersensitive response after it has detected the presence of a pathogen (Baker and Orlandi 1995, Goodman and Novacky 1994, Mehdy 1994, Sutherland 1991). This results in the rapid localized death of the host plant tissue around the infected area. A hypersensitive response is often coordinated with the activation of other defense mechanisms in a plant (Baker and Orlandi 1995, Goodman and Novacky 1994, Mehdy 1994, Sutherland 1991). For instance, the plant cell membranes at the infected area may break down and release of toxic substances from the vacuole (Goodman and Novacky 1994, Mehdy 1994, Sutherland 1991). In addition, phenolic oxidases are produced and utilized to synthesize quinones from phenols (Nicholson and Hammerschmidt 1992). Phenols are precursors of lignin which is utilized to seal off an infection. In surrounding cells, PR-proteins (pathogenesis related proteins) and phytoalexins are synthesized (Goodman and Novaky 1994, Nicholson and Hammerschmidt 1992, Mehdy 1994). Phytoalexins are antimicrobial compounds produced by plants after disease infection or treatment with biotic or abiotic elicitors (Hammerschmidt 1999). The role of phytoalexins and phenolic compounds in defense have been reviewed (Nicholson and Hammerschmidt 1992, VanEtten et al. 1989) and have been identified in numerous plant species. The interaction between the hypersensitive response and PR-proteins, peroxidase, and phytoalexin production helps to contain and combat an invading pathogen.

Oxalic acid is produced by white mold as a pathogenicity factor (Godoy et al. 1990). After soybean was inoculated with *S. sclerotiorum*, browning of the epidermal cells within 24 h resembled a hypersensitive response (Sutton and Deverall 1984). Several phytoalexins have been identified in *Glycine* spp. which are good indicators of disease expression (Table 3). Ingham (1982) reported that *Glycine max* produced up to twelve phytoalexins including four isomers of glyceollin. Soybean phytoalexins were produced in response to diseases, nematodes, herbicides, or external stimuli to whole plants, excised plants, and cell suspensions (Table 4). Methods to isolate, purify, separate, and detect glyceollin and other phytoalexins were reviewed by Ingham (1982). Phytoalexins have been isolated from soybean tissue using several techniques, but the separation of phytoalexins using thin-layer chromatography (TLC) and a chloroform:acetone:NH_4OH (50:50:1) mobile phase was summarized in Figure 2 (Ingham et al. 1981). An antifungal bioassay *in situ* with *Cladosporium cucumerinum* was also utilized to identify phytoalexins on the TLC plates (Keen et al. 1971).

Glyceollin is an isoflavonoid pterocarpan phytoalexin. Soybean foliage (200 g) may produce 5 to 10 mg of glyceollin I and 2 to 5 mg of glyceollin III (Giannini et al. 1991). Near-isogenic lines of 'Harosoy' (susceptible) and 'Harosoy 63' (resistant) have been evaluated for glyceollin production in the hypersensitive response to *Phytophthora megasperma* infection (Yoshikawa et al. 1978) and several other studies (Table 4). However, when other cultivars were evaluated, glyceollin was not considered the reason for differential tolerance to *Phytophthora megasperma* (Olah et al. 1985). Nonetheless, several correlative studies have reported the importance of glyceollin in the resistance of

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'Harosoy 63' to *Phytophthora megasperma* (Table 4). Similarly, glyceollin I accumulated in a nematode resistant soybean cultivar 8 h after penetration while the susceptible cultivar had minimal glyceollin production (Huang and Barker 1991). These studies have indicated that soybean phytoalexins like glyceollin may play an important role in disease resistance.

The mode of action of glyceollin (isomers I, II, and III) was evaluated by Kaplan et al. (1980). Glyceollin inhibited electron transport, but did not affect oxidative phosphorylation in isolated soybean mitochondria (Boydson et al. 1983, Kaplan et al. 1980). The inhibition of electron transport was associated with the inner membrane of the mitochondria (Boydson et al. 1983). Glyceollin inhibited proton transport by the plasmalemma (ED_{50} of 50 μM) at a lower rate than the plasma membrane (ED_{50} of 80 μM) of red beet (*Beta vulgaris* L.) (Giannini et al. 1988). The effects of glyceollin on ATPase may affect the transport of solutes across the cell membrane (Giannini et al. 1988). The isomers of glyceollin were compared to rotenone (Kaplan et al. 1980), a common insecticide and piscicide produced from the roots of the bean family, and mode of action similarities were reported by Boydson et al. (1983).

Favaron et al. (1988) reported that glyceollin was produced as a result of polygalacturonase (PG) activity by *S. sclerotiorum*. Three endogenous and one exogenous PG were produced by *S. sclerotiorum* (Favaron et al. 1988). PG cleaves pectin molecules hydrologically. Both PG II and IV have been isolated during *S. sclerotiorum* infection (Favaron et al. 1993). This interaction could be significant since glyceollin inhibits white mold ascospore germination and hyphae growth (Sutton and Deverall 1984). If there was

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a high accumulation of glyceollin, *S. sclerotiorum* mycelial growth could be inhibited. However, as much as 200 μg glyceollin ml^{-1} was required to inhibit white mold mycelium growth *in vitro* (Sutton and Deverall 1984).

Methods to Control White Mold. Integrated methods to manage white mold are currently the most practical methods to reduce the prevalence of this disease. Crop rotation, plant population, tillage, chemicals, cultivar selection, row spacing, and effective weed control may help reduce the incidence of white mold (Table 1) and yield loss caused by white mold. Planting white mold tolerant or resistant soybean cultivars is the recommended strategy to reduce yield loss. In addition, avoiding cultivars with parentage from 'Williams' or 'Asgrow A3127', which are both susceptible to white mold and had higher DSI than cultivars that did not have these genotypes in their parentage, may help also reduce the incidence of this disease (Kim et al. 1999). Table 5 summarizes resistant soybean cultivars that have been reported in the literature in the field or greenhouse.

Disease escape means like canopy architecture, maturity, plant height, flowering, row spacing, and plant population may affect the incidence of white mold for a given cultivar in the field (Table 1). For instance, taller plants may lodge more due to excessive vegetative growth which was correlated with an increase in the incidence of white mold (Buzzell et al. 1993). Buzzell et al. (1993) recommended planting early maturing cultivars that are tolerant to lodging to reduce white mold infestations. Lodging has been correlated with disease severity (Kim et al. 1999), but white mold can be the cause of increased lodging rather than the result. In addition, cultural practices may help escape white mold problems in the field. Irrigation timing is critical to reduce white mold. Soybeans that

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were irrigated before and after flowering had higher yields than soybean irrigated season long (Grau and Radkey 1984). In addition, wide-row soybean may reduce or prevent extended leaf wetness periods needed for white mold growth and development.

Several studies have evaluated canopy development in dry bean that may be useful for soybean producers and researchers. Canopy development influenced the density of apothecia which resulted in a larger number of apothecia produced in the row compared to between the row (Schwartz and Steadman 1978). A dense canopy was cooler, wetter, and had more disease than an open canopy (Blad et al. 1978). Compact bean plants produced by TIBA (2,3,5-triiodobenzoic acid) treatment were more susceptible to *S. sclerotiorum* than trellis-grown plants (Coyne et al. 1974). Flowering characteristics (determinate vs. indeterminate) also influenced disease severity in bean (Schwartz et al. 1978). Determinate beans and wide- (76 cm) rows were less susceptible to white mold than a near-isogenic indeterminate line and a narrow-row (25 cm) bean culture (Steadman et al. 1973). Fuller et al. (1984) reported that the incidence of white mold on bean cultivars was greatly affected by the adjacent row and the growth characteristics of the bean in that row. Thus, avoidance mechanisms for white mold may not be effectively evaluated when single rows are evaluated in dry edible bean cultivar comparisons. We can conclude that such changes in the canopy development may be useful in reducing the incidence of this disease in soybean.

A combination of tillage and crop rotation may reduce the incidence of white mold. Abawi and Grogan (1975) reported that plowed fields had fewer sclerotia; however, the sclerotia buried below the soil were still viable. Limited research on the

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effects of tillage on white mold development has been conducted or reported; however, sclerotia generally germinate near the soil surface. *S. sclerotiorum* has a wide crop host range (Table 6) and is often greater when susceptible or host species such as soybean, lettuce (*Lactuca sativa* L.), alfalfa (*Medicago sativa* L.), red clover (*Trifolium pratense* L.), celery (*Apium graveolens* L.), crown vetch (*Coronilla varia* L.) (Marinelli et al. 1998, Maxwell and Lumsden 1970), bean (Abawi and Grogan 1975), peanut (*Arachis hypogaea* L.) (Marinelli et al. 1998, Phipps 1982), or sunflower (*Helianthus annuus* L.) (Köhler and Friedt 1999, Marciano et al.1983, Marinelli et al. 1998) are grown in a crop rotation with soybean. In a rotational year, planting a non-host crop in no-till while the use of deep tillage to bury sclerotia prior to planting soybean may help reduce infestations. However, a three-year crop rotation in bean had no effect on the sclerotia in the field even though germinated apothecia were observed in sugar beet in the rotation year (Schwartz and Steadman 1978). Some weeds such as pigweed spp. (*Amaranthus* spp.), common ragweed (*Ambrosia artemisiifolia* L.), mustard (*Brassica* spp.), velvetleaf (*Abutilon theophrasti* Medicus), common lambsquarters (*Chenopodium album* L.), and Canada thistle (Bronsten and Sands 1986) may also serve as hosts for white mold when a non-host crop is planted in the rotation. Research has evaluated white mold as a biocontrol agent for Canada thistle in Montana (Bronsten and Sands 1986), but the use in crop production is limited due to the susceptibility of several crops to the disease. Nonetheless, the control of host weeds in a rotational crop may be important to help reduce the sclerotia in the soil for years when soybean are grown. A reduction in diseased soybean plants in the field has reduced the number of sclerotia m^{-2} (Buzzell et al. 1993). The management of white mold

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sclerotia is similar to managing the weed seed bank in grain crops.

The application of fungicides for white mold control is costly and a single application has met with limited success. The use of a fungicide like thiophanate methyl (Topsin®)[1] may control white mold in soybean (Crop Protection Chemicals Reference 1997). Topsin should be applied from R1 to R2 and a subsequent application may be made one to two weeks later (Crop Protection Chemicals Reference 1998). Similarly, an application of benomyl (Benlate®)[2] has been utilized in white bean to control white mold (Natti 1971). In other crops, several fungicides have been utilized to control *Sclerotinia* spp. (Table 4). Due to the cost of these products, their use has not been justified on a large scale in soybean. In addition, several applications of the systemic induced resistance activator 2,4-dichloroisonicotinic acid or benzothiadiazole reduced the disease severity of white mold in susceptible ('Williams 82') cultivars compared to tolerant ('S 19-90') cultivars (Dann et al. 1998).

Effects of Herbicides on Diseases and White Mold. A herbicide may increase, decrease, or have no effect on a disease depending on the herbicide mode of action, application timing, and application rate. Most of the research conducted and reported has focused on soil-applied herbicides. In Altman and Campbell's (1977) review of the effect of herbicides on plant disease, only seven postemergence herbicides were reported. Since 1977, numerous postemergence herbicides have been discovered, introduced, and utilized

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for weed control in soybean and other agronomic crops, and the recent introduction of herbicide-resistant crops has allowed new opportunities and options for weed control in these crops. The effects of glyphosate on several plant fungi and the interaction between the plant and fungus were reported and variable responses were observed (Black et al. 1996, Johal and Rahe 1984, Lévesque and Rahe 1992, Rahe et al. 1990). Lévesque and Rahe (1992) recently reviewed the interaction between glyphosate and plant pathogens. Glyphosate prevented phaseollin production by bean (Johal and Rahe 1990) and glyceollin production by soybean at sub-lethal rates, but did not affect hypersensitive cell death (Holliday and Keen 1982). Similarly, glyphosate inhibited the synthesis of medicarpin of lucerne (*Medicago sativa* L.) when challenged with *Verticillium albo-atrum* (Latunde-Dada and Lucas 1985), and enhanced mycelia growth of *Alternaria cassiae* on leaves of of sicklepod (*Cassia obtusifolia* L.) (Sharon et al. 1993). Variable results on the incidence of disease have been reported when glyphosate was applied to glyphosate-resistant (Lee and Penner 1999, Penner et al. 1997, Sanoto et al. 2000) and non-resistant soybean (Holliday and Keen 1982, Keen et al. 1982, Sanogo et al. 2000) in controlled environments. Several studies with glyphosate involved non-glyphosate resistant soybean cultivars and no research has reported the effects of glyphosate on glyceollin production with glyphosate-resistant cultivars. However, no effect on *S. sclerotiorum* was reported in the field or greenhouse when glyphosate was applied to 'Asgrow 2701' or 'GL 2600' soybean (Lee and Penner 1999, Penner et al. 1997).

The diphenyl ether herbicides have directly affected the incidence of white mold (Dann et al. 1999) and phytoalexin production by soybean (Dann et al. 1999, Kömives and

Cassida 1983, Levene et al. 1998). Acifluorfen increased phenylalanine amonia-lyase activity in soybean which was followed by tissue necrosis in spinach (Kömives and Cassida 1982). The aromatic amino acids, phenylalanine or tyrosine, are precursors of secondary metabolites that were related to the observed phytotoxicity (Kömives and Cassida 1982). Phytotoxicity caused by the herbicide was needed to produce these secondary metabolites in soybean (Kömives and Cassida 1983). Acifluorfen at 5 ppm induced production of glyceollin (79 ppm) and glyceofuran (16 ppm) in soybean (Kömives and Cassida 1983). The quantities of glyceollin I, II, and III in soybean seedlings following treatment with 5 ppm acifluorfen were 7, 19, and 38 $\mu g\ g^{-1}$ fresh weight, respectively (Kömives 1983). In other research, V4 soybean treated with lactofen increased glyceollin production by 8.3 to 10.2 $\mu g\ g^{-1}$ in 'Williams 82' and 'S 19-90' 4 DAT as the rate of lactofen increased from 0.04 to 0.11 kg ha^{-1} (Dann et al. 1999). 'S 19-90' had higher levels of glyceollin when treated with lactofen compared to 'Williams 82' (Dann et al. 1999). An application of lactofen to soybean not resistant to glyphosate at R1 reduced white mold disease severity and increased yield under high infestations; however, under low infestations yield was reduced by lactofen (Dann et al. 1999). Soybean leaves in this study were harvested 3 DAT, 4 DAT, and 6 WAT and therefore researchers could not determine if glyceollin levels peaked and were subsequently reduced or remained high during white mold infection. Similarly, when acifluorfen, lactofen, bentazon, COC, or NIS (X-77) were applied at the V3 stage of development soybean cyst nematode (*Heterodera glycines* Ichonohe) egg production was reduced 50 to 60% 8 weeks after application, and acifluorfen, bentazon, COC and NIS increased glyceollin levels 1.3 to 1.9 fold in the root

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(Levene et al. 1998). Fluazifop, sethoxydim, and imazethapyr did not affect egg production, and lactofen had no effect on glyceollin levels in the root (Levene et al. 1998).

Herbicides may act directly or indirectly on the growth and infectious ability of a disease on a plant species. Foliar blight of soybean by *Rhizoctonia solani* Kühn AG-1 IA was reduced with alachlor, paraquat, and glufosinate more than with pendimethalin, acifluorfen, or glyphosate (Black et al.1996). However, Duncan and Paxton (1981) reported a synergistic effect between *Phytophthora* root rot and trifluralin on soybean. Similarly, Millhollon and Koike (1985) reported reduced sugarcane (*Saccharum officinarum* L.) yield when plants were injured by hexazinone and sugarcane mosaic virus or ratoon stunting disease (*Clavibacter xyli*). The presence of a pathogen in the soil may aid the herbicide in controlling certain plant species by predisposing the plant to injury which allows the invasion of the disease. Lévesque and Rahe (1992) reported a ten-fold difference in LD_{50} values for glyphosate applied to plants grown in raw compared with autoclaved soils. Higher rates of glyphosate were needed on soils with low fungal levels (Lévesque and Rahe 1992).

The physiological effects of herbicides on white mold are not completely understood. The effects of several herbicides on the incidence of *Sclerotinia* spp. has been summarized (Table 7). Radke and Grau (1986) discussed the possible effects of the herbicide mode of action of several herbicides including the triazines on the carpogenic germination of white mold. Simazine and atrazine affected apothecia development, but metribuzin had no effect (Radke and Grau 1986). However, cultural methods like

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moldboard plowing reduced *Phytopthora* root rot more than herbicide use (chloramben, sethoxydim, and metribuzin) (Lévesque and Rahe 1992).

Other reviews have summarized the effect of herbicides on diseases (Altman and Cambell 1977, Katan and Eshel 1973, Lévesque and Rahe 1992). Disease resistance and herbicide resistance is desirable in crop species due to the advantages for crop protection and improved yields. Herbicides may stimulate resistance to disease by preventing disease infection (Uchimiya et al. 1993), or a herbicide may stimulate phytoalexin production after the herbicide is applied (Dann et al. 1999, Levene et al. 1998). Similarly, weeds that were atrazine resistant biotypes of *Epilobioum ciliatum* and *Senecio vulgaris* were also resistant to powdery mildew (*Sphaerotheca epilobii* [Wallr.] Sacc.) and *Epilobium cilatum* var. *fischeri* [Blumer] Braun (Clay et al. 1991). However, herbicide resistant *Lolium rigidum* also had the toxicant *Clavibacter toxicus* present in the grass (Riley and Gill 1994). Linkage between herbicide resistance and disease susceptibility in released crop cultivars could have detrimental effects on crop yield.

Postemergence herbicides, such as thifensulfuron, lactofen, and acifluorfen, cause chlorosis, necrosis, or stunting of soybean (Hart and Roskamp 1998, Kapusta et al. 1986, Wichert and Talbert 1993). Such herbicide injury may persist in weed-free conditions up to 21 d after treatment yet result in no yield loss (Kapusta et al. 1986). However, at low levels of white mold, lactofen reduced soybean yield when applied for white mold suppression (Dann et al. 1999). The use of postemergence herbicides may alter the canopy and delay canopy closure such that late germinating weeds or difficult to control weeds may escape control (Mickelson and Renner 1997, Nelson and Renner 1999).

The leaf area index is the ratio of the canopy area to the ground area and could be used to determine the openness and the length of the disease free period available for producers. Rapid light interception is essential to maintain yield, suppress late germinating weeds, and quickly reach critical photosynthetic rates (Hatfield and Carlson 1978, Sakamoto and Shaw 1967). Interactions between diseases or insects and herbicides can reduce yields more than either factor acting alone (Browde et al. 1994). Browde et al. (1994) reported that yield reductions were primarily attributed to a reduction in PAR interception by soybean at R4 to R5 (Browde et al. 1994). Postemergence applications of acifluorfen and bentazon reduced soybean LAI and yield in two of the three years evaluated (Browde et al. 1994). Similarly, postemergence tank mixture treatments of bentazon/acifluorfen + thifensulfuron + sethoxydim and lactofen + bentazon + clethodim applied to V5 soybean injured soybean 14 to 21%, and reduced vegetative development 7 DAT, reproductive development 20 and 80 DAT, height, and dry weight (Nelson and Renner 1998). The leaf area index was also reduced by these postemergence tank mixtures up to 52 DAT, and canopy development was delayed 70 and 80 DAT depending on the cultivar (Nelson and Renner 1998). Soybean yield with herbicides other than glyphosate was reduced 130 to 270 kg ha^{-1} compared to the untreated control (Nelson and Renner 1998). Soybean that are not injured have more rapid canopy closure which reduces light quality and quantity at the soil surface. This is important to reduce late germinating weeds and potential weed seed production, and to maximize soybean yield potential. However, changes in the time of maximum leaf area and canopy development may affect the

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Table 1. The effect of the environment and soybean growth characteristics on the infection and incidence of white mold in the field (F) and greenhouse or laboratory (G).

Characteristic and the effect on white mold infection or incidence[a]		Source
Apothecia		
(F)	Disease severity increased as the number of apothecia increased.	Boland and Hall 1988b
Fertility		
(G)	Increased fertility reduced lesion length.	Chun et al. 1987
Flowering		
(F)	Lesions resulted from tissue in direct contact with dead blossoms.	Boland and Hall 1982
(F)	White mold symptoms developed after flowering.	Boland and Hall 1988a
(F)	White flower cultivars had a greater incidence than purple flower cultivars.	Grau et al. 1982
(F)	Flower timing influenced DSI under irrigated culture.	Grau and Radkey 1984
Height		
(F)	Correlated disease incidence to increased height over 3 years.	Boland and Hall 1987
(F)	Correlated disease incidence to height in 1 environment with 18 similar genotypes.	Kim et al. 1999
Injury		
(G)	Mechanical injury caused longer lesions to develop than lesions originating from leaf axil.	Chun et al. 1987
Inoculum		
(G)	Dried sclerotia reduced apothecia production.	Abawi and Grogan 1975
(G)	Young ascospores (5 to 8 d old) were more infectious than 11 d old ascospores.	Chun et al. 1987
Irrigation		
(F)	Irrigated soybean had a greater DSI than non-irrigated.	Grau and Radkey 1984
(F)	All season irrigation had greater DSI than a pre/post flower irrigation timing.	Grau and Radkey 1984
Light		
(G)	No effect on lesion length.	Chun et al. 1987
(F)	After canopy closure disease increased.	Boland and Hall 1988a
(G)	As PAR increased from 475 $\mu mol \cdot m^{-2}s^{-1}$, soybean tolerance to white mold increased.	Pennypacker and Risius 1999

Table 1 (cont'd.

Lodging

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Table 1 (cont'd).

Lodging		
(F)	Correlated disease incidence to increased lodging for two years.	Boland and Hall 1987
(F)	Earlier maturing soybean had a lower disease incidence even under severe lodging.	Buzzell et al. 1993
(F)	Increased DSI was correlated with increased lodging.	Kim et al. 1999
Maturity group		
(F)	Correlated reduced incidence to shorter maturity.	Boland and Hall 1987
(F)	Earlier maturing soybean had less disease.	Buzzell et al. 1993
(F)	No correlation between white mold incidence and group I, II, and III soybean, but group 0 had the lowest incidence of disease.	Chun et al. 1987
(F)	Shorter maturity reduced susceptiblity.	Grau et al. 1982
(F)	Infrequent correlations between maturity group and DSI were reported.	Kim et al. 1999
(F)	Maturity group affected disease severity and had a linear relationship with the incidence of disease.	Yang et al. 1999
Plant age		
(G)	Younger plants (3 week old) had larger lesions than 7 week old soybean.	Chun et al. 1987
(G)	Five to six week old plants developed the largest lesions.	Chun et al. 1987
Rainfall		
(F)	Leaf wetness of 44 to 66 h was needed to insure infection.	Boland and Hall 1982
(F)	Leave wetness for 40 to 112 h was needed for infection.	Boland and Hall 1988a
(G)	A controlled environment at 20 C needed 70 to 120 h of plant wetness for infection.	Boland and Hall 1988a
(F)	Soil moisture greater than -5 bars was needed for good moist conditions for white mold.	Boland and Hall 1988a
Row spacing		
(F)	No effect of row spacing (23 to 69 cm) on disease incidence.	Buzzell et al. 1993
(F)	Greater DSI in 25 to 38 cm rows than 76 cm in 2 of 3 years.	Grau and Radkey 1984
(F)	Some cultivars had larger differences in incidence as row width increased.	Grau and Radkey 1984
(F)	Increased seeding rates had higher incidences of white mold.	Phipps 1983
(F)	Yield was higher in narrow- than wide-rows in the presence of white mold.	Phipps 1983

Table 1 (cont'd

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[a]Abbreviations
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Table 1 (cont'd).

Temperature		
(F)	As the corn heat unit rating increased from 2400 to 3300 for different cultivars, there was a higher incidence of disease.	Buzzell et al. 1993
(G)	Soybean incubated at 15 to 20 C had longer lesions than 25 to 30 C.	Chun et al. 1987
(F)	Lower air temperature was associated with increased disease incidence.	Grau and Radkey 1984
(G)	Shorter lesions were observed at 15 compared to 20 and 25 C.	Nelson et al. 1991a
(G)	10 to 22 C was considered an optimal temperature treatment for *S. sclerotiorum* growth.	Pennypacker and Risius 1999
Correlations between the Greenhouse and Field		
	A positive correlation between a lab assay for cultivar susceptibility and field susceptibility of some cultivars was reported.	Chun et al. 1987
	Excise stem technique had limited use.	Nelson et al. 1991b
	Soluble stem pigment and lesion length caused by oxalic acid were repeatable measurements in the laboratory.	Wegulo et al. 1998

[a]Abbreviations: DSI, disease severity index; F, field; G, greenhouse or laboratory; PAR, photosynthetically active radiation.

Table 2. Cl

Source
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Kim et al 19
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Cline and Jaco

Table 2. Class designations for white mold disease severity calculations.

Source	Class designation
Chun et al. 1987, Dann et al. 1998 and 1999, Grau et al. 1982, Grau and Radkey 1984, Kim et al. 1999	0. No symptoms 1. Lesions on the lateral branches only 2. Lesions on the main stem, wilting, some pods, no effect on pod fill 3. Lesions on the main stem, plant death, poor pod fill
Boland and Hall 1988a	0. No symptoms 1. < 5 cm lesions 2. Expanding lesions on the stem or branches 3. Up to ½ of the branches or stem are colonized 4. > ½ of the branches or stem are colonized and/or plant death
Boland and Hall 1986	0. No symptoms 1. Small flecks on the stem 2. Lesions < 2 mm in diameter 3. 1/3 of stem encircled by lesions 4. 2/3 of stem encircled by lesions 5. Complete encircling of the stem by lesions and/or plant death
Cline and Jacobsen 1983	0. No symptoms 1. Small lesions and water soaked flowers 2. Water soaked petioles 3. Lesions on the main stem and 25% mycelium growth. 4. Mycelium growth on 50% of the plant 5. Dead plants

Table 3. Phyt

Glycine spp
Glycine canes
Glycine clandi
Glycine falcata
Glycine gracili
Glycine latrobe
Glycine max
Glycine soya
Glycine tabacin
Glycine tomento

Table 3. Phytoalexins isolated from *Glycine* spp. (Ingham 1982).

Glycine spp.		Phytoalexin common name
Glycine canescens	Pterocarpans	(-)-canescacarpin
		(-)-glyceollin I
		(-)-glyceollin II
Glycine clandestina	Pterocarpans	(-)-clandestacarpin
Glycine falcata	Pterocarpans	(-)-glyceollin I
		(-)-glyceollin II
Glycine gracilis	Pterocarpans	(-)-glyceollin II
		(-)-glyceollin III
Glycine latrobeana	Ptercarpans	(-)-glyceollin I
		(-)-glyceollin II
		(-)-glyceollin III
Glycine max	Isoflavones	daidzein
		isoformononetin
	Pterocarpans	(-)-glyceocarpin
		(-)-glyceofuran
		(-)-glyceollin I
		(-)-glyceollin II
		(-)-glyceollin III
		(-)-glyceollin IV
		(-)-glycinol
		(-)-9-*O*-methylglyceofuran
	Coumestans	coumestrol
		sojagol
Glycine soya	Ptercarpans	(-)-glyceollin I
		(-)-glyceollin III
		(-)-glyceollin IV
Glycine tabacina	Pterocarpans	(-)-clandestacarpin
Glycine tomentella	Pterocarpans	(-)-clandestacarpin

Table 4. Effects of disease or stimulus on soybean phytoalexins and the associated cultivar.

Phytoalexin	Cultivar	Disease/Stimulus	Effect	Source
Glycoeocarpin	Harosoy	*Pseudomonas pisi* or aqueous sodium iodoacetate	Stimulated production in leaf extract	Ingham et al 1981

Table 4. Effects of disease or stimulus on soybean phytoalexins and the associated cultivar.[a]

Phytoalexin	Cultivar	Disease/Stimulus	Effect	Source
Glyceocarpin	Harosoy	*Pseudomonas pisi* or aqueous sodium iodoacetate	Stimulated production in leaf extract.	Ingham et al.1981
Glyceofuran		Acifluorfen	16 fold increase in glyceofuran 72 h after treatment.	Kömives and Cassida 1983
Glyceofuran and 9-*O*-methylglyceofuran	Harosoy	*Pseudomonas pisi* or aqueous sodium iodoacetate	Stimulated production in leaf extract.	Ingham et al.1981
Glyceollin	Harosoy	*Phytopthora megasperma* f. sp. *glycinea*	Low concentrations were reported, and 13 d old plants had 81 to 1044 μg g^{-1} fresh weight depending on the trifoliate.	Bhattacharyya and Ward 1986
Glyceollin	Harosoy 63	*Phytopthora megasperma* f. sp. *glycinea*	Resistant cultivar had a fast hypersensitive response. High concentrations were reported, and 13 d old plants had 400 to 4654 μg g^{-1} fresh weight.	Bhattacharyya and Ward 1986
Glyceollin	S 19-90	Lactofen at 0.11 kg ha^{-1}	Increased glyceollin 38.2 μg g^{-1} fresh weight compared to the untreated control 3 DAT, but had no effect 6 WAT.	Dann et al. 1999
Glyceollin	Williams 82	Lactofen at 0.11 kg ha^{-1}	Increased glyceollin 7.4 μg g^{-1} fresh weight compared to the untreated control 3 DAT, but had no effect 6 WAT.	Dann et al. 1999
Glyceollin	Soriano	1 M H_2O_2	Increased glyceollin in etiolated seedlings from 0 to 160 μg g^{-1} fresh weight after 50 h.	Degousée et al. 1994
Glyceollin	Soriano	3 mM IA	Increased glyceollin in etiolated seedlings from 0 to 40 μg g^{-1} fresh weight after 50 h.	Degousée et al. 1994

Table 4 (cont'd)

Glyceollin	*Soriano*	*8 mM PCMBS*	*Increased glyceollin in etiolated seedlings from 0 to 35 μg g⁻¹ fresh weight after 50 h*	Degousée et al. 1994

Table 4 (cont'd).

Glyceollin	Soriano	8 mM PCMBS	Increased glyceollin in etiolated seedlings from 0 to 35 $\mu g\ g^{-1}$ fresh weight after 50 h.	Degousée et al. 1994
Glyceollin	Soriano	10 mM tBuOOH	Increased glyceollin in etiolated seedlings from 0 to 125 $\mu g\ g^{-1}$ fresh weight after 50 h.	Degousée et al. 1994
Glyceollin	Harosoy	*Pseudomonas syrinae* pv. *glycinea*	Glyphosate at 10 $\mu g\ ml^{-1}$ partially inhibited glyceollin, and phenylalanine and tyrosine helped overcome the effects of glyphosate 72 h after inoculation.	Holliday and Keen 1982
Glyceollin	Harosoy 63	*Phytopthora megasperma* f. sp. *glycinea*	Low rates of glyphosate (10 $\mu g\ ml^{-1}$) reduced glyceollin production and had increased fungal growth. Glyphosate applied 6 h after inoculation or earlier was required to inhibit glyceollin production.	Keen et al. 1982
Glyceollin		Acifluorfen	79 fold increase in glyceollin 72 h after treatment.	Kömives and Cassida 1983
Glyceollin	Corsoy 79	*Heterodera glycines* Ichinohe[b]	COC[c], NIS[d], bentazon, and acifluorfen increased glyceollin levels 1.5 to 1.9 fold in the roots.	Levene et al. 1998
Glyceollin	Harosoy 63	*Phytopthora megasperma* f. sp. *glycinea*	Found glyceollin in the lesions 24 h after inoculation, and had 4668 and 4285 $\mu g\ g^{-1}$ fresh weight in the absence and presence of daidzin, respectively.	Morris et al. 1991
Glyceollin	A76-304005	*Phytopthora megasperma* f. sp. *glycinea*	Peak level of 344 $\mu g\ g^{-1}$ fresh weight 144 h after inoculation.	Olah et al. 1985
Glyceollin	Corsoy	*Phytopthora megasperma* f. sp. *glycinea*	Peak level of 154 $\mu g\ g^{-1}$ fresh weight 96 h after inoculation.	Olah et al. 1985
Glyceollin	Corsoy 79	*Phytopthora megasperma* f. sp. *glycinea*	Peak level of 158 $\mu g\ g^{-1}$ fresh weight 96 h after inoculation.	Olah et al. 1985

Table 4 (cont'd)

Glyceollin	*Harosoy 63*	*Phytophthora megasperma f sp. glycinea*	Peak level of 149 μg g^{-1} fresh weight 96 h after inoculation	Olah et al. 1985
Glyceollin	*L 60-347*	*Phytophthora megasperma f*	Peak level of 204 μg g^{-1} fresh weight 96 h after	Olah et al. 1985

Table 4 (cont'd).

Glyceollin	Harosoy 63	*Phytopthora megasperma* f. sp. *glycinea*	Peak level of 149 μg g^{-1} fresh weight 96 h after inoculation.	Olah et al. 1985
Glyceollin	L60-347	*Phytopthora megasperma* f. sp. *glycinea*	Peak level of 204 μg g^{-1} fresh weight 96 h after inoculation.	Olah et al. 1985
Glyceollin	Voris	*Phytopthora megasperma* f. sp. *glycinea*	Peak level of 132 μg g^{-1} fresh weight 96 h after inoculation.	Olah et al. 1985
Glyceollin	Lee	*Sclerotinia sclerotiorum*	Glyceollin in vitro prevented ascospore germination at 100 μg ml^{-1}, germ tube extension at 50 μg ml^{-1}, and mycelial growth at 200 μg ml^{-1}.	Sutton and Deverall 1984
Glyceollin	Harosoy, Harosoy 63	*Phytopthora megasperma* f. sp. *glycinea*	Localized detection around the infection site 8 h and accumulated 12 h after inoculation of resistant Harosoy 63. ED_{90} glyceollin levels acheived in 8 h after inoculation. Greater than 200 μg g^{-1} fresh weight was considered the ED_{90}.	Yoshikawa et al. 1978
Glyceollin	Harosoy 63	Blasticidin S	Inhibited glyceollin production.	Yoshikawa et al. 1978
Glyceollin	Harosoy 63	*Phytopthora megasperma* f. sp. *glycinea*	Exogenous β-1,3-endoglucanase from 0 to 500 μg ml^{-1} increased glyceollin 5 fold.	Yoshikawa et al. 1990
Glyceollin I	Harosoy	*Phytopthora megasperma* f. sp. *glycinea*	Accumulated up to 15 μg 10^{-1} lesions in 26 h.	Bhattacharyya and Ward 1987
Glyceollin I	Harosoy 63	*Phytopthora megasperma* f. sp. *glycinea*	Accumulated up to 150 μg 10^{-1} lesions in 26 h.	Bhattacharyya and Ward 1987
Glyceollin I	Sibley		80 μM was not as active or rapid as glyceollin III.	Giannini et al. 1991

Table 4 (cont'd)

Glycecolin I	*Canton*	*Sclerotinia sclerotiorum*	Etiolated soybean treated with oxalic acid caused an increase in glycecolin I from 22 to 100 μg g^{-1} fresh weight at the	Favaron et al 1988

Table 4 (cont'd).

Glyceollin I	Canton	*Sclerotinia sclerotiorum*	Etiolated soybean treated with oxalic acid caused an increase in glyceollin I from 22 to 100 $\mu g\ g^{-1}$ fresh weight as the oxalic acid rate increased from 0 to 5 mM, respectively.	Favaron et al. 1988
Glyceollin I, III	Centennial	*Heterodera glycines* Ichinohe[b]	Time course study indicated peak glyceollin I 4 to 6 d after inoculation (19 $\mu g\ g^{-1}$ root), and glyceollin III reached 8 $\mu g\ g^{-1}$ root 8 d after inoculation.	Huang and Barker 1991
Glyceollin I, III	Centennial	*Heterodera glycines* Ichinohe[b]	No effect on glyceollin III, but 8 $\mu g\ g^{-1}$ root of glyceollin I accumulated 4 d after inoculation.	Huang and Barker 1991
Glyceollin I, II, III	Harosoy	*Pseudomonas pisi* or aqueous sodium iodoacetat	Stimulated production in leaf extract.	Ingham et al.1981
Glyceollin I, II, III	Centennial	*Meloidogyne incognita*[b]	Glyceollins reduced nematode motility and O_2 uptake. Isomer III inhibited motility greater than I or II. ED_{50} value of 12 to 20 $\mu g\ ml^{-1}$.	Kaplan et al. 1980
Glyceollin I, II, III	Centennial	*Meloidogyne javanica*[b]	No effect on nematode motility at up to 40 $\mu g\ ml^{-1}$.	Kaplan et al. 1980
Glyceollin III	Sibley		Caused rapid leakage of protons from red beet tonoplasts within 2 minutes after added at 60 μM.	Giannini et al. 1991
Isoformononetin	Harosoy	*Pseudomonas pisi* or aqueous sodium iodoacetate	Stimulated production in leaf extract.	Ingham et al.1981

[a]Abbreviations: DAT, d after treatment; WAT, weeks after treatment.
[b]Nematode.
[c]COC, Crop oil concentrate, Prime Oil, Riverside/Terra Corp., Sioux City, IA 51101.
[d]NIS, X-77, Valent U.S.A. Corp., 1333 North California Boulevard, Walnut Creek, CA 94596.

Table 5. Soy
greenhouse

Cultivar
Ace
Asgrow A25
Clay
Colfax
Corsoy
Corsoy 79
Dassel
Evans
Hardin
Hodgson
Hodgson 78
Hawk
Maple Arrow
Maple Presto
McCall
Parker
Pioneer 16?7
Portage
S?9-96
Ubon

Table 5. Soybean cultivars with reported tolerance to white mold in the field or greenhouse.

Cultivar	Source
Ace	Boland and Hall 1987
Asgrow A2506	Kim et al. 1999
Clay	Nelson et al. 1991b
Colfax	Kim et al. 1999
Corsoy	Chun et al. 1987, Cline and Jacobsen 1983, Grau et al. 1982, Grau and Radkey 1984
Corsoy 79	Boland and Hall 1986, Dann et al. 1998, Kim et al. 1999, Yang et al. 1999
Dassel	Wegulo et al. 1998, Yang et al. 1999
Evans	Chun et al. 1987
Hardin	Chun et al. 1987
Hodgson	Boland and Hall 1986, Grau et al. 1982, Grau and Radkey 1984
Hodgson 78	Chun et al. 1987, Grau et al. 1982
Hawk	Boland and Hall 1986
Maple Arrow	Boland and Hall 1987
Maple Presto	Boland and Hall 1987, Nelson et al. 1991b
McCall	Boland and Hall 1987, Nelson et al. 1991b
Parker	Wegulo et al. 1998
Pioneer 1677	Boland and Hall 1986
Portage	Nelson et al. 1991b
S19-90	Dann et al. 1998 and 1999, Kim et al. 1999, Wegulo et al. 1998, Yang et al. 1999
Union	Cline and Jacobsen 1983

Table 6. Fungi
(1998)

Fungicide
Beniate (Du Pe
Betran (Gowar
Bravo (ISK Bio
Captec (Micro F
Folicure (Bayer)
Mecap (Rhône-P
Rovral (Rhône-P
Topsin (Elf Atoc

Table 6. Fungicides for *Sclerotinia* spp. control (Crop Protection Chemicals Reference 1998).

Fungicide	Crop	Application rate
Benlate (Du Pont)		
	Bean	24 to 64 oz A^{-1}
	Brassica	32 to 96 oz A^{-1}
	Carrot	4 to 48 oz A^{-1}
	Dandelion	8 to 32 oz A^{-1}
	Tomato	8 to 80 oz A^{-1}
Botran (Gowan)		
	Cucumber	1.3 lbs/A
	Celery	2 to 5.3 lbs/A
	Florence fennel	2 to 5.3 lbs/A
	Lettuce	2 to 3.3 lbs/A
	Potato	2 lbs/A
	Snap bean	2 to 4 lbs/A
	Sweet potato	3 to 3.75 lbs/A
Bravo (ISK Biosciences)		
	Peanut	2.5 pt A^{-1}
Captec (Micro Flow)		
	Dichondra	1 qt/100 gal at 1 gal 100 ft^{-2}
Folicure (Bayer)		
	Peanut	7.2 oz A^{-1}
Mocap (Rhône-Poulenc)		
	Peanut	2.1 lbs 1000 ft^{-2} of row
	Lettuce	1.5 to 2 pt/A
	Peanut	2 pt A^{-1}
	Potato	2 pt A^{-1}
Rovral (Rhône-Poulenc)		
	Bean	1.5 to 2 pt A^{-1}
Topsin (Elf Altochem)		
	Bean	1 to 1.5 lb A^{-1}
	Soybean	0.75 to 1 lb A^{-1}

Table 1. Effect of herbicides and adjuvants on *Sclerotinia* spp.

Herbicide	Disease	Effect	Source
Acifluorfen	*S. sclerotiorum*	Reduced DSI at one location	Dann et al. 1999

Table 7. Effect of herbicides and adjuvants on *Sclerotinia* spp.

Herbicide	Disease	Effect	Source
Acifluorfen	*S. sclerotiorum*	Reduced DSI at one location.	Dann et al. 1999
Alachlor	*S. sclerotiorum*	Variable effects on sclerotia germination.	Radkey and Grau 1986
Atrazine	*S. sclerotiorum*	No effect on stipes or sclerotia. Abnormal apothecia were caused by the active ingredient. No ascospores were produced.	Casale and Hart 1986
	S. sclerotiorum	Stimulated sclerotia germination and increased the number of apothecia that germinated and stipes.	Radkey and Grau 1986
Bentazon	*S. sclerotiorum*	No effect on DSI.	Dann et al. 1999
Bromoxynil	*S. trifoliorum*	Increased DSI in controlled environment, but had no effect in the field.	Reichard et al. 1997
Chlorimuron	*S. sclerotiorum*	No effect on DSI.	Dann et al. 1999
Cloramben	*S. sclerotiorum*	Variable effects on sclerotia germination.	Radkey and Grau 1986
Crop oil adjuvant	*S. sclerotiorum*	No effect on DSI	Dann et al. 1999
2,4-DB	*S. trifoliorum*	Reduced DSI in controlled environment, but had no effect in the field.	Reichard et al. 1997
DASH HC[b]	*S. trifoliorum*	Increased DSI in controlled environment, but had no effect in the field.	Reichard et al. 1997
DNBP	*S. sclerotiorum*	Inhibited sclerotia germination and prevented growth at high rates.	Radkey and Grau 1986
Diuron	*S. sclerotiorum*	Inhibited mycelial growth. Normal apothecia were produced. Reduced growth more than the triazine herbicides.	Casale and Hart 1986
Fomesafen	*S. sclerotiorum*	No effect on DSI.	Dann et al. 1999
Glyphosate	*S. sclerotiorum*	No effect on DSI.	Lee and Penner 1999, Penner et al. 1997
	S. sclerotiorum	No effect on white mold incidence when applied to a glyphosate-resistant soybean cultivar.	Penner et al. 1997
Imazethapyr	*S. trifoliorum*	No effect on DSI in controlled environment or in the field.	Reichard et al. 1997
	S. sclerotiorum	No effect on DSI.	Dann et al. 1999

Table 7 (cont'd)

Lactofen	*Sclerotinia*	Reduced DSI under high disease pressure, but had no effect at low disease pressures. DSI was related to the herbicide rate	Dann et al. 1999

Table 7 (cont'd).

Lactofen	*S. sclerotiorum*	Reduced DSI under high disease pressure, but had no effect at low disease pressures. DSI was related to the herbicide rate.	Dann et al. 1999
Linuron	*S. sclerotiorum*	Inhibited sclerotia germination.	Radkey and Grau 1986
Metribuzin	*S. sclerotiorum*	Inhibited mycelial growth. Normal apothecia were produced. Prevented carpogenic germination.	Casale and Hart 1986
	S. sclerotiorum	Stimulated sclerotia and increased the number of apothecia that germinated and stipes.	Radkey and Grau 1986
Pentachlorophenol	*S. laxa*	Reduced the incidence of disease on almond.	Katan and Eshel 1973
Pendimethalin	*S. sclerotiorum*	Stimulated sclerotia and increased the number of apothecia that germinated.	Radkey and Grau 1986
Pronamide	*S. trifoliorum*	Reduced DSI in controlled environment, but had no effect in the field.	Reichard et al. 1997
Sethoxydim	*S. trifoliorum*	Increased DSI in controlled environment, but had no effect in the field.	Reichard et al. 1997
Simazine	*S. sclerotiorum*	No effect on stipes or sclerotia. Apothecia were abnormal. No ascospores were produced.	Casale and Hart 1986
	S. sclerotiorum	Stimulated sclerotia and increased the number of apothecia that germinated and stipes.	Radkey and Grau 1986
Thifensulfuron	*S. sclerotiorum*	No effect on DSI.	Dann et al. 1999
Trifluralin	*S. sclerotiorum*	Stimulated sclerotia and increased the number of apothecia that germinated.	Radkey and Grau 1986

[a]Petroleum based adjuvant.
[b]Abbreviations: DSI, disease severity index.

Figure 1. Sclerotinia sclerotiorum (Lib.) de Bary disease cycle in soybean in the field. White mold, caused by the fungus *Sclerotinia sclerotiorum* (Lib.) de Bary, is prevalent in soybean cultures conducive to high yields (1). Favorable environmental conditions (usually cool, moist soil and canopy wetness) stimulate the carpogenic germination of soil-borne sclerotia to produce tan to brown apothecia which subsequently produce ascospores (2). Ascospores are wind dispersed. Ascospores germinate and produce mycelium which invades senescent plant parts like flowers. Mycelium produces oxalic acid which kills soybean cells and allows the infection of soybean tissue (3). White cottony appearing mycelium grows as an early sign of white mold in and on soybean petioles, stems, and branches (4). Mycelium growth on the stem girdles the stem, kills the plant, causes soybean lodging, and produces sclerotia (5). Plants wilt and turn grey-green followed by partial or whole plant necrosis which results in premature pod abortion (6). Sclerotia are black "mouse dropping" appearing structures found on and in the stem (7). Sclerotia are dispersed by harvest equipment, manure, tillage equipment, and seed contamination (8). Seed and sclerotia separation may be difficult due to similar size and infection of seed (usually shriveled) with mycelium (9). Sclerotia serve as the overwintering structures and may survive in the soil for several years (10). Crop rotation, tillage, row spacing, population, and clean seed may help reduce white mold in the field (11).

a R

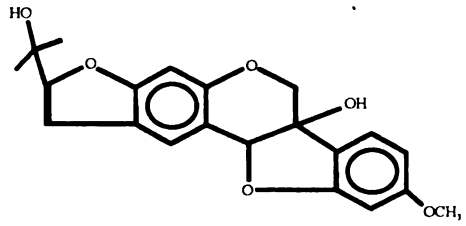
b R

c R

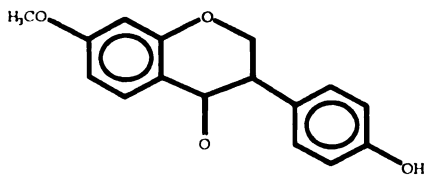
d R

Figure 2 R
absorbance ac

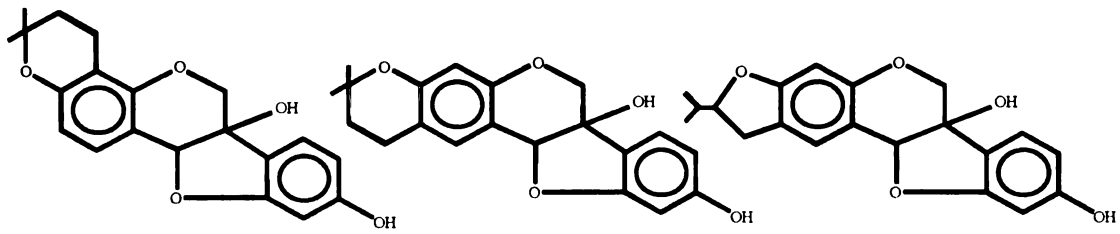
a. Rf 0.71 Methyl ester of glyceofuran.



b. Rf 0.56 Isoformonoetin.



c. Rf 0.5 is a mixture of glyceollin I, II, and III.



d. Rf 0.25 Glyceofuran and the precursor of glyceollin II and III (Keen 1982).

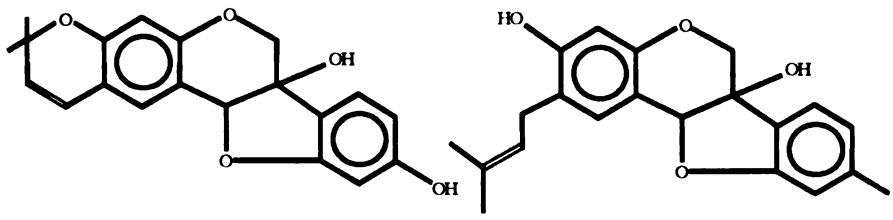


Figure 2. Rf-values of phytoalexins separated with thin-layer chromatography using chloroform:acetone:NH_4OH (50:50:1) (Ingham et al. 1981).

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CHAPTER 3

SOYBEAN (*Glycine max* L.) CULTIVAR AND HERBICIDE SELECTION AFFECTS SOYBEAN DEVELOPMENT AND THE INCIDENCE OF WHITE MOLD [*Sclerotinia sclerotiorum* (Lib.) de Bary]

Abstract: Postemergence herbicides can affect soybean [*Glycine max* (L.) Merr.] development and the incidence of white mold [*Sclerotinia sclerotiorum* (Lib.) de Bary] in glyphosate-resistant and non-resistant soybean. The effects of glyphosate, lactofen, and thifensulfuron on soybean canopy development, flower number plant^{-1}, *S. sclerotiorum* lesion diameter, phytoalexin production, white mold incidence, and yield of glyphosate-resistant and near isogenic non-resistant soybean cultivars were evaluated in field experiments in 1998 and 1999 at East Lansing, MI. Near isogenic glyphosate-resistant and non-resistant cultivars had similar canopy development, flower number plant^{-1}, *S. sclerotiorum* lesion diameter, phytoalexin production, and white mold disease severity. Yield of glyphosate-resistant and non-resistant soybean near isolines was similar except GL2415 yielded 390 kg ha^{-1} greater than GL2600 Roundup Ready® (RR). Soybean tolerance to *S. sclerotiorum* was not related to glyphosate-resistance. S 12-49, S14-M7 (RR), S 19-90, and S20-B9 (RR) had a lower incidence of white mold than GL2415, GL2600 (RR), P9281, and P93B01 (RR). S 12-49, S14-M7 (RR), S 19-90, and S20-B9 (RR) had peak flowering one week before apothecia were observed while GL2415, GL2600 (RR), P9281, and P93B01 (RR) had peak flowering one week after apothecia were observed in the field. Glyphosate at 840 g ae ha^{-1} plus 28% urea ammonium nitrate (UAN) at 2.3 L ha^{-1} did not injure soybean, and relative humidity or temperature at the soil surface 19 d after treatment (DAT), reproductive development, canopy development,

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Pioneer 93

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flower number plant^{-1}, *S. sclerotiorum* lesion size, phytoalexin production, disease severity, or yield did not differ from untreated soybean. Lactofen at 70 g ai ha^{-1} plus 0.25% v/v nonionic surfactant (NIS) plus UAN at 2.3 L ha^{-1} reduced *S. sclerotiorum* lesion diameter 2 to 26 DAT, increased phytoalexin production 2 to 26 DAT, and reduced white mold disease severity by 50% compared to thifensulfuron, glyphosate, or untreated soybean. Lactofen and thifensulfuron at 4.5 g ai ha^{-1} plus 0.25% v/v NIS plus UAN at 2.3 L ha^{-1} injured soybean 3 to 17% 7 DAT, reduced canopy development 3 to 28 DAT, and delayed reproductive development, but neither herbicide affected soybean yield compared to untreated soybean. The reduction in white mold incidence following an application of lactofen may be attributed to increased phytoalexin production, reduced canopy development, and delayed reproductive development.

Nomenclature: glyphosate, *N*-(phosphonomethyl)glycine; lactofen, (±)-2-ethoxy-1-methyl-2-oxoethyl 5-[2-chloro-4-(trifluoromethyl)phenoxy]-2-nitorbenzoate; thifensulfuron, 3-[[[[(4-methoxy-6-methyl-1,3,5-triazin-2-yl)amino]carbonyl]amino]sulfonyl]-2-thiophenecarboxylic acid, soybean, *Glycine max* (L.) Merr.; white mold, *Sclerotinia sclerotiorum* (Lib.) de Bary.

Additional index words: acetolactate synthase-inhibitor, canopy development, cultivar, diphenyl ether, disease severity, herbicide resistant, isolines, leaf area index, phytotoxicity, postemergence, protoporphyrinogen inhibitor, Roundup Ready®, white mold.

Abbreviations: AMS, ammonium sulfate; 'Novartis S 19-90', 'S 19-90'; 'Novartis S20-B9', 'S20-B9'; 'Novartis S14-M7', 'S14-M7'; 'Novartis S 12-49', 'S 12-49', 'Great Lakes 2415', 'GL2415'; 'Great Lakes 2600', 'GL2600'; 'Pioneer 9281', 'P9281'; 'Pioneer 93B01','P93B01'; DAT, d after treatment; DSI, disease severity index; LAI, leaf area index; NIS, nonionic surfactant; PDA, potato dextrose agar; UAN, 28% urea ammonium nitrate.

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INTRODUCTION

White mold or Sclerotinia stem rot [*Sclerotinia sclerotiorum* (Lib.) de Bary], a common disease in the north central region of the United States as well as other locations in the world, is caused by a soil-borne ascomycete fungus, *S. sclerotiorum* (Marinelli et al. 1998, Thompson and Van der Westhuizen 1979). A recent Illinois survey reported *S. sclerotiorum* in 45% of the sampled soybean fields (Hartman et al. 1998). Symptoms of white mold include wilting and premature death of Group 0 to Group III soybean. *S. sclerotiorum* has a broad crop host range (Abawi and Grogan 1975, Köhler and Friedt 1999, Marciano et al.1983, Marinelli et al. 1998, Maxwell and Lumsden 1970) and is more prevalent in soybean when other host species are grown as a rotational crop. Several soybean growth characteristics, environmental conditions, and cultural practices that influence the infection and incidence of *S. sclerotiorum* have been evaluated in field and controlled environments (Table 1). These studies have shown that the management of this disease is complicated and depends heavily on the environmental conditions of a given year, adopted cultural methods, and cultivar selection.

S. sclerotiorum causes premature soybean death which results in the production of shriveled soybean pods with little or no seed. Soybean cultivars differ in white mold tolerance, but yield losses may not always occur under low levels of disease due to yield compensation of nearby soybean plants (Hart 1998). A negative correlation between white mold disease severity or incidence and soybean yield in Illinois (Hoffman et al. 1998), Iowa (Yang et al. 1999), Michigan (Chun et al. 1987, Kim et al. 1999), and Wisconsin (Grau and Radkey 1984) has been reported. Yield reductions caused by *S.*

sclerotiorum may range from 147 to 370 kg ha^{-1} (2 to 5.5 bu A^{-1}) for every 10% increase in disease severity depending on the environment and cultivar. Planting soybean cultivars that are tolerant to white mold is strongly recommended to reduce yield loss (Boland and Hall 1986, Boland and Hall 1987, Chun et al. 1987, Cline and Jacobsen 1983, Dann et al. 1998 and 1999, Grau et al. 1982, Grau and Radkey 1984, Kim et al. 1999, Nelson et al. 1991b, Wegulo et al. 1998, Yang et al. 1999). Avoiding cultivars with parentage from white mold sensitive 'Williams' or 'Asgrow A3127' cultivars may reduce the incidence of this disease (Kim et al. 1999). Limited research has evaluated *S. sclerotiorum* tolerance in glyphosate-resistant soybean, and no published research has evaluated the effect of glyphosate and other postemergence herbicides on the incidence of white mold in glyphosate-resistant soybean.

A herbicide treatment may prevent disease infection (Uchimiya et al. 1993) or stimulate phytoalexin production (Dann et al. 1999, Levene et al. 1998) which may help reduce the incidence of disease. Phytoalexins are antimicrobial compounds produced by plants after disease infection or treatment with biotic or abiotic elicitors (Hammerschmidt 1999). *Glycine* spp. produce twelve known phytoalexins (Ingham 1982). The amount and type of these compounds produced may vary depending on the cultivar, disease, or stimulus evaluated. The production of soybean phytoalexins in response to herbicides (Dann et al. 1999, Holliday and Keen 1982, Keen et al. 1982, Kömives and Cassida 1983, Levene et al. 1998), other chemicals (Degousée et al. 1994, Favaron et al. 1988, Ingham et al. 1981, Yoshikawa et al. 1978), or diseases (Huang and Barker 1991, Ingham et al. 1981, Morris et al. 1991, Olah et al. 1985, Yoshikawa et al. 1978), and the effect of

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soybean phytoalexins on *S. sclerotiorum* (Sutton and Deverall 1984), *Phytopthora megasperma* f. sp. *glycinea* (Bhattacharyya and Ward 1986, Yoshikawa et al. 1978), nematodes (*Heterodera glycines* Ichonohe) (Kaplan et al. 1980, Levene et al. 1998) have been reported. These studies suggest that phytoalexins are an important component of soybean disease tolerance.

The recent introduction of herbicide-resistant crops including glyphosate-resistant soybean, has raised concerns regarding the susceptibility of glyphosate-resistant soybean to disease when glyphosate is applied for weed control (Lee and Penner 1999, Sanogo et al. 2000). Lévesque and Rahe (1992) reviewed the interaction between glyphosate and plant diseases. Glyphosate reduced or prevented phytoalexin production in bean (*Phaseolus vulgaris* L.) (Johal and Rahe 1990), lucerne (*Medicago sativa* L.) (Latunde-Dada and Lucas 1985), soybean (Holliday and Keen 1982), and enhanced mycelia growth of *Alternaria cassiae*, a biocontrol agent of sicklepod (*Cassia obtusifolia* L.) (Sharon et al. 1993). In addition, glyphosate increased the severity and frequency of *Fusarium solani* f. sp. *glycines* in the roots of glyphosate-resistant and non-resistant soybean cultivars (Sanogo et al. 2000). The research to date with glyphosate and the effects on phytoalexins has involved non-glyphosate resistant soybean cultivars; however, no research has reported the effects of glyphosate on phytoalexin production with glyphosate-resistant cultivars. A linkage between herbicide resistance and increased disease susceptibility in commercial crop cultivars could have detrimental effects on crop yield and the acceptance of a cultivar in the market. The interaction between herbicide treatments

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and the incidence of white mold could significantly affect soybean production practices and weed control recommendations.

Postemergence herbicides, such as thifensulfuron, lactofen, and acifluorfen, cause chlorosis, necrosis, and/or stunting of soybean (Hart and Roskamp 1998, Kapusta et al. 1986, Wichert and Talbert 1993). Postemergence herbicides may alter the canopy and delay canopy closure such that late germinating weeds or difficult to control weeds may not be controlled (Mickelson and Renner 1997, Nelson and Renner 1998). Postemergence tank mixture treatments of bentazon/acifluorfen plus thifensulfuron plus sethoxydim and lactofen plus bentazon plus clethodim applied to V5 soybean reduced reproductive development 20 and 80 DAT and canopy development up to 52 DAT (Nelson and Renner 1998). Changes in soybean morphology, the time of maximum leaf area, and canopy development may affect the microclimate in the soybean canopy and the potential for *S. sclerotiorum* infection and white mold development.

The objectives of this research were to determine if: 1) glyphosate-resistant cultivars differed in reproductive development, canopy development, flowering characteristics, phytoalexin production, white mold disease severity, and soybean yield compared to non-resistant near isolines and 2) if postemergence herbicides including glyphosate, lactofen, and thifensulfuron influenced soybean response, reproductive development, canopy development, flowering characteristics, phytoalexin production, white mold disease severity, and soybean yield compared to untreated soybean.

MATERIALS AND METHODS

Field research was conducted in 1998 and 1999 at the Michigan State University Research Farm *S. sclerotiorum* nursery at East Lansing (42° 43' N, 84° 33' W). The soil was a Capac sandy loam (fine-loamy, mixed mesic Aeric Ochraqualf) with 1.5% organic matter and pH 6.5 in 1998. The 1998 site was fall chisel plowed, soil finished three times in the spring, and fertilized with 170 kg ha^{-1} of 0-0-60 prior to planting. In 1999, the soil was a Capac loam with pH 6.8 and 1.7% organic matter. The field was soil finished twice in the spring and fertilized with 170 kg ha^{-1} of 6-24-24 prior to planting. Plots were maintained weed-free throughout the season.

Research was arranged in a split-plot design with three replications. Cultivar was the main plot and herbicide treatment was the sub-plot. Plots were 1.5 by 6.1 m and trimmed back to 1.5 by 4.3 m prior to harvest. Near isolines of glyphosate-resistant and non-resistant soybean, 'S 12-49', 'S14-M7' Roundup Ready® (RR), 'S 19-90', 'S20-B9' (RR), 'GL2415', 'GL2600' (RR), 'P9281', and 'P93B01' (RR), were planted May 13, 1998 and May 11, 1999 in 38 cm rows at 543,000 seeds ha^{-1} with a final stand of 474,000 plants ha^{-1}. Herbicide treatments included an untreated control, thifensulfuron at 4.5 g ai ha^{-1} plus 0.25% v/v nonionic surfactant[3] (NIS) plus 28% urea ammonium nitrate (UAN) at 2.3 L ha^{-1}, lactofen at 70 g ai ha^{-1} plus 0.25% v/v nonionic surfactant plus UAN at 2.3 L

[3]Nonionic surfactant was Activator-90, a mixture of alkyl polyoxyethylene ether and free fatty acids, Loveland Industries Inc., P.O. Box 1289, Greeley, CO 80632.

ha^{-1}, and glyphosate (formulated as Roundup Ultra®)[4] at 840 g ae ha^{-1} plus UAN at 2.3 L ha^{-1}. Glyphosate was only applied to glyphosate-resistant soybean.

Herbicide treatments were applied on June 25, 1998 and June 20, 1999 to soybean 20 to 25 cm tall, in the V5 to V6 stage of development, and prior to R1 for all cultivars (Fehr and Caviness 1977). Treatments were applied with a CO_2 propelled hand-boom calibrated to deliver 178 L ha^{-1} at 207 kPa, traveling 6.3 km h^{-1}, and equipped with 8003[5] flat-fan nozzles spaced 51 cm apart and 48 cm above the soybean canopy. The air temperature was 32 C with 52% relative humidity in 1998, and 28 C with 40% relative humidity in 1999. Supplemental irrigation was provided in the evenings beginning July 5, 1998 and July 3, 1999 10 and 13 d after herbicides were applied, respectively. Approximately 3 mm of water was provided daily during soybean flowering to encourage white mold development.

Soybean injury from 0 to 100% (0 = no visual crop injury and 100 = complete crop death) was evaluated 3, 7, 14, and 28 DAT based on the combined visual effects of the herbicides on necrosis, chlorosis, and stunting. Relative humidity and temperature at the soil surface were measured 19 DAT in all plots at solar noon since the greatest differences in microclimate conditions were reported at this time of the day in other research (Blad et al. 1978). Flower number of three randomly selected plants in each plot was recorded and averaged.

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[4]Roundup Ultra, Monsanto Co., 800 N. Lindbergh Blvd., St. Louis, MO 63167.

[5]Teejet flat-fan tips. Spraying Systems Co., North Ave. and Schmale Road, Wheaton, IL 60188.

Light measurements were recorded at 7 to 14 d intervals from the time of herbicide application until 85 DAT with a SunScan Canopy Analysis System[6]. Five measurements were recorded with a one-m light measurement device diagonal to the soybean row. Incident and diffused light measurements have been utilized as an effective non-destructive method to measure soybean LAI (Walker et al. 1988).

Trifoliolate leaves located at the fifth node (fourth trifoliolate) were harvested from three separate herbicide treated plants in each plot 0, 2, 4, 7, 12, and 26 DAT except thifensulfuron applied to S 12-49, S14-M7, GL2415, GL2600, P9281, and P93B01 cutivars. In addition, untreated trifoliolates located at the ninth node (eighth trifoliolate) were harvested 26 DAT from three separate plants in each plot except thifensulfuron applied to S 12-49, S1 4-M7, GL2415, GL2600, P9281, and P93B01 cutivars. One leaf from each trifoliolate was evaluated in a detached leaf bioassay, and the other leaves were stored at -20 C and evaluated for antifungal compounds using a TLC phytoalexin bioassay. Excised leaves used in the detached leaf bioassay were placed in a 150 by 15 mm petri dish[7] with moistened filter paper to ensure a humid environment for *S. sclerotiorum* growth. A potato dextrose agar (PDA) plug, 2.4 mm in diameter, from the margin of *S. sclerotiorum* was placed on the leaves and the diameter of growth was measured with an electronic digital caliper after incubating 48 h. The TLC phytoalexin bioassay utilized *Cladosporium cucumerinum* to determine and quantify antifungal compounds produced by soybean (Keen et al. 1971). This bioassay was used to evaluate

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[6]Dynamax Inc., 10808 Fallstone #350, Houston TX 77099.

[7]VWR Scientific Products, 800 E. Fabyan Pkwy., Batavia, IL 60510.

the relative total antifungal activity (phytoalexin production) between treatments. Soybean leaves were weighed, cut into 0.5 to 1.0 cm pieces, and extracted with 40:60 ethanol:water v/v for 4 h. The extract was evaporated[8] at 40 C under reduced pressure to approximately 1 ml and transferred to a 10 ml test tube. The flask was then rinsed with 1 ml water. The extract and water rinsate was combined and extracted three times with 1 ml ethyl acetate. The ethyl acetate extract was dried with anhydrous magnesium sulfate and then evaporated under a flow of N_2. The residue was resuspended in ethyl acetate at 50 µl g^{-1} leaf fresh weight and applied to a silica-gel (250 µm) thin-layer chromatography plate[9] at 50 µl in 1998 and 30 µl in 1999. The plate was developed in an acetone:chloroform:ammonia hydroxide solution at 50:50:1 v/v. *C. cucumerinum* spores were suspended in a 100 ml solution of 0.7 g KH_2PO_4, 0.4 g KNO_3, 0.3 g Na_2HPO_4, 0.1 g $MgSO_4$, 0.1 g NaCl, and 5.0 g D-glucose that was adjusted to pH 6.5. A fine spray of suspended spores was applied to the TLC plate and the plate was placed in a sealed chamber with 100% humidity. Plates were incubated for 2 to 3 d and stored at -20 C. Photo copies[10] of the plates were made and clear areas with no *C. cucumerinum* growth were quantified with a leaf area meter[11]. Total areas were calculated that corresponded to Rf values for the methyl ester of glyceofuran (Rf 0.71); isoformonoetin and a mixture of glyceollin I, II, and III (Rf 0.5 to 0.56); and glyceofuran and the precursor of glyceollin II

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[8]Rotavapor, Büchi, Labortechnik AG, Postfach, CH-9230, Flawil.

[9]Analtech, Inc., P. O. Box 7558, 75 Blue Hen Dr., Newark, DE 19713.

[10]Cannon, imageRUNNER 330S, One Cannon Plaza, Lake Success, NY 11042.

[11]LI-3000, LI-COR, 4421 Superior Street, Lincoln, NE 68504.

and III (Rf 0.25) previously reported using chloroform:acetone:NH_4OH (50:50:1 v/v) as the mobile phase and a silica-gel TLC plate as the stationary phase (Ingham et al. 1981, Keen 1982).

Near physiological maturity, thirty plants per plot were evaluated for the incidence of white mold. The disease severity index was calculated according to the scale (0 = no symptoms, 1 = lesions on the lateral branches only, 2 = lesions on the main stem but no effect on pod fill, and 3 = lesions on the main stem and pod fill was reduced) described by Grau and Radkey (1984). Soybean were harvested with a Massey 10[12] small plot harvester and moisture adjusted to 13%. An analysis of variance was conducted and percent data for visual injury were transformed to the arcsine prior to the analysis. The transformation did not affect the conclusions so original data is presented. Data were combined over years and main effects presented where interactions were not observed. TLC phytoalexin bioassay data were subjected to an *F Max* test for homogeneity (Kuehl 1994) and data were combined over location since variances for both locations were homogenous. Means were separated using Fisher's Protected LSD at $p \leq 0.05$.

RESULTS AND DISCUSSION

Soybean Response. Soybean response to herbicide treatments differed by cultivar (Table 2), and was characteristic of the herbicide family (Gunsolus and Curran 1992). Glyphosate did not injure glyphosate-resistant soybean while thifensulfuron injury to soybean was minimal (Table 2). Lactofen caused 8 to 12% injury 3 DAT and injury

[12]Kincaid Equipment Manufacturing, P.O. Box 400, Haven, KS 47543.

increased to 13 to 17% 7 DAT. Injury was no longer visually evident 35 DAT from any herbicide treatment (data not presented).

Reproductive and Canopy Development. Reproductive development of soybean is classified according to flowering, pod development, seed development, and maturity (Fehr and Caviness 1977). Soybean in R1 are in beginning flower, R2 are in full flower, and R3 are in beginning pod. Flowering has almost completely ceased by R4 (full pod), and most of the flowers at this stage are concentrated near the top of the plant. The reproductive stage of soybean 14 and 21 DAT (Table 3) as well as canopy development (Figure 1) varied by cultivar and year. In 1998, all cultivars were at the same reproductive stage except P93B01 14 DAT, and all cultivars were at full bloom with some of the late Group I or early Group II soybean producing pods by 21 DAT (Table 3). In 1999, the late Group I and early Group II soybean were at full flower and P93B01 had barely begun flowering 14 DAT. One week later, S 12-49 started producing pods while the other cultivars were primarily in full bloom except P93B01. Drier growing conditions two weeks prior and one week following herbicide application (5.4 cm in 1998 and 2.3 cm in 1999) coupled with cooler (6 to 7 C) minimum and maximum average temperatures in the week prior to herbicide application probably delayed reproductive development in 1999 compared to 1998. Soybean cultivar LAI was similar between glyphosate-resistant and non-resistant near isolines for all measurement dates except S 12-49 and S14-M7, and GL2415 and GL2600 21 DAT in 1998 (Figure 1). In 1999, LAI was only different between P9281 and P93B01 isolines 7 DAT. S 12-49 had one of the lowest LAI throughout the season in

1998. Peak canopy development for all of the cultivars was 35 DAT in 1998 and 35 to 48 DAT in 1999.

Glyphosate did not affect reproductive development when compared to the untreated control (data not presented). However, thifensulfuron and lactofen delayed soybean reproductive stage, flowering, pod development 3 to 35 DAT (Figure 2), and reduced canopy development 3 to 28 DAT (Figure 3) in 1998 and 1999. These results are similar to research conducted with postemergence tank mixtures applied to V5 soybean (Nelson and Renner 1998). Canopy development was reduced by thifensulfuron and lactofen more in 1999 than 1998 compared to the untreated control. These differences were probably due to rainfall prior to and following herbicide application. Yearly differences in herbicide effects on canopy ground cover ratings have been attributed to rainfall differences following herbicide application (Donald 1998). In 1999, only 1.8 cm of rain fell from 0 to 7 DAT which decreased LAI of treated soybean compared to the untreated control. Irrigation was not initiated until 10 and 13 d after herbicides were applied in 1998 and 1999, respectively (Figure 3).

Soybean Flowering. Postemergence herbicide treatments influenced flower number plant^{-1} and peak flowering for all cultivars (Figure 4). Interactions were expected and observed due to the maturity differences of the cultivars evaluated; therefore, data were analyzed for the paired isolines separately. Flower number plant^{-1} in glyphosate-resistant cultivars was no different than non-resistant soybean (data not presented). Peak flowering was 14 DAT for S 12-49, S14-M7, S 19-90, and S20-B9 and 28 DAT for GL2415, GL2600, P9281, and P93B01. Thifensulfuron and lactofen reduced S 12-49, S14-M7, S

19-90, and S20-B9 flower number plant^{-1} 7 and 14 DAT, and reduced GL2415 and GL2600 flower number plant^{-1} 14 DAT. Conversely, P9281 and P93B01 treated with lactofen had more flowers plant^{-1} than the untreated control 28 DAT.

Soybean flowering is primarily controlled by photoperiod (Jeffers 1987). Lactofen stressed soybean such that reproductive development was delayed, supporting previous research (Nelson and Renner 1998). Lactofen reduced the flower number of Group I soybeans (S 12-49, S14-M7, S 19-90, and S20-B9); had no effect on mid-Group II soybean flower number (GL2415 and GL2600); and increased late Group II flower number (P9281 and P93B01) compared to untreated soybean. Thus, lactofen's affect on soybean flowering may increase when applied at or near the time of flowering since the reproductive stage of the cultivars 14 and 21 DAT indicated some of the Group II soybean were beginning to flower or were at full flower at this point in time (Table 3).

The first appearance of *S. sclerotiorum* apothecia under the soybean canopy was observed 21 DAT when the mid- and late-Group II soybean were in full bloom and had peak flowering one week later. The relative humidity in the irrigated soybean canopy at the soil surface was 73 to 76% at 1230 h 19 DAT and did not differ between cultivars, herbicides, or years (data not presented). However, air temperature at the soil surface 19 DAT was slightly higher in the thifensulfuron (26.2 C) and lactofen (26.1 C) treatments compared to the untreated control (25.6 C) and glyphosate (25.6 C) treatments. Environmental conditions can affect apothecia production, ascospore germination, and mycelial growth (Abawi and Grogan 1975). A dense canopy with a cool and wet microclimate had a greater incidence of disease than an open, warm and dry canopy in

bean (Blad et al. 1978). Therefore, soybean injury caused by thifensulfuron and lactofen that delayed reproductive development, reduced canopy development, and influenced flower numbers plant^{-1} could influence the severity of white mold in glyphosate-resistant and non-resistant cultivars.

Detached Leaf and TLC Phytoalexin Bioassays. No recurrent differences between glyphosate-resistant and non-resistant isolines were observed using the *S. sclerotiorum* detached leaf bioassay (data not presented). Therefore, only data for the non-glyphosate resistant soybean cultivars are presented to demonstrate the validity of the bioassay and differences between cultivars through the sample period (Figure 5). *S. sclerotiorum* lesion diameter was greater for GL2415 and P9281 compared to S 12-49 and S 19-90 prior to herbicide treatments. This cultivar difference continued from 2 to 12 DAT. *S. sclerotiorum* lesion diameter was reduced by lactofen from 2 to 26 DAT compared to thifensulfuron (data not shown), the untreated control, or glyphosate treatments (Figure 6). The effect of thifensulfuron on lesion diameter was only evaluated on S 19-90 and S20-B9. None of the herbicide treatments affected the *S. sclerotiorum* lesion diameter of the eighth trifoliolate (untreated leaf) 26 DAT (data not shown).

The TLC phytoalexin bioassay indicated the presence of antifungal compounds that corresponded to Rf values previously reported for the methyl ester of glyceofuran (Rf 0.71); isoformonoetin and a mixture of glyceollin I, II, and III (Rf 0.5 to 0.56); and glyceofuran and the precursor of glyceollin II and III (Rf 0.25) (Ingham et al. 1981, Keen 1982). Soybean phytoalexin production did not differ due to cultivar from 0 to 7 DAT (data not presented). However, S 12-49 inhibited *C. cucumerinum* growth more than

S14-M7 while P9281 inhibited *C. cucumerinum* growth more than P93B01 12 DAT (Table 4). There was no difference in total antifungal compound production between cultivars for the fourth trifoliolate (herbicide treated) 26 DAT (data not presented). However, S 12-49 and S20-B9 produced more phytoalexins in the eighth trifoliolate (untreated leaves) than GL2415, GL2600, P9281, or P93B01 (Table 4).

Extracts from lactofen-treated plants inhibited *C. cucumerinum* growth more than the untreated control 2 to 26 DAT thus indicating increased phytoalexin production with this treatment (Figure 7). Glyphosate and thifensulfuron did not affect phytoalexin production compared to the untreated control. The diphenyl ether herbicides like lactofen inhibit protoporphyrinogen oxidase which causes the accumulation of protoporphryin IX (Scalla and Matringe 1994). The exposure to light causes the peroxidative destruction of membrane fatty acids and subsequent cell death (Scalla and Matringe 1994). Similar observations have been reported when plants undergo a hypersensitive response to disease infection (Baker and Orlandi 1995, Bhattacharyya and Ward 1986, Mehdy 1994, Sutton and Deverall 1984, Yoshikawa et al. 1978) or abiotic factors (Degousée et al. 1994, Ingham et al.1981) which have resulted in phytoalexin production. Phytoalexins reduced *S. sclerotiorum* growth *in vitro* (Sutton and Deverall 1984) and have been related to increased white mold tolerance of soybean treated with lactofen in the field (Dann et al. 1999).

White Mold Disease Severity. No difference in disease severity between glyphosate resistant and non-resistant near isoline cultivars was observed (Table 5). S 12-49, S14-M7, S 19-90, and S20-B9 had a lower incidence of disease than GL2415, GL2600,

P9281, and P93B01 (Table 5). A linear response between the detached leaf bioassay of *S. sclerotiorum* lesion diameter 0 to 26 DAT and disease severity at the end of the season was established by averaging the *S. sclerotiorum* lesion diameter mean for the six harvest dates of the fourth trifoliolate. Linear regression analysis indicated soybean with small lesion diameters had a low incidence of disease late in the season (P = 0.0001) (Figure 8).

White mold disease severity was reduced by all lactofen treatments compared to the untreated control (Table 5). Thifensulfuron or glyphosate did not affect white mold severity in the field compared to the untreated control (Table 5). This research does not support research that glyphosate (Holliday and Keen 1982, Keen et al. 1982, Sanogo et al. 2000) or ALS-inhibiting herbicides (Sanogo et al. 2000) increase the incidence of disease. Our research supports other research showing similar disease susceptibility in glyphosate-resistant and non-resistant cultivars (Lee and Penner 1999, Sanogo et al. 2000) and a reduction in the incidence of disease following a lactofen application (Dann et al. 1999, Sanogo et al. 2000).

Soybean Yield. No interaction between cultivar and herbicide treatment was observed for soybean yield; therefore, yield by cultivar combined over herbicide treatment is presented (Table 5). Soybean treated with lactofen, thifensulfuron, or glyphosate treatments had yields similar to the untreated control. Yield of GL2415, S 12-49, P9281, S14-M7, and S 19-90 was similar in the presence of white mold when combined over herbicide treatments. GL2415 was the only cultivar that yielded more than its glyphosate-resistant near isoline (GL2600). Seed of GL2600 was a fifth backcross derivative of GL2415 at this point in time. When data was sorted and analyzed by herbicide treatment,

GL2415 yielded 480 and 720 kg ha^{-1} more than GL2600 when treated with thifensulfuron and lactofen, respectively, and S 19-90 yielded 400 kg ha^{-1} more than S20-B9 when treated with lactofen.

SUMMARY

Apothecia, the fruiting bodies that produce ascospores which are responsible for *S. sclerotiorum* infection of soybean plants, were first observed (personal observation) in the field approximately 21 DAT (mid-July) (Figure 9). Soybean LAI during this time was 6.5 to 8.0 in 1998 and 6.5 to 7.5 in 1999 (Figure 1). The cottony "white mold" appeared approximately two weeks later on the lower portion of the soybean stem (Figure 9) and at other locations on the plants during the peak LAI period of the growing season (Figure 1).

Soybean tolerance to *S. sclerotiorum* was not related to glyphosate-resistance. Glyphosate-resistant and non-resistant soybean cultivars had similar canopy development (Figure 1), flower numbers (data not presented), *S. sclerotiorum* lesion diameters (data not presented), phytoalexin production (Table 4), incidence of white mold (Table 5), and yield (Table 5) except for GL2415 which yielded 390 kg ha^{-1} more than GL2600. Yield differences between GL2415 and GL2600 did not appear to be related to canopy development differences between near isolines (Figure 1). The yield difference between glyphosate-resistant and non-resistant soybean averaged over herbicide treatment was probably related to the combination of herbicide injury in the presence of white mold (Table 5) or this difference may be due to the genetic differences between the near isoline cultivars since GL2600 was only a fifth backcross derivative of GL2415.

In our research, cultivar selection had a large influence on reducing the incidence of white mold in the field (Table 5). The detached leaf bioassay utilized in this and other research (Dann et al. 1998 and 1999) may provide a simple method for predicting the relative sensitivity of soybean cultivars to *S. sclerotiorum* (Figure 8). Lesion diameter (Figure 5) and phytoalexin production (Table 4) indicated an inherent difference in cultivar susceptibility to *S. sclerotiorum*; however, maturity (Table 3), canopy development (Figure 1), and peak flowering (Figure 4) may also help to reduce disease in more tolerant cultivars like S 12-49, S14-M7, S 19-90, and S20-B9. In our research, later maturing cultivars had a greater incidence of white mold than earlier maturing cultivars which was similar to other studies (Boland and Hall 1987, Buzzell et al. 1993, Chun et al. 1987, Grau et al. 1982, Yang et al. 1999).

This research indicated that glyphosate did not affect soybean response, reproductive development, canopy development, flower number plant^{-1}, *S. sclerotiorum* lesion size, phytoalexin production, disease severity, or yield compared to untreated soybean. Table 6 summarizes the effects of thifensulfuron, and lactofen on the parameters evaluated in this research compared to no herbicide treatment. Thifensulfuron and lactofen affected soybean development, but only lactofen affected *S. sclerotiorum* lesion diameter, phytoalexin production, and disease severity. Therefore, increased phytoalexin was related to reduced disease in lactofen-treated soybean. However, lactofen affected canopy development (LAI) more than thifensulfuron (Figure 3). In this research, lactofen and thifensulfuron treatments probably did not affect soybean yield compared to untreated soybean because nearby plants are known to help compensate for yield losses (Hart 1998).

A reduction in disease severity would also reduce sclerotia production. Buzzel et al. (1993) reported a reduction in the number of sclerotia present in the soil when the incidence of white mold was reduced. Herbicide treatments and cultivars evaluated in this research that reduced the incidence of white mold may therefore reduce sclerotia production. The management of *S. sclerotiorum* sclerotia is similar to managing the weed seed bank in grain crops.

In summary, glyphosate-resistant and non-resistant soybean were equally susceptible to white mold; glyphosate did not affect the incidence of white mold in glyphosate-resistant soybean; and lactofen may be used as a tool to help manage white mold, but increased soybean yield may not always occur. This information will help soybean growers to make informed decisions on how weed management programs impact soybean development, incidence of white mold, and soybean yield.

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Table 1. Soybean growth characteristics, environmental conditions, and cultural practices that influence the infection and incidence of *S. sclerotiorum*.

Soybean growth characteristic, environmental condition, or cultural practice that was evaluated.	Source
Apothecia	Boland and Hall 1988b
Fertility	Chun et al. 1987
Flowering	Boland and Hall 1982, Boland and Hall 1988a, Grau et al. 1982, Grau and Radkey 1984
Height	Boland and Hall 1987, Kim et al. 1999
Injury	Chun et al. 1987
Inoculum	Abawi and Grogan 1975, Chun et al. 1987
Irrigation	Grau and Radkey 1984
Light	Chun et al. 1987, Boland and Hall 1988a, Pennypacker and Risius 1999
Lodging	Boland and Hall 1987, Buzzell et al. 1993, Kim et al. 1999
Maturity group	Boland and Hall 1987, Buzzell et al. 1993, Chun et al. 1987, Grau et al. 1982, Kim et al. 1999, Yang et al. 1999
Plant age	Chun et al. 1987
Rainfall	Boland and Hall 1982, Boland and Hall 1988a
Row spacing	Buzzell et al. 1993, Grau and Radkey 1984, Phipps 1983
Temperature	Buzzell et al. 1993, Chun et al. 1987, Grau and Radkey 1984, Nelson et al. 1991a, Pennypacker and Risius 1999

Table 2. Soybean injury from postemergence herbicides 3 and 7 d after treatment (DAT) in 1998 and 1999.[a]

	3 DAT				7 DAT			
Soybean cultivar	Untreated	Thifensulfuron	Lactofen	Glyphosate	Untreated	Thifensulfuron	Lactofen	Glyphosate
	%				%			
S 12-49	0	3	12	—[c]	0	5	16	—
S14-M7	0	3	11	0	0	3	16	0
S 19-90	0	3	10	—	0	3	13	—
S20-B9	0	3	8	0	0	6	13	0
GL2415	0	5	11	—	0	6	16	—
GL2600	0	5	10	0	0	7	15	0
P9281	0	2	11	—	0	5	15	—
P93B01	0	5	12	0	0	8	17	0
$LSD_{(p\leq 0.05)}$	2				3			

[a]Lactofen at 70 g ai ha^{-1} and thifensulfuron at 4.5 g ha^{-1} were applied with 0.25% v/v nonionic surfactant plus UAN (28% urea ammonium nitrate) at 2.3 L ha^{-1}. Glyphosate (formulated as Roundup Ultra®) at 840 g ha^{-1} was applied with UAN at 2.3 L ha^{-1}.
[b]Group I soybean included S 12-49, S14-M7, S 19-90, and S20-B9. Group II soybean included GL2415, GL2600, P9281, and P93B01.
[c]Treatment was not applied to non-resistant soybean.

Table 3. Reproductive stage of development for soybean cultivars combined over herbicide in 1998 and 1999 14 and 21 d after herbicide treatment (DAT).

	14 DAT		21 DAT	
Cultivar[b]	1998	1999	1998	1999
S 12-49	1.8	1.4	2.4	2.6
S14-M7	1.9	1.5	2.7	2.1
S 19-90	1.7	1.2	2.4	2.1
S20-B9	1.9	1.0	2.4	2.0
GL2415	1.7	0.5	2.0	1.9
GL2600	1.6	0.6	2.1	2.0
P9281	1.6	0.6	2.0	2.0
P93B01	1.0	0.2	2.0	1.4
$LSD_{(p\leq 0.05)}$	0.4	0.3	0.3	0.4

[a]Lactofen at 70 g ai ha^{-1} and thifensulfuron at 4.5 g ha^{-1} were applied with 0.25% v/v nonionic surfactant plus UAN (28% urea ammonium nitrate) at 2.3 L ha^{-1}. Glyphosate (formulated as Roundup Ultra®) at 840 g ha^{-1} was applied with UAN at 2.3 L ha^{-1}.
[b]Data were combined over herbicide treatments. Group I soybean included S 12-49, S14-M7, S 19-90, and S20-B9. Group II soybean included GL2415, GL2600, P9281, and P93B01.

Table 4. Inhibited area of the fourth (treated) trifoliolate 12 d after treatment (DAT) and the eighth trifoliolate 26 DAT for glyphosate-resistant and non-resistant soybean cultivars averaged over herbicide treatment using a *Cladosporium cucumerinum* TLC phytoalexin bioassay in 1998 and 1999.[a]

	12 DAT	26 DAT
Soybean cultivar[b]	fourth trifoliolate	eighth trifoliolate
	cm^2	
S 12-49	12.4	8.4
S14-M7	10.8	6.2
S 19-90	10.1	7.1
S20-B9	10.9	8.8
GL2415	10.1	5.8
GL2600	11.4	5.8
P9281	10.8	4.2
P93B01	9.2	4.7
$LSD_{(p\leq 0.05)}$	1.6	2.2

[a]Lactofen at 70 g ai ha^{-1} and thifensulfuron at 4.5 g ha^{-1} were applied with 0.25% v/v nonionic surfactant plus UAN (28% urea ammonium nitrate) at 2.3 L ha^{-1}. Glyphosate (formulated as Roundup Ultra®) at 840 g ha^{-1} was applied with UAN at 2.3 L ha^{-1}.
[b]Data were combined over the untreated control, lactofen, and glyphosate treatments.

Table 5. White mold disease severity index and soybean yield for glyphosate-resistant and non-resistant soybean isolines treated with postemergence herbicides in 1998 and 1999.[a]

Soybean cultivar	DSI					Yield				
	Untreated	Thifensulfuron	Lactofen	Glyphosate	Mean[b]	Untreated	Thifensulfuron	Lactofen	Glyphosate	Mean[b]
						kg ha^{-1}				
S 12-49	15	9	4	—	9	3650	4100	3690	—	3810
S14-M7	24	20	6	15	16	3590	3750	3700	3630	3670
S 19-90	24	25	10	—	20	3750	3420	3750	—	3640
S20-B9	28	21	11	24	21	3160	3420	3350	3810	3430
GL2415	54	46	27	—	42	3610	3840	4120	—	3860
GL2600	50	58	32	59	50	3490	3360	3400	3620	3470
P9281	54	54	27	—	45	3700	3630	3710	—	3680
P93B01	50	56	26	60	48	3650	3530	3460	3490	3530
LSD $_{(p\leq 0.05)}$	—	11	—	—	– 9 –	– NS –	– 420 –	– 380 –	– NS –	- 300 -
Mean[c]	37	36	18	39		3570	3630	3650	3630	
LSD $_{(p\leq 0.05)}$	—	4	—	—		—	—	NS	—	

[a]Lactofen at 70 g ai ha^{-1} and thifensulfuron at 4.5 g ha^{-1} were applied with 0.25% v/v nonionic surfactant plus UAN (28% urea ammonium nitrate) at 2.3 L ha^{-1}. Glyphosate (formulated as Roundup Ultra®) at 840 g ha^{-1} was applied with UAN at 2.3 L ha^{-1}.
[b]Cultivar main effect data were combined over the untreated control, thifensulfuron, lactofen, and glyphosate treatments.
[c]Herbicide main effect data were combined over cultivar.

Table 6. Summary of the effects of thifensulfuron and lactofen on soybean development, physiology, incidence of white mold and yield compared to untreated soybean.[a]

Parameter[b]	Thifensulfuron	Lactofen
1. Injury	Increased	Increased
2. Relative humidity 19 DAT[c]	No Effect	No Effect
3. Temperature 19 DAT[c]	Increased	Increased
4. Reproductive stage	Delayed	Delayed
5. Canopy development (LAI)	Reduced	Reduced
6. Flower number plant^{-1}	Reduced or No Effect	Reduced or Increased
7. *S. sclerotiorum* lesion diameter	No Effect[d]	Reduced
8. Phytoalexin production	No Effect[d]	Increased
9. Disease severity index	No Effect	Reduced
10. Yield	No Effect	No Effect

[a]Lactofen at 70 g ai ha^{-1} and thifensulfuron at 4.5 g ha^{-1} were applied with 0.25% v/v nonionic surfactant plus UAN (28% urea ammonium nitrate) at 2.3 L ha^{-1}.
[b]Abbreviations: DAT, days after treatment; and LAI, leaf area index.
[c]Measured at the soil surface.
[d]S 19-90 and S20-B9 were the only cultivars evaluated.

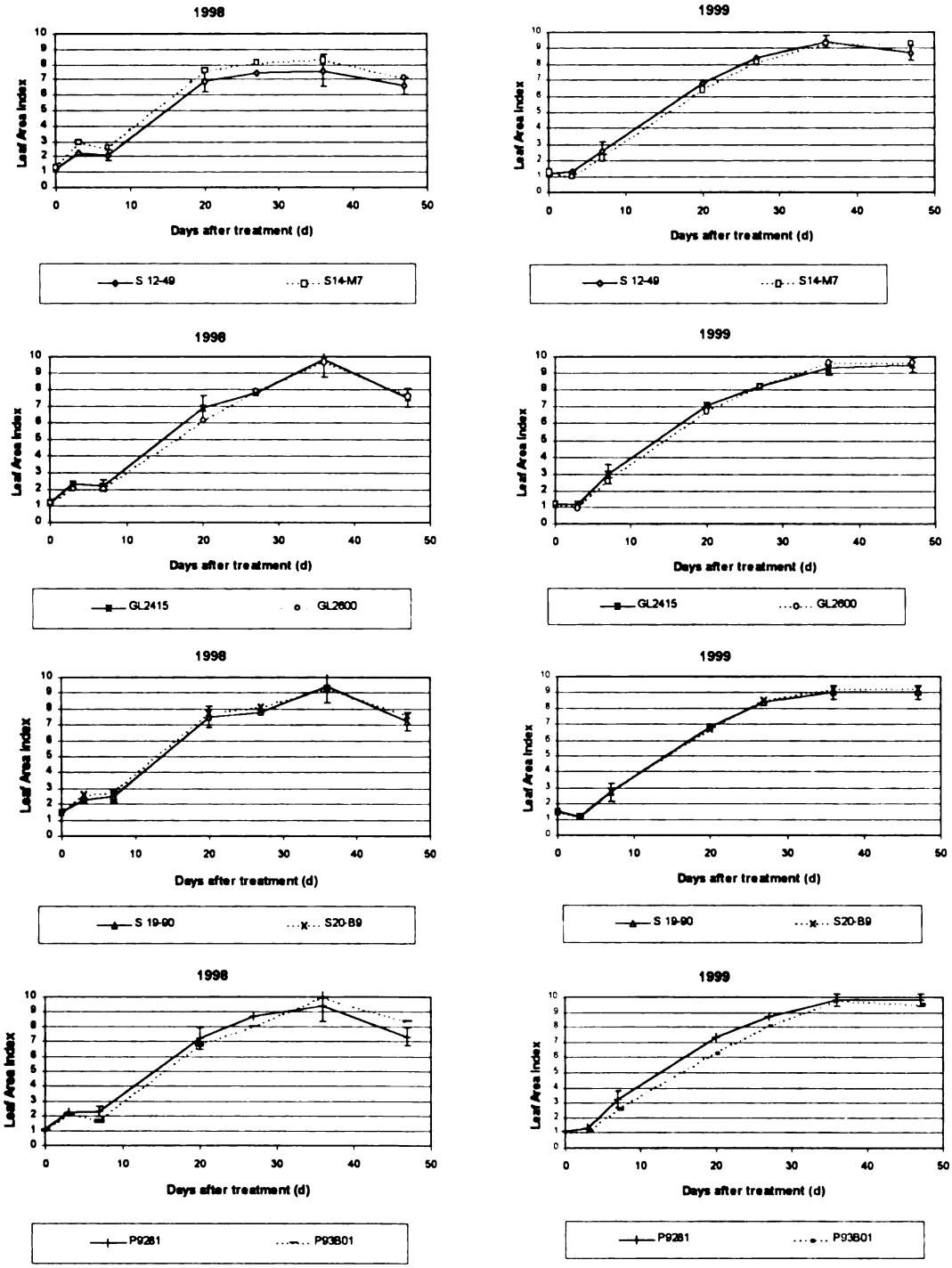


Figure 1. Soybean leaf area index for cultivar combined over herbicide treatment in 1998 and 1999. Vertical lines indicate the LSD ($p \leq 0.05$). Comparisons between cultivars for similar days within years are valid.

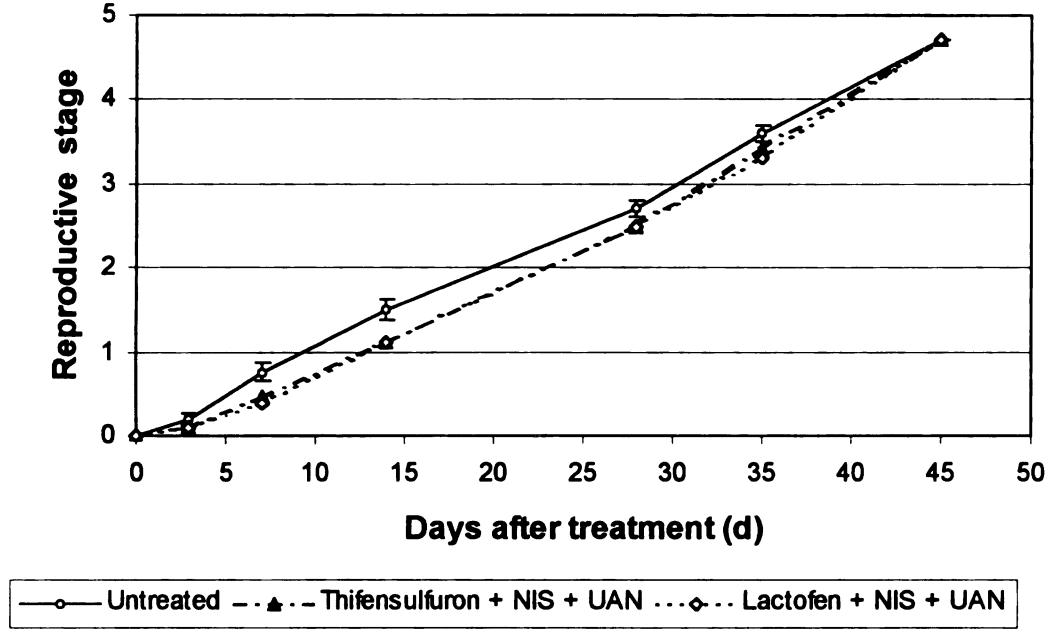
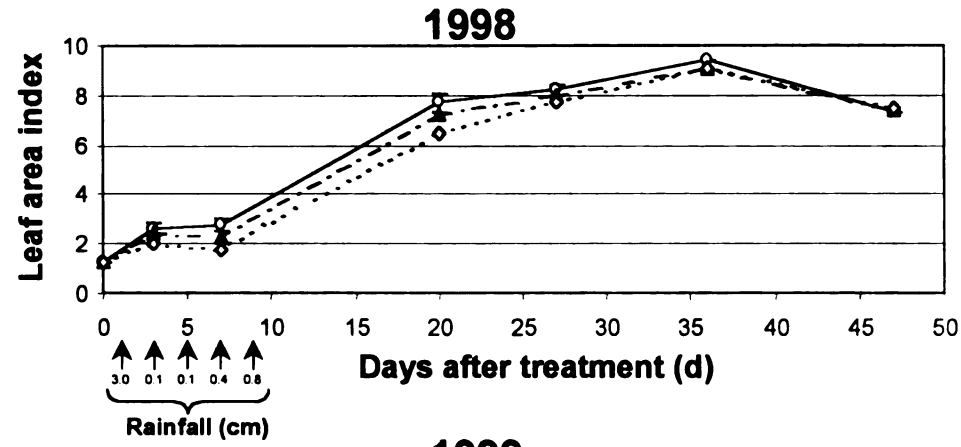


Figure 2. Soybean reproductive stage for the untreated control, thifensulfuron at 4.5 g ha^{-1} plus 0.25% v/v nonionic surfactant (NIS) plus 28% urea ammonium nitrate (UAN) at 2.3 L ha^{-1}, and lactofen at 70 g ai ha^{-1} plus 0.25% v/v nonionic surfactant plus UAN at 2.3 L ha^{-1} combined over cultivar in 1998 and 1999. Vertical lines indicate the LSD ($p \leq 0.05$).

a.



b.

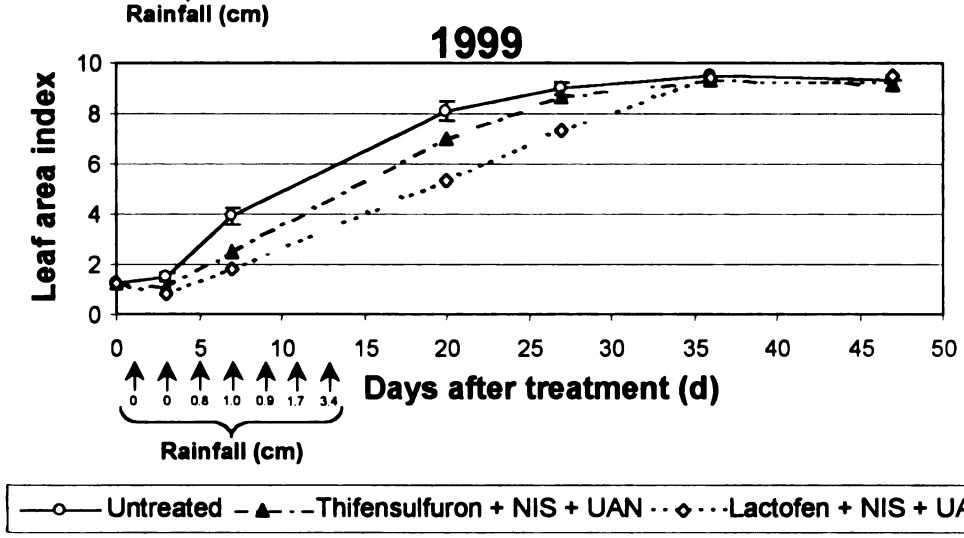
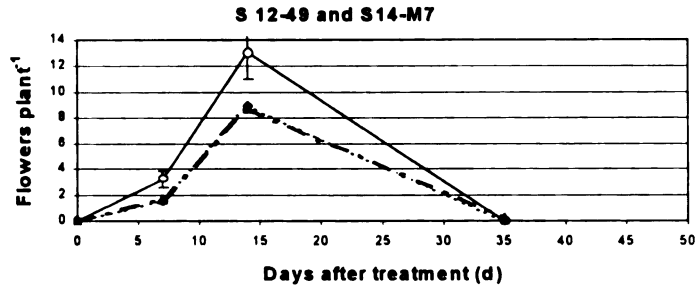


Figure 3. Soybean leaf area index for the untreated control, thifensulfuron at 4.5 g ha^{-1} plus 0.25% v/v nonionic surfactant plus UAN at 2.3 L ha^{-1} and lactofen at 70 g ai ha^{-1} plus 0.25% v/v nonionic surfactant plus UAN at 2.3 L ha^{-1} treatments combined over cultivar in 1998 and 1999. Vertical lines indicate the LSD ($p \leq 0.05$).

a.



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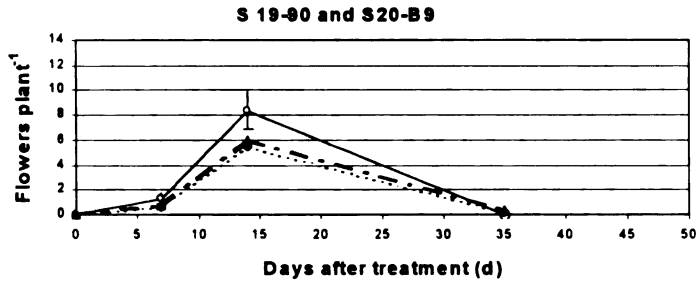

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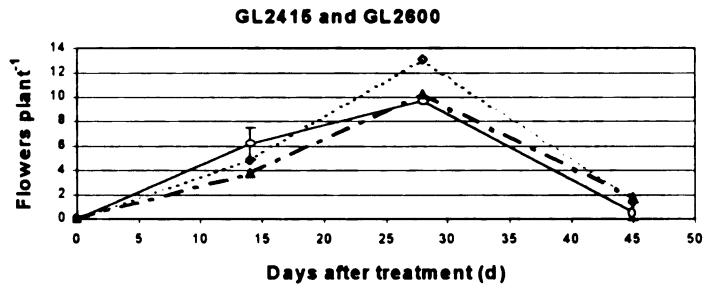
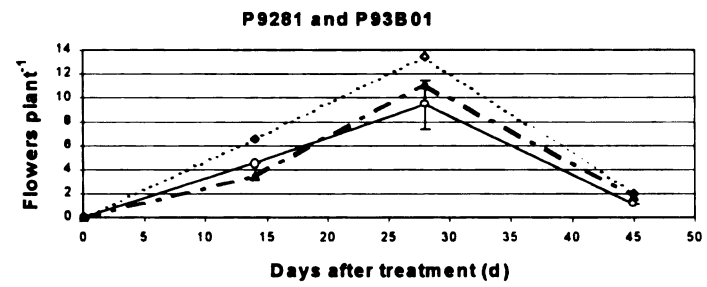


d.



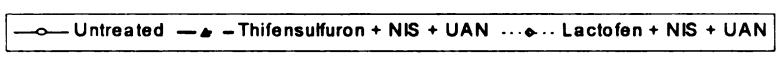


Figure 4. Soybean flowers plant^{-1} for thifensulfuron at 4.5 g ha^{-1} plus 0.25% v/v nonionic surfactant plus UAN at 2.3 L ha^{-1} and lactofen at 70 g ai ha^{-1} plus 0.25% v/v nonionic surfactant plus UAN at 2.3 L ha^{-1} combined over glyphosate-resistant and non-resistant isolines in 1998 and 1999. Group I soybean included S 12-49, S14-M7, S 19-90, and S20-B9. Group II soybean included GL2415, GL2600, P9281, and P93B01. Vertical lines indicate the LSD ($p \leq 0.05$).

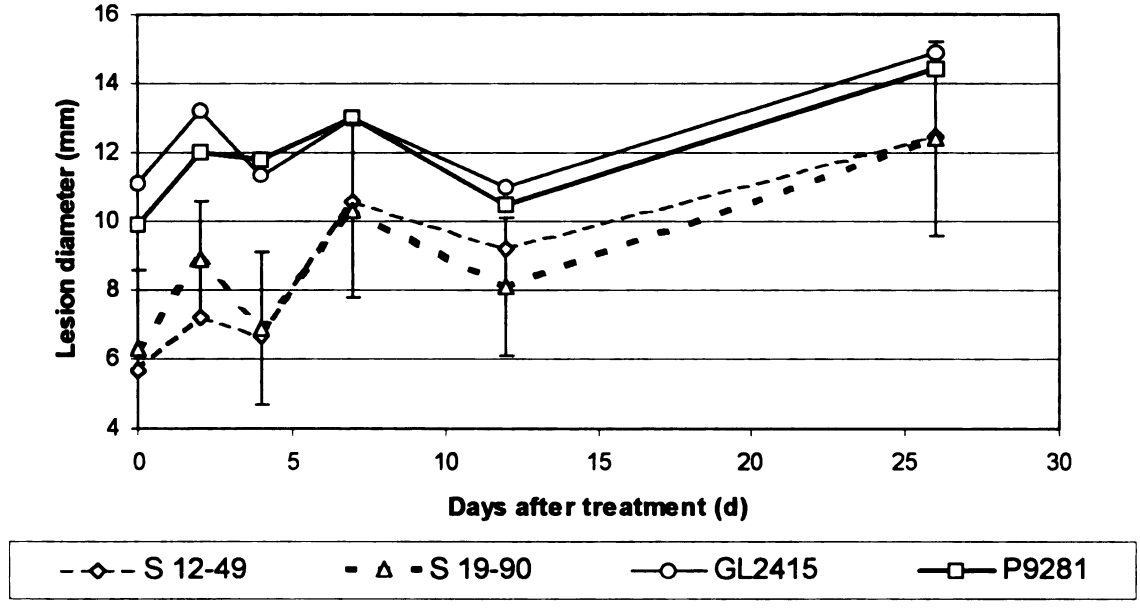


Figure 5. S. sclerotiorum lesion diameter of soybean cultivars using a detached leaf bioassay of the fourth trifoliolate (treated). Data were combined over herbicide treatments for soybean cultivars not resistant to glyphosate in 1998 and 1999. Vertical lines indicate the LSD ($p \leq 0.05$).

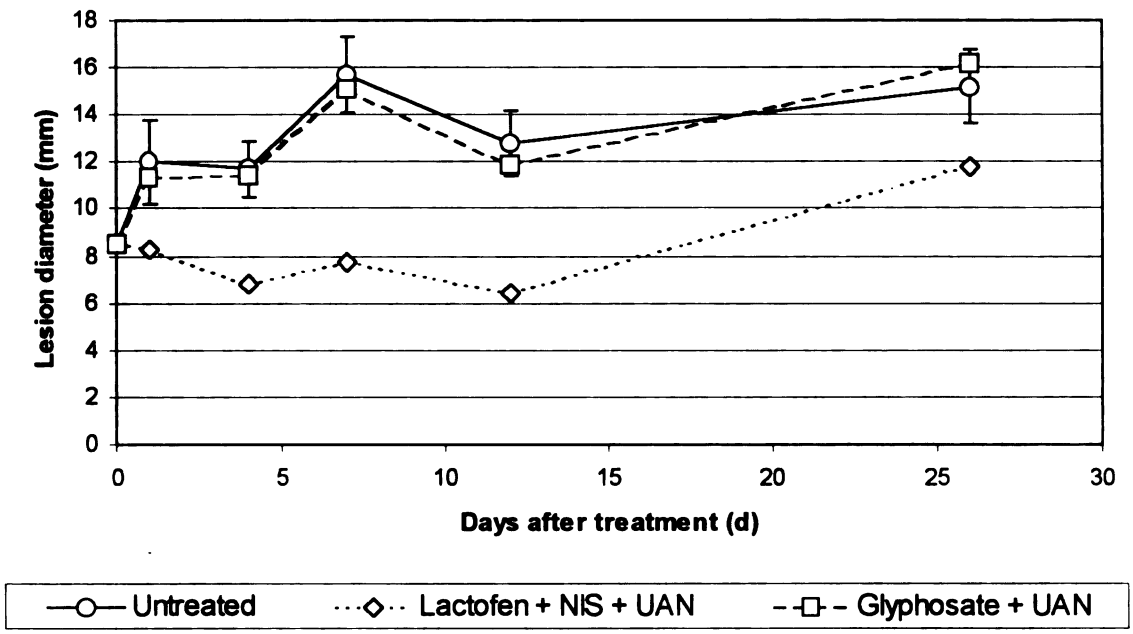


Figure 6. S. sclerotiorum lesion diameter using a detached leaf bioassay of the fourth trifoliolate treated with herbicides. Herbicide treatments included an untreated control, glyphosate at 840 g ha^{-1} plus 0.25% v/v nonionic surfactant plus UAN at 2.3 L ha^{-1}, and lactofen at 70 g ai ha^{-1} plus 0.25% v/v nonionic surfactant plus UAN at 2.3 L ha^{-1}. Data were combined over cultivars in 1998 and 1999. Vertical lines indicate the LSD ($p \leq 0.05$).

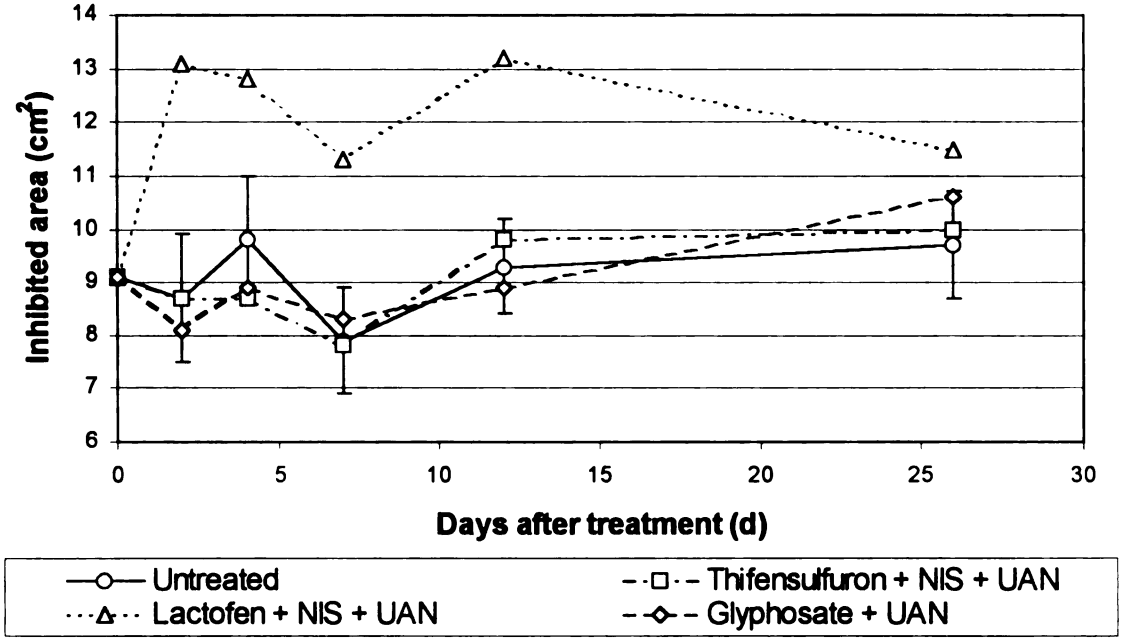


Figure 7. TLC phytoalexin bioassay using *Cladosporium cucumerinum.* The untreated control, thifensulfuron at 4.5 g ha^{-1} plus 0.25% v/v nonionic surfactant plus UAN at 2.3 L ha^{-1}, glyphosate at 840 g ha^{-1} plus 0.25% v/v nonionic surfactant plus UAN at 2.3 L ha^{-1}, and lactofen at 70 g ai ha^{-1} plus 0.25% v/v nonionic surfactant plus UAN at 2.3 L ha^{-1} treatments were combined over cultivars in 1998 and 1999. S 19-90 and S20-B9 were the only cultivars evaluated with thifensulfuron. Vertical lines indicate the LSD ($p \leq 0.05$).

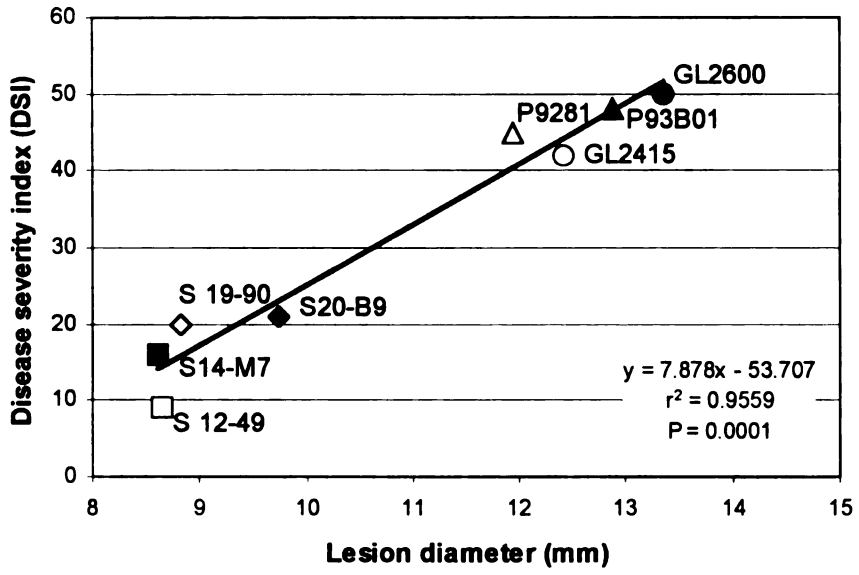


Figure 8. Linear regression of the detached leaf bioassay of *S. sclerotiorum* lesion diameter of the fourth trifoliolate (treated) averaged over harvest date (0 to 26 DAT) and disease severity index for glyphosate-resistant (solid shapes) and non-resistant (open shapes) cultivars. All data were combined over herbicide treatment in 1998 and 1999.

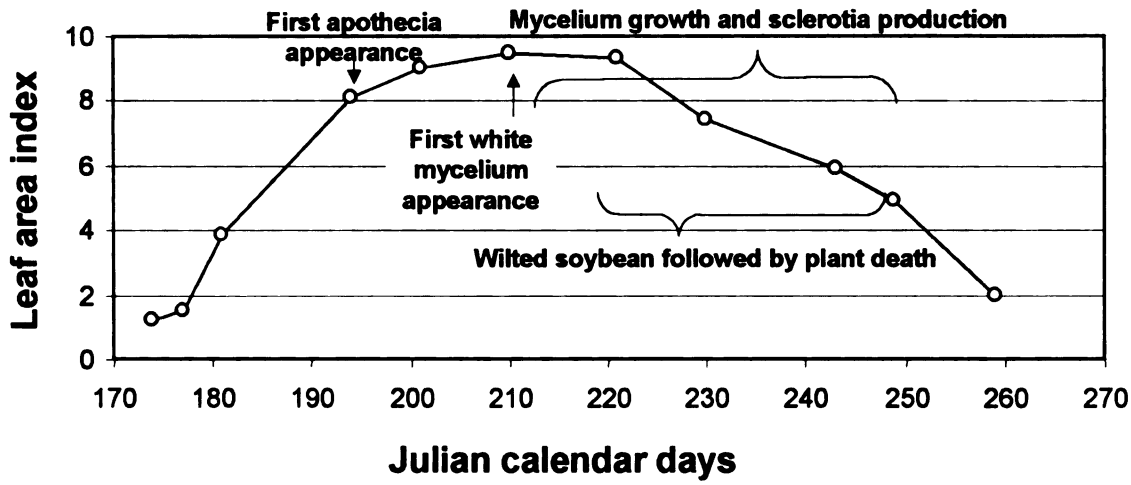


Figure 9. Development of *S. sclerotiorum* in soybean at East Lansing, MI.

CHAPTER 4

EFFECTS OF PROTOPORPHYRINOGEN OXIDASE INHIBITORS ON SOYBEAN (*Glycine max* L.) GROWTH RESPONSE, *Sclerotinia sclerotiorum* DISEASE DEVELOPMENT, AND PHYTOALEXIN PRODUCTION BY SOYBEAN

Abstract. Greenhouse research indicated protoporphyrinogen oxidase (protox)-inhibiting herbicides other than lactofen may provide additional options for white mold [*Sclerotinia sclerotiorum* (Lib.) de Bary] management. Research was conducted to determine soybean growth response, *Sclerotinia sclerotiorum* lesion development, and phytoalexin production in soybean treated with protox-inhibiting herbicides. Postemergence (POST) injury to soybean from oxyfluorfen at 17.5 g ai ha^{-1}, carfentrazone at 1.8 g ai ha^{-1}, sulfentrazone at 9.0 g ai ha^{-1}, fomesafen at 280 g ai ha^{-1}, acifluorfen at 425 g ai ha^{-1}, flumiclorac at 30 g ai ha^{-1}, CGA-248757 at 4 g ai ha^{-1}, and oxadiazon at 280 g ai ha^{-1} was equal to or less than injury from lactofen at 70 g ai ha^{-1}. Several protox-inhibiting herbicides reduced *S. sclerotiorum* lesion diameter in a detached leaf bioassay depending on the cultivar. All protox-inhibiting herbicides except acifluorfen and CGA-248757 reduced *S. sclerotiorum* lesion development on treated leaves compared to the untreated control plants in a detached leaf bioassay. Phytoalexin production in treated leaves and stems following a postemergence application of sulfentrazone and lactofen was determined. Lactofen and sulfentrazone increased leaf phytoalexin production similarly, but neither herbicide affected stem phytoalexin production compared to the untreated control. Sulfentrazone applied preemergence (PRE) at 210 g ha^{-1} reduced soybean fresh weight 8 to 69% depending on the cultivar and increased phytoalexin production of S 19-

90 and S20-B9 Roundup Ready® (RR) compared to untreated S 19-90 and S20-B9 (RR) cultivars. Sulfentrazone PRE also increased phytoalexin production of S 19-90 and S20-B9 (RR) cultivars compared to GL2415, GL2600 (RR), P9281, and P93B01 (RR) cultivars. This research indicated that a POST application of protox-inhibiting herbicides other than lactofen as well as a PRE application of sulfentrazone may increase phytoalexin production which could increase soybean tolerance to white mold.

Nomenclature: acifluorfen, 5-[2-chloro-4-(trifluoromethyl)phenoxy]-2-nitrobenzoic acid; carfentrazone, α,2-dichloro-5-[4-(difluoromethyl)-4,5-dihydro-3-methyl-5-oxo-1*H*-1,2,4-triazol-1-yl]-4-fluorobenzenepropanoic acid; CGA-248757, [[2-chloro-4-fluoro-5-[(tetrahydro-3-oxo-1*H*, 3*H*-[1,3,4]thiadiazolo[3,4-a]pyridazin-1-yliden)amino]phenyl]thio]acetate (proposed common name fluthiacet-methyl); flumiclorac, [2-chloro-4-fluoro-5-(1,3,4,5,6,7-hexahydro-1,3-dioxo-2*H*-isoindol-2-yl)phenoxy]acetic acid; fomesafen, 5-[2-chloro-4-(trifluoromethyl)phenoxy]-*N*-(methylsulfonyl)-2-nitrobenzamide; lactofen, (±)-2-ethoxy-1-methyl-2-oxoethyl 5-[2-chloro-4-(trifluoromethyl)phenoxy]-2-nitorbenzoate; oxadiazon, 3-[2,4-dichloro-5-(1-methylethoxy)phenyl]-5-(1,1-dimethylethyl)-1,3,4-oxadiazol-2-(3*H*)-one; oxyfluorfen, 2-chloro-1-(3-ethoxy-4-nitrophenoxy)-4-(trifluoromethyl)benzene; sulfentrazone, *N*-[2,4-dichloro-5-[4-(difluoromethyl)-4,5-dihydro-3-methyl-5-oxo-1*H*-1,2,4-triazol-1-yl]phenyl]methanesulfonamide; protox inhibitor, protoporphyrinogen oxidase inhibitor; soybean, *Glycine max* (L.) Merr.; white mold, *Sclerotinia sclerotiorum* (Lib.) de Bary.

Additional index words: aryl triazinone, cultivar, cyclic imide, diphenyl ether, herbicide resistant, isolines, oxadiazole, phytotoxicity, postemergence, protoporphyrinogen inhibitor, Roundup Ready®, white mold.

Abbreviations: 'Novartis S 19-90', 'S 19-90'; 'Novartis S20-B9', 'S20-B9'; 'Great Lakes 2415', 'GL2415'; 'Great Lakes 2600', 'GL2600'; 'Pioneer 9281', 'P9281'; 'Pioneer 93B01','P93B01'; DAT, d after treatment; NIS, nonionic surfactant; PDA, potato dextrose agar; UAN, 28% urea ammonium nitrate.

INTRODUCTION

A herbicide may increase, decrease, or have no effect on the incidence of disease depending on the herbicide mode of action, application timing, and application rate. Several reviews have summarized the effect of herbicides on disease (Altman and Cambell

1977, Katan and Eshel 1973, Lévesque and Rahe 1992). In Altman and Campbell's (1977) review, only seven postemergence herbicides were discussed. Since 1977, numerous postemergence herbicides have been discovered, introduced, and utilized for weed control in soybean and other agronomic crops. Recent studies (Table 1) have evaluated the effect of postemergence herbicides and adjuvants on the incidence and development of *Sclerotinia* spp. White mold is caused by the fungus *Sclerotinia sclerotiorum* (Lib.) de Bary. This disease is commonly found in soybean produced in the north central states and may reduce soybean yield under favorable environmental conditions (Chun et al. 1987, Hoffman et al. 1998, Kim et al. 1999, Yang et al. 1999).

The protoporphyrinogen oxidase (protox)-inhibiting herbicides are used for selective preplant incorporated, preemergence, post-directed, or postemergence weed control in soybean, corn (*Zea mays* L.), peanut (*Arachis hypogaea* L.), cotton (*Gossypium hirsutum* L.), rice (*Oryza sativa* L.), and other crops (Ahrens 1994, Hatzios 1998). The inhibition of protox in the chloroplasts and mitochondria with these herbicides causes an inhibition of heme and chlorophyll synthesis (Dayan et al. 1997, Scalla and Matringe 1994). An accumulation of protoporphyrinogen IX leads to an overflow of protoporphyrinogen IX into the thylakoid membrane which is oxidized to protoporphyrin IX (Scalla and Matringe 1994). In the presence of light, protoporphyrin IX generates singlet oxygen which can cause lipid peroxidation and membrane leakage which causes subsequent death of susceptible plant species (Dayan et al. 1997, Scalla and Matringe 1994). The protox-inhibiting herbicides include the cyclic imide (CGA-248757 and flumiclorac), diphenyl ether (acifluorfen, fomesafen, lactofen, and oxyfluorfen), oxadiazole

(oxadiazon), and aryl triazinone (carfentrazone and sulfentrazone) chemical families (Ahrens 1994, Hatzios 1998, Scalla and Matringe 1994). Lactofen (Hart et al. 1997, Wichert and Talbert 1993), acifluorfen (Hart et al. 1997), fomesafen (Hart et al. 1997), CGA-248757 (Fausey and Renner 1999), flumiclorac (Fausey and Renner 1999), sulfentrazone (Dayan et al. 1996, Krausz et al. 1998) are used for weed control in soybean.

The interaction between lactofen and the incidence of disease in soybean has been reported in other research. Lactofen reduced the incidence of *Fusarium solani* f. sp. *glycines* (Sanogo et al. 2000) and *S. sclerotiorum* (Dann et al. 1999, Nelson and Renner 1999) in soybean. Lactofen currently has a supplemental label for white mold suppression in soybean (Anonymous 1998). Acifluorfen and lactofen are known to increase phytoalexin production in soybean which may help reduce the incidence of disease in the field (Dann et al. 1999, Kömives and Cassida 1983, Levene et al. 1998, Nelson and Renner 1999). Phytoalexins are antimicrobial compounds produced by plants after disease infection or treatment with biotic or abiotic elicitors (Hammerschmidt 1999). Glyceollin production in 'Williams 82' and 'S 19-90' increased from 8.3 to 10.2 $\mu g\ g^{-1}$ when treated at the V4 stage with lactofen at 0.04 to 0.11 kg ha^{-1} 4 DAT (Dann et al. 1999). Phytoalexin production increased as early as 2 DAT and persisted up to 26 DAT following lactofen applied at the V5 stage in other research (Nelson and Renner 1999). Acifluorfen, bentazon, and nonionic surfactant applied at the V3 stage of development reduced soybean cyst nematode (*Heterodera glycines* Ichonohe) egg production by 50 to 60% and increased phytoalexin production 4 DAT (Levene et al. 1998). The effects of these and

other herbicides on the incidence of disease could affect soybean weed management practices in the north central states.

The susceptibility of soybean cultivars to *S. sclerotiorum* has been related to the parentage of the cultivar (Kim et al. 1999). Planting white mold tolerant or resistant soybean cultivars is the recommended strategy to reduce yield loss to white mold (Boland and Hall 1986, Boland and Hall 1987, Chun et al. 1987, Grau and Radkey 1984, Kim et al. 1999, Nelson et al. 1991b, Wegulo et al. 1998, Yang et al. 1999). Avoiding cultivars with parentage from 'Williams' or 'Asgrow A3127' may reduce the incidence of white mold (Kim et al. 1999). The effect of the protox-inhibiting herbicides on the incidence of white mold in glyphosate-resistant and non-resistant cultivars has been restricted to lactofen (Nelson and Renner 1999, Sanogo et al. 2000). The effects of many of the other protox-inhibiting herbicides on the incidence of disease has not been studied.

This research extends the evaluation of protox-inhibiting herbicides beyond lactofen to other diphenyl ether herbicides and the cyclic imide, oxadiazole, and aryl triazinone chemical families using a challenge inoculation of *S. sclerotiorum*. This research reports on the relative phytoalexin production between glyphosate-resistant and non-resistant cultivars, and the implications regarding the systemic effect of lactofen and sulfentrazone on phytoalexin production. Our objectives were to determine: 1) soybean tolerance to the protox-inhibiting herbicides, 2) the effect of protox-inhibiting herbicides treatments to a challenge inoculation of *S. sclerotiorum*, 3) the effect of sulfentrazone and lactofen on phytoalexin production in the stem and leaf, and 4) the effect of sulfentrazone applied preemergence on whole plant phytoalexin production.

MATERIALS AND METHODS

Greenhouse methods. Soybean were planted in 910 ml plastic pots filled with BACCTO potting soil[13]. Soybean were grown in a greenhouse with a 16-h photoperiod of natural and supplemental sodium vapor lighting which provided an additional photosynthetic photon flux density[14] of 120 μE $m^{-2} \cdot s^{-1}$. Herbicides were applied using a traveling-belt sprayer traveling at 1.5 km h^{-1}, delivering 234 L ha^{-1} at 193 kPa, and equipped with an 8001 even flat-fan nozzle[15]. All postemergence herbicide treatments included nonionic surfactant[16] (NIS) at 0.25% and 28% urea ammonium nitrate (UAN) at 2.3 L ha^{-1}.

Soybean tolerance.

Postemergence protox-inhibiting herbicide rate screen. 'S 19-90' soybean were planted and grown in the conditions previously described. Oxyfluorfen, carfentrazone, sulfentrazone, lactofen, fomesafen, acifluorfen, flumiclorac, CGA-248757, and oxadiazon were applied at several application rates with NIS and UAN. Herbicide treatments were applied at 1430 to 1600 h. Soybean were 13 cm tall and at the V2 stage of development (Fehr and Caviness 1977) at the time of application. Whole plant visual injury was rated on a scale from 0 (no visual injury, necrosis, chlorosis, or stunting) to 100 (complete plant death), and soybean height was measured and the percent height reduction

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[13]BACCTO is a product of Michigan Peat Co. Houston, TX 77098.

[14]LI-COR. 4421 Superior Street, Lincoln, NE 68504.

[15]Teejet flat-fan tips. Spraying Systems Co., North Ave. and Schmale Road, Wheaton, IL 60188.

[16]Nonionic surfactant was Activator-90, a mixture of alkyl polyoxyethylene ether and free fatty acids, Loveland Industries Inc., P.O. Box 1289, Greeley, CO 80632.

calculated 14 DAT. The application rate of each protox-inhibiting herbicide was selected based on injury that was similar or less than lactofen applied at 70 g ai ha^{-1}. The study had four replications and was repeated four times.

Necrosis of the first trifoliolate of S 19-90 and S20-B9 treated postemergence with protox-inhibiting herbicides. Growing conditions and herbicide applications methods in this experiment were described previously. Soybean were 13 cm tall and at the V2 growth stage of development at the time of application. Herbicide application rates (Table 2) that resulted in soybean injury equal to or less than lactofen were determined from the prior experiment. The whole plant was covered with a plastic bag with only the first trifoliolate exposed to the herbicide treatments. Visual necrosis to the treated trifoliolate was determined 7 DAT on a scale from 0 (no necrosis) to 100 (complete leaf necrosis). This experiment was a two factor factorial with three replications and was repeated in time. Factors included soybean cultivar and herbicide treatment.

***S. sclerotiorum* growth on protox-inhibiting herbicide treated and untreated leaves.** 'S 19-90', 'S20-B9', 'GL2415', 'GL2600', 'P9281', and 'P93B01' soybean cultivars were planted as previously described. Herbicide treatments were applied as previously described at the rates listed in Table 3. The whole plant was covered with a plastic bag with only the first trifoliolate exposed to the herbicide treatments. Soybean were at the V2 stage of development with the second trifoliolate beginning to open at the time of application. The first (treated) and second (untreated) trifoliolates were harvested from each treatment 7 DAT to determine if plant defense was localized or translocated to actively growing tissue in the soybean. The excised leaves were placed in a 150 by 15 mm

petri dish[17] with a moistened filter paper to ensure a humid environment for white mold growth. A PDA (potato dextrose agar) plug, 2.4 mm in diameter, from the margin of *S. sclerotiorum* was placed on the leaves and the horizontal, vertical, and tangential diameter of growth was measured with an electronic digital caliper after incubating 48 h. The three diameter measurements were averaged prior to an analysis of variance. This experiment was a three factor factorial with three replications and was repeated in time. Factors included cultivar, herbicide treatment, and harvested leaf.

Phytoalexin production in the leaf and stem. Herbicide treatments were applied as previously described. Soybean were 16 cm tall and at the V3 growth stage at the time of application. Treated leaves (first and second trifoliolate) and the stem plus petioles between the treated leaves were analyzed for phytoalexins using a TLC (thin-layer chromatography) phytoalexin bioassay. Leaves attached to the plant were harvested and evaluated in the phytoalexin bioassay.

The TLC phytoalexin bioassay utilized *Cladosporium cucumerinum* to determine and quantify antifungal compounds produced by soybean (Keen et al. 1971). This bioassay was used to evaluate the relative total antifungal activity (phytoalexin production) between treatments. Soybean leaves were weighed, cut into 0.5 to 1.0 cm pieces, and extracted with 40:60 ethanol:water v/v for 4 h. The extract was evaporated[18] at 40 C under reduced pressure to approximately 1 ml and transferred to 10 ml test tube. The flask was then rinsed with 1 ml water. The extract and water rinsate was combined

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[17]VWR Scientific Products, 800 E. Fabyan Pkwy., Batavia, IL 60510.

[18]Rotavapor, Büchi, Labortechnik AG, Postfach, CH-9230, Flawil.

and extracted three times with 1 ml ethyl acetate. The ethyl acetate extract was dried with anhydrous magnesium sulfate and evaporated under a flow of N_2. The residue was resuspended in ethyl acetate at 50 µl g^{-1} leaf fresh weight and applied to a silica-gel (250 µm) thin-layer chromatography plate[19] at 30 µl g^{-1} fresh weight. The plate was developed in an acetone:chloroform:ammonia hydroxide solution at 50:50:1 v/v. *C. cucumerinum* spores were suspended in a 100 ml solution of 0.7 g KH_2PO_4, 0.4 g KNO_3, 0.3 g Na_2HPO_4, 0.1 g $MgSO_4$, 0.1 g NaCl, and 5.0 g D-glucose that was adjusted to pH 6.5. A fine spray of suspended spores was applied to the TLC plate and the plate was placed in a sealed chamber with 100% humidity. Plates were incubated for 2 to 3 d and stored at -20 C. Photo copies[20] of the plates were made and clear areas with no *C. cucumerinum* growth were quantified with a leaf area meter[21]. Total areas were calculated that corresponded to Rf values for the methyl ester of glyceofuran (Rf 0.71); isoformonoetin and a mixture of glyceollin I, II, and III (Rf 0.5 to 0.56); and glyceofuran and the precursor of glyceollin II and III (Rf 0.25) previously reported using chloroform:acetone:NH_4OH (50:50:1 v/v) as the mobile phase and a silica-gel TLC plate as the stationary phase (Ingham et al. 1981, Keen 1982).

The sum of the total inhibited area was calculated and the change in phytoalexin production was equal to the difference in phytoalexin production between the untreated cultivar and the same cultivar treated with sulfentrazone or lactofen. The experiment was

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[19]Analtech, Inc., P. O. Box 7558, 75 Blue Hen Dr., Newark, DE 19713.

[20]Cannon, imageRUNNER 330S, One Cannon Plaza, Lake Success, NY 11042.

[21]LI-3000, LI-COR, 4421 Superior Street, Lincoln, NE 68504.

a three factor factorial with three replications and was repeated in time. The factors included herbicide treatment, soybean cultivar, and leaf or stem sample.

Phytoalexin production with sulfentrazone PRE. Two 'S 19-90', 'S20-B9', 'GL2415', 'GL2600', 'P9281', and 'P93B01' soybean seed were planted three cm deep in 910 ml plastic pots of Spinks loamy sand (sand, mixed, mesic Psammentic Hapludalfs) with pH 5.2 and 1.1% organic matter. Sulfentrazone at 210 g ha^{-1} was applied preemergence as previously described and pots were watered with 50 ml water to simulate 0.6 cm of rainfall for herbicide activation. Pots were misted lightly to ensure adequate moisture for germination. The whole plant was harvested 28 d after application, fresh weights were measured, and fresh weight reduction calculated for each cultivar. The whole plants were evaluated for phytoalexin production using the methods previously described. This study was a two factor factorial design with three pots for each of the four replications and was repeated in time. The factors were herbicide treatment and cultivar.

Statistical protocol. All research was arranged as a randomized complete block design. Data were subjected to an analysis of variance according to the factorial description in each section. Percent data for visual injury or necrosis were transformed to the arcsine prior to the analysis. The transformation did not affect the conclusions so original data is presented. Visual necrosis rating data for the experiment evaluating *S. sclerotiorum* growth on protox-inhibiting herbicide treated and untreated leaves were subjected to an *F Max* test for homogeneity (Kuehl 1994) and data were combined over run since variances

for both runs were homogenous. Data were combined over time and means separated using Fisher's Protected LSD at $p \leq 0.05$.

RESULTS AND DISCUSSION

Soybean Tolerance.

Postemergence protox-inhibiting herbicide rate screen. Lactofen injured soybean 8% and reduced height -2% 14 DAT (data not presented). Visual injury from oxyfluorfen at 17.5 g ha^{-1}, carfentrazone at 1.8 g ha^{-1}, fomesafen at 280 g ha^{-1}, acifluorfen at 425 g ha^{-1}, flumiclorac at 30 g ha^{-1}, CGA-248757 at 4 g ha^{-1}, and oxadiazon at 280 g ha^{-1} was from 6 to 14% and was no different from lactofen at 70 g ha^{-1} (data not presented). Sulfentrazone at 8.8, 17.5, and 32 g ha^{-1} reduced soybean height 18, 23, and 27%, respectively (data not presented). Acifluorfen also reduced soybean height by 15% (data not presented). Some herbicide treatments stimulated soybean height, but such observations have been reported with sub-lethal doses of herbicides which exhibit hormetic growth increases (Brain and Cousens 1989). Injury to soybean from sulfentrazone at 4.4 g ha^{-1} was 14%. Therefore, the rate was reduced in the following study. The accumulation of protoporphyrin IX in the presence of light results in the generation of singlet oxygen which causes the peroxidation of membranes and subsequent cell destruction (Dayan et al 1997b, Scalla and Matringe 1994). This results in the bronzing of soybean foliage which causes necrotic lesions characteristic of the injury symptoms observed.

Necrosis of the first trifoliolate of S 19-90 and S20-B9 treated postemergence with protox-inhibiting herbicides. NIS plus UAN did not cause soybean necrosis (Table 2).

Oxyfluorfen, acifluorfen, oxadiazon, and lactofen caused leaf necrosis that did not differ between herbicides and cultivars (Table 2). Soybean treated with fomesafen had necrosis equal to that of the other protox-inhibitors when applied to S20-B9, but not S 19-90. Sulfentrazone at 1.1 g ha^{-1} did not injure soybean. Therefore, further preliminary research determined the sulfentrazone rate (9 g ha^{-1}) which caused leaf necrosis equal to lactofen (data not presented).

***S. sclerotiorum* growth on protox-inhibiting herbicide treated and untreated soybean leaves.** Soybean response to challenge inoculations of *S. sclerotiorum* was dependent on the cultivar and herbicide treatment (Table 3). In the absence of herbicides, GL2415 and GL2600 had larger lesions than S 19-90 and S20-B9. A correlation between lesion diameter and disease severity in the field indicated cultivars with smaller lesion diameters had a lower incidence of white mold in the field (Nelson and Renner 1999). There was no difference in lesion diameters between untreated controls of glyphosate-resistant and non-resistant near isolines 7 DAT (Table 3). These results were similar to field research using the same near isogenic cultivars (Nelson and Renner 1999). NIS plus UAN did not affect *S. sclerotiorum* lesion diameter compared to untreated soybean; therefore, the adjuvant and fertilizer portion of the herbicide treatment may not affect the incidence of white mold. Similarly, adjuvants did not affect the incidence of white mold in field research (Lee and Penner 1999), and the addition of adjuvants to protox-inhibiting herbicides (Levene et al. 1998) did not have an additive effect on reducing soybean cyst nematode reproduction. However, adjuvants are known to increase phytotoxicity (Dayan

et al. 1996, Fausey and Renner 1999) and foliar absorption (Dayan et al. 1996) of the protox-inhibiting herbicides.

The *S. sclerotiorum* lesion diameter differed by cultivar and herbicide treatment (Table 3). Oxifluorfen and lactofen reduced *S. sclerotiorum* lesion diameter of five of the cultivars evaluated compared to the untreated controls. All treatments averaged over cultivar reduced *S. sclerotiorum* lesion diameter compared to the untreated control (Table 3). Oxifluorfen, sulfentrazone, and oxadiazon reduced lesion size similar to lactofen; however, sulfentrazone and lactofen are the only herbicides labeled for weed control in soybean (Crop Protection Chemicals Reference 1998).

Treated and untreated soybean leaves responded differently to challenges of *S. sclerotiorum* depending on the herbicide treatment (Table 3). The second trifoliolate (untreated) of untreated, NIS plus UAN, oxyfluorfen, carfentrazone, sulfentrazone, lactofen, fomesafen, treatments had larger *S. sclerotiorum* lesion diameters than the first trifoliolate (treated). This may be due to a thinner cuticle and younger tissue which may allow easier disease penetration by a disease. Similarly, older soybean leaves were more resistant to a *Phytophthora megasperma* challenge on both susceptible or resistant cultivars compared to less mature leaves (Bhattaaryya and Ward 1986). NIS and UAN did not affect *S. sclerotiorum* lesion diameter of the first (treated) or second (untreated) trifoliolate compared to untreated soybean. All protox-inhibiting herbicides reduced *S. sclerotiorum* lesion diameter compared to untreated plants when averaged over cultivar except the first trifoliolate treated with acifluorfen and CGA-248757. All protox-inhibiting herbicides reduced *S. sclerotiorum* lesion diameter on the second trifoliate.

A reduction in lesion diameter in the second trifoliate was expected since acifluorfen plus NIS increased root phytoalexin levels 4 DAT (Levene et al. 1998). The protox-inhibiting herbicide treatments evaluated induced a systemic induced resistance response that caused a reduction in the lesion diameter of *S. sclerotiorum* in the untreated trifoliolate.

Oxyfluorfen and lactofen provided consistent *S. sclerotiorum* suppression. Oxyfluorfen caused 48 to 87% necrosis while lactofen caused 24 to 48% necrosis (data not presented). Oxadiazon and acifluorfen caused injury similar or greater than that from oxyfluorfen or lactofen (data not presented), but did not consistently reduce *S. sclerotiorum* lesion diameter. After soybean was inoculated with *S. sclerotiorum*, browning of the epidermal cells within 24 h resembled a hypersensitive response (Sutton and Deverall 1984). The protox-inhibiting herbicides may mimic such a hypersensitive response that results in the production of phytoalexins.

Several of the protox-inhibiting herbicide treatments evaluated suppressed *S. sclerotiorum* lesion diameter growth. However, since sulfentrazone is labeled for weed control in soybean, a reduced rate of sulfentrazone (96% of the preemergence application rate) could be a cost-effective treatment for white mold suppression. Other researchers have reported severe postemergence injury from sulfentrazone (35 to 55%) at 34 to 56 g ha^{-1} (Dayan et al. 1996). Further research should evaluate the postemergence tolerance of soybean to sulfentrazone in the field.

Phytoalexin production in the leaf and stem. Lactofen was selected in this experiment as a standard because it consistently reduced *S. sclerotiorum* diameter in the previous experiment (Table 3) and its effect on phytoalexin production has been previously

reported (Dann et al. 1999, Nelson and Renner 1999). Sulfentrazone was selected because this herbicide reduced *S. sclerotiorum* lesion size nearly as much as lactofen in the previous study (Table 3) and a reduced rate of this herbicide could provide cost-effective white mold suppression. Leaf and stem fresh weight and phytoalexin production in this study differed by herbicide treatment (Table 4). Lactofen and sulfentrazone reduced soybean leaf fresh weight more than stem fresh weight which could be related to abscised leaves and necrosis caused by the herbicide treatment. Lactofen and sulfentrazone reduced stem fresh weight 8 to 12%, but had no effect on total phytoalexin production and had no net effect on the relative phytoalexin production in the stem. Total phytoalexin production of the untreated control was similar in the leaf and stem when averaged over cultivar. Lactofen and sulfentrazone did not induce phytoalexin production in the stem, but increased phytoalexin production in the leaf compared to untreated soybean.

Total antifungal production was greater in S 19-90 and S20-B9 compared to GL2415 or GL2600 (Table 5). S20-B9 produced more total leaf phytoalexins than S 19-90 while P93B01 produced more total leaf phytoalexins than P9281. All cultivar leaves had more total and relative phytoalexin production compared to stems regardless of cultivar.

We did not observe an increase in total phytoalexin production in the stem when plants were treated with lactofen or sulfentrazone (Table 4). *S. sclerotiorum* lesions usually originate from the leaf axils of the lower soybean nodes and develop along the length of the stem which causes the premature death of the entire soybean plant (Cline and Jacobsen 1983, Boland and Hall 1982, Grau et al. 1982). *S. sclerotiorum* mycelial growth

might be inhibited if there was an accumulation of phytoalexins in the stem (Sutton and Deverall 1984) which could help explain a reduction in disease severity by lactofen (Dann et al. 1999, Nelson and Renner 1999).

Phytoalexin production is a good indicator of defense expression (Dann et al. 1999, Hammerschmidt 1999, Levene et al. 1998). However, the protox-inhibiting herbicides generate active oxygen that may activate other soybean defense mechanisms. Active oxygen species produced in response to a pathogen have been considered elicitors in a hypersensitive response and responsible for cell wall lignification and lipid peroxidation (Baker and Orlandi 1995, Mehdy 1994, Sutherland 1991). The mechanical and diffusion barriers formed as a result of an oxidative burst may also explain reduced *S. sclerotiorum* lesion growth. A combination of defense mechanisms other than phytoalexin production alone may contribute to a reduction in *S. sclerotiorum* lesion growth.

Phytoalexin production with sulfentrazone PRE. Sulfentrazone is applied PRE at 210 g ha^{-1} in Michigan and other states for weed control in soybean (Crop Protection Chemicals Reference 1998). Sulfentrazone PRE reduced fresh weight of all soybean cultivars except GL2415 compared to untreated cultivars (Table 6). Fresh weight was reduced 8 to 69% compared to the untreated control depending on the cultivar. Sulfentrazone reduced GL2600 fresh weight 43% more than GL2415. Differences in soybean cultivar tolerance to sulfentrazone have been reported (Dayan et al. 1996 and 1997b, Swantek et al. 1998). Herbicide tolerance for sensitive and tolerant varieties was related to the rapid metabolism, but differences in sensitivity were also related to the tolerance of the cultivars to the stresses caused by peroxidation (Dayan et al 1997b).

Phytoalexin production was greater in S 19-90 and S20-B9 treated with sulfentrazone than untreated soybean and GL2415, GL2600, P9281, or P93B01 treated with sulfentrazone. Whole plant phytoalexin analysis ranked phytoalexin production S 19-90 = S20-B9 > GL2415, GL2600, P9281, and P93B01.

Soybean tolerance to the protox-inhibiting herbicides varies and is rate dependent. Several of the protox-inhibiting herbicides reduced *S. sclerotiorum* lesion diameter. The effect of the protox-inhibiting herbicides appeared to be systemic and increased in leaves that were actively growing at the time of application, yet not in the stem tissue. However, a reduction of *S. sclerotiorum* lesion diameter was not always related to the degree of leaf tissue necrosis following a herbicide treatment. Sulfentrazone at 9 g ha^{-1} increased leaf phytoalexin production similar to lactofen, but neither treatment affected phytoalexin production in the stem. Sulfentrazone PRE may suppress white mold if soybean phytoalexin production remained high from nine to ten weeks following planting and application. However, soybean cultivar tolerance would be important to consider if this treatment was used for white mold suppression. Other postemergence herbicides with a mode of action similar to lactofen may provide white mold suppression. Further considerations of the cost-effectiveness, phytotoxicity, and efficacy of these treatments are needed.

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Table 1. Effect of herbicides and adjuvants on *Sclerotinia* spp.

Herbicide	*Sclerotinia* spp.	Effect	Source
Acifluorfen	*S. sclerotiorum*	Reduced DSI at one location.	Dann et al. 1999
Alachlor	*S. sclerotiorum*	Variable effects on sclerotia germination.	Radkey and Grau 1986
Atrazine	*S. sclerotiorum*	No effect on stipes or sclerotia. Abnormal apothecia were caused by the active ingredient. No ascospores were produced.	Casale and Hart 1986
	S. sclerotiorum	Stimulated sclerotia germination and increased the number of apothecia that germinated and stipes.	Radkey and Grau 1986
Bentazon	*S. sclerotiorum*	No effect on DSI.	Dann et al. 1999
Bromoxynil	*S. trifoliorum*	Increased DSI in controlled environment, but had no effect in the field.	Reichard et al. 1997
Chlorimuron	*S. sclerotiorum*	No effect on DSI.	Dann et al. 1999
Cloramben	*S. sclerotiorum*	Variable effects on sclerotia germination.	Radkey and Grau 1986
Crop oil adjuvant	*S. sclerotiorum*	No effect on DSI	Dann et al. 1999, Lee and Penner 1999
2,4-DB	*S. trifoliorum*	Reduced DSI in controlled environment, but had no effect in the field.	Reichard et al. 1997
DASH HC[b]	*S. trifoliorum*	Increased DSI in controlled environment, but had no effect in the field.	Reichard et al. 1997
DNBP	*S. sclerotiorum*	Inhibited sclerotia germination and prevented growth at high rates.	Radkey and Grau 1986
Diuron	*S. sclerotiorum*	Inhibited mycelial growth. Normal apothecia were produced. Reduced growth more than the triazine herbicides.	Casale and Hart 1986
Fomesafen	*S. sclerotiorum*	No effect on DSI.	Dann et al. 1999
Glyphosate	*S. sclerotiorum*	No effect on DSI.	Lee and Penner 1999, Nelson and Renner 1999, Penner et al. 1997
	S. sclerotiorum	No effect on white mold incidence when applied to a glyphosate-resistant soybean cultivar.	Penner et al. 1997
Imazethapyr	*S. trifoliorum*	No effect on DSI in controlled environment or in the field.	Reichard et al. 1997
	S. sclerotiorum	No effect on DSI.	Dann et al. 1999

Table 1 (cont'd).

Lactofen	*S. sclerotiorum*	Reduced DSI under high disease pressure, but had no effect at low disease pressures. DSI was related to the herbicide rate. DSI was reduced and phytoalexin production increased 2 to 26 d after treatment.	Dann et al. 1999, Nelson and Renner 1999
Linuron	*S. sclerotiorum*	Inhibited sclerotia germination.	Radkey and Grau 1986
Metribuzin	*S. sclerotiorum*	Inhibited mycelial growth. Normal apothecia were produced. Prevented carpogenic germination.	Casale and Hart 1986
	S. sclerotiorum	Stimulated sclerotia and increased the number of apothecia that germinated and stipes.	Radkey and Grau 1986
Nonionic surfactant	*S. sclerotiorum*	No effect on DSI.	Lee and Penner 1999
Pentachlorophenol	*S. laxa*	Reduced the incidence of disease on almond.	Katan and Eshel 1973
Pendimethalin	*S. sclerotiorum*	Stimulated sclerotia and increased the number of apothecia that germinated.	Radkey and Grau 1986
Pronamide	*S. trifoliorum*	Reduced DSI in controlled environment, but had no effect in the field.	Reichard et al. 1997
Sethoxydim	*S. trifoliorum*	Increased DSI in controlled environment, but had no effect in the field.	Reichard et al. 1997
Simazine	*S. sclerotiorum*	No effect on stipes or sclerotia. Apothecia were abnormal. No ascospores were produced.	Casale and Hart 1986
	S. sclerotiorum	Stimulated sclerotia and increased the number of apothecia that germinated and stipes.	Radkey and Grau 1986
Thifensulfuron	*S. sclerotiorum*	No effect on DSI.	Dann et al. 1999, Nelson and Renner 1999
Trifluralin	*S. sclerotiorum*	Stimulated sclerotia and increased the number of apothecia that germinated.	Radkey and Grau 1986

[a]Petroleum based adjuvant.

[b]Abbreviations: DSI, disease severity index.

Table 2. Necrosis of the first trifoliate of S 19-90 and S20-B9 7 DAT treated with a postemergence application of protox-inhibiting herbicides in the greenhouse.

		Necrosis	
Herbicide treatment	Rate	S 19-90	S20-B9
	g ai ha^{-1}	%	
Untreated		0	0
NIS + UAN		0	0
Oxyfluorfen + NIS + UAN	17.5	78	79
Carfentrazone + NIS + UAN	1.8	19	1
Sulfentrazone + NIS + UAN	1.1	0	0
Lactofen + NIS + UAN	70	63	63
Fomesafen + NIS + UAN	280	33	57
Acifluorfen + NIS + UAN	425	72	77
Flumiclorac + NIS + UAN	30	18	16
CGA-248757 + NIS + UAN	4	18	8
Oxadiazon + NIS + UAN	280	64	60
$LSD_{(p\leq 0.05)}$		18	

[a]NIS and UAN were applied at 0.25% v/v and 2.3 L ha^{-1}, respectively.

Table 3. Diameter of the *S. sclerotiorum* lesion on leaves of S 19-90, S20-B9, GL2415, GL2600, P9381, and P93B01 treated with protox-inhibiting herbicides. Data were averaged over the first and second trifoliolate when presented by cultivar. Data for *S. sclerotiorum* lesion diameter on leaves of the first (treated) and second (untreated) soybean trifoliolate treated with protox-inhibiting herbicides were averaged over cultivar.

		Cultivar							Trifoliolate	
Herbicide Treatment[a]	Rate	S 19-90	S20-B9	GL2415	GL2600	P9281	P93B01	Mean[b]	first (treated)	second (untreated)
	g ai ha^{-1}	mm								
Untreated		7.2	7.9	12.4	14.6	9.7	10.2	10.3a	8.8	11.9
NIS + UAN		7.3	7.0	10.5	13.2	9.8	11.4	9.9a	8.8	11.0
Oxyfluorfen + NIS + UAN	17.5	3.6	3.2	5.9	6.1	6.6	4.3	5.2d	3.3	6.6
Carfentrazone + NIS + UAN	1.8	4.9	6.9	5.8	8.4	8.6	9.2	7.3b	6.1	8.5
Sulfentrazone + NIS + UAN	9	5.4	5.6	8.1	4.3	7.8	8.1	6.6bcd	5.2	7.9
Lactofen + NIS + UAN	70	3.9	5.1	8.1	6.2	5.0	5.5	5.6cd	4.9	6.4
Fomesafen + NIS + UAN	280	5.5	5.4	10.0	9.6	6.2	6.1	7.1b	4.8	9.5
Acifluorfen + NIS + UAN	425	6.3	4.9	9.4	10.5	7.6	6.7	7.6b	7.4	7.8
Flumiclorac + NIS + UAN	30	4.6	6.7	5.7	8.5	8.2	9.1	7.1b	6.5	7.8
CGA-248757 + NIS + UAN	4	6.2	7.7	5.5	9.2	9.3	8.0	7.6b	7.0	8.3
Oxadiazon + NIS + UAN	280	4.6	4.8	10.5	4.3	7.9	8.1	6.7bcd	6.6	6.8
$LSD_{(p\leq 0.05)}$		3.3							1.9	

[a]NIS and UAN were applied at 0.25% v/v and 2.3 L ha^{-1}, respectively.

[b]Mean of herbicide treatments combined over cultivar, trifoliolate, and run. Means followed by the same letter are not significantly different ($p\leq 0.05$).

Table 4. Fresh weight reduction and TLC phytoalexin bioassay of phytoalexin production in the leaf and stem of soybean treated with lactofen and sulfentrazone averaged over S 19-90, S20-B9, GL2415, GL2600, P9281, and P93B01 cultivars.

				TLC phytoalexin bioassay			
		Fresh weight reduction		Inhibition area		Δ inhibition area[a]	
Treatment[b]	Rate	Stem	Leaf	Stem	Leaf	Stem	Leaf
	g ai ha^{-1}	%		cm^2			
Untreated		0	0	2.1	3.0	0	0
Lactofen	70	12	49	2.0	7.6	-0.2	4.6
Sulfentrazone	9	8	41	1.5	7.5	-0.6	4.4
$LSD_{(p\leq0.05)}$		7		1.0		0.9	

[a]Δ inhibition area on the TLC plate was the difference between the treated plants and the untreated plants for each cultivar to calculate a relative phytoalexin production for each cultivar.

[b]All herbicide treatments included nonionic surfactant at 0.25% v/v and 28% urea ammonium nitrate at 2.3 L ha^{-1}.

Table 5. Stem and leaf weight reduction and phytoalexin production of S 19-90, S20-B9, GL2415, GL2600, P9281, and P93B01 soybean cultivars averaged over untreated, lactofen, and sulfentrazone herbicide treatments.

			TLC phytoalexin bioassay			
	Fresh weight reduction		Inhibition area		Δ inhibition area	
Cultivar	Stem	Leaf	Stem	Leaf	Stem	Leaf
	%		cm^2		cm^2	
S 19-90	6	22	3.0	5.9	-0.5	3.5
S20-B9	5	28	2.7	7.8	-0.5	4.2
GL2415	9	35	1.2	4.6	0.2	1.9
GL2600	1	24	1.0	4.6	-0.4	2.0
P9281	10	39	1.5	5.5	0.2	3.0
P93B01	9	32	1.9	7.9	-0.7	3.3
$LSD_{(p\leq 0.05)}$	NS		1.4		1.3	

[a]Δ inhibitition area on the TLC plate was the difference between the treated plants and the untreated plants for each cultivar to calculate a relative phytoalexin production for each cultivar.

Table 6. Fresh weight, fresh weight reduction, and TLC phytoalexin bioassay for phytoalexin production of sulfentrazone applied preemergence on soybean cultivars.

	Fresh weight		Fresh weight reduction[b]		TLC phytoalexin bioassay inhibition area		
Soybean cultivar	Untreated	Sulfentrazone[a]	Untreated	Sulfentrazone	Untreated	Sulfentrazone	Mean[c]
	g plant^{-1}		%		cm^2		
S 19-90	2.4	0.8	0	69	3.2	8.1	5.7
S20-B9	2.3	0.7	0	66	3.8	9.1	6.4
GL2415	1.1	0.9	0	8	2.5	2.1	2.3
GL2600	1.5	0.7	0	51	1.8	2.8	2.3
P9281	1.3	0.8	0	34	1.8	3.1	2.4
P93B01	1.3	1.0	0	25	1.6	2.4	2.0
$LSD_{(p\leq0.05)}$	0.3		25		2.7		1.9

[a]Sulfentrazone was applied at 210 g ha^{-1}.
[b]Calculated as a percent of the untreated cultivar.
[c]Data were averaged over herbicide treatment.

CHAPTER 5

YELLOW NUTSEDGE LITERATURE REVIEW

Yellow nutsedge (*Cyperus esculentus* L.) is a problematic perennial weed found throughout the world (Bendixen and Nandihalli 1987, Lapham 1985, Schippers et al. 1995). The anatomy, morphology, chemical composition, and growth of yellow nutsedge were reviewed by Doll (1983) and Wills (1987). Yellow nutsedge is a prolific weed primarily due to its ability to propagate through rhizomes and tubers. Several buds on a tuber allow for easy reinfestation after tillage or selective herbicide treatments. In reviews by Glaze (1987) and William and Bendixen (1987), infestation prevention, crop selection and rotation, row spacing, tillage, fumigation, and herbicide treatments were some of the options available to help manage yellow nutsedge. The long-term control of this weed depends on killing the parent tuber and preventing daughter tuber production.

Yellow nutsedge has four varieties described as: var. *esculentus* found in Africa, India, southern Europe, and northern North America; var. *leptostachyus* found in North America, South America, and Europe; var. *macrostachyus* found in Central America, southern United States, and the Netherlands; and var. *heermannii* found in southeastern United States and the Netherlands (Schippers et al. 1995). Varietal differences were based primarily upon the size and shape of the floral structures (Schippers et al. 1995). In a morphological description of Illinois *Cyperacea,* two perennial varieties of *Cyperus esculentus* were prevalent (Mohlenbrock 1960). *Cyperus esculentus* var. *esculentus* was described as a typical weed with a wide distribution throughout the state while var. *leptostachyus* was less prevalent and had longer spikelets and achenes than *C. esculentus*

(Mohlenbrock 1960). Additionally, var. *leptostachyus* had more leaves per shoot, wider leaves, shorter leaf length, more rhizomes, lighter tubers, and a greater number of flowering plants compared to var. *esculentus* (Costa and Appleby 1976). There were also differences in the susceptibility of the yellow nutsedge varieties to various herbicide treatments; therefore, it was important to identify the variety that was researched (Costa and Appleby 1976). A taxonomic key (Table 1) was described to determine differences between varieties based on floral characteristics of *Cyperus esculentus* grown over different growing conditions (Schippers et al. 1995).

MORPHOLOGY, GROWTH, AND DEVELOPMENT

Leaves and Shoots. Yellow nutsedge may produce over 139 shoots m^{-2} (Keeley et al. 1983), grow up to 40 cm tall, and produce a leaf area over 2500 cm^2 (Holt and Orcutt 1991). The leaves of yellow nutsedge are green with a mix of yellow and have parallel venation characteristic of a Kranz anatomy found in C_4 or monocot plants (Cañal et al. 1990, Wills et al. 1980). Axillary buds originate at the prophyllar which is the first structure that emerges from the basal bulb (Jansen 1971). Leaves grow from the basal bulb and the intercalary meristem located at the base of each leaf (Stoller and Woolley 1983, Wills et al. 1980). Individual leaves grow for 24 to 40 d at a sigmoid growth rate of 2.2 to 4.5 cm d^{-1} (Jansen 1971). New leaves emerged every 4 to 5 d (Jansen 1971), and leaves develop and green under low light conditions (3 $\mu E\ m^{-2} \cdot s^{-1}$) (Stoller and Woolley 1983). A thick waxy cuticle (1 to 1.3 μm) covers a layer of epidermal cells with large vacuoles on the adaxial leaf surface (Wills et al. 1980). The cuticle on the lower leaf surface is half the thickness of the adaxial surface (Wills et al. 1980). On a cellular level,

the xylem is adaxial to the phloem in the vascular bundles (Wills et al. 1980). The photosynthetic tissue was concentrated in two cell layers that surround the vascular bundles in leaves (Wills et al. 1980). Stomates are concentrated at approximately 400 mm^{-2} on the abaxial leaf surface in parallel rows (Wills et al. 1980).

Basal Bulbs. Basal bulbs are similar to a tuber, but the leaf primordia in the basal bulb produce leaves instead of dormant leaf primordia that surround the tuber (Gifford and Bayer 1995, Wills et al. 1980). Basal bulbs are the origination of the primary vegetative growth structure (Jansen 1971). Basal bulbs develop from a leaf associated with the rhizome and are the result of stem swelling from the rhizomes that originate from a tuber or adventitious rhizomes (Bendixen 1970b, Gifford and Bayer 1995, Stoller and Woolley 1983, Wills et al. 1980). The meristematic activity in the basal bulb produces above ground leaf material, and roots and rhizomes which may develop into secondary shoots or tubers (Bendixen 1970b, Gifford and Bayer 1995, Stoller and Woolley 1983). Basal bulbs may produce approximately 15 rhizomes that develop into secondary and tertiary plants or tubers (Jansen 1971).

Basal bulb development has been related to light exposure, growth medium, and temperature. Basal bulb development was stimulated by exposure to light for 10 to 15 min (Stoller et al. 1972). In the absence of a growth medium (moist cotton), basal bulb development may be hindered (Stoller and Woolley 1983). When no medium was present, light exposure had no effect, but a temperature treatment greater than 10 C stimulated basal bulb development (Stoller and Woolley 1983). However, both light and temperature stimulated basal bulb development in a medium (expanded mica) (Stoller and Woolley

1983). Once temperatures were reduced below 10 C, light was the primary stimulus for basal bulb development (Stoller and Woolley 1983). Light quality did not affect basal bulb development since red and far-red light had similar effects on basal bulb initiation as white light (Stoller and Woolley 1983). The most favorable conditions for basal bulb development were achieved at high temperatures (20/30 C and 25/35 C) (Stoller and Woolley 1983).

Rhizomes. Yellow nutsedge contributes up to 74% of its dry weight to root and rhizome material (Holt and Orcutt 1991). Rhizomes are underground stems that are important for the spread and vegetative propagation of yellow nutsedge. The rhizome length between the basal bulb and tuber may be long or short depending on the stimulus discussed above. Rhizomes develop from axillary buds in the basal bulb (Gifford and Bayer 1995) which produce secondary shoots, additional rhizomes, and tubers (Bendixen 1970b).

Rhizomes usually do not grow longer than 40 cm long (Tumbleson and Kommedahl 1961); however, Jansen (1971) observed rhizomes that were up to 60 cm in length and had over 33 internodes before developing into a basal bud or tuber. Extensive rhizome growth was observed when plants were grown under long photoperiods (Jansen 1971). Rhizomes also grow from the tuber during sprouting (Stoller et al. 1972) and are covered by scale leaves that protect the apical meristem (Wills et al. 1980). These small (0.5 to 1.0 cm long), scale-like leaves called cladophylls or dormant leaf primordia are produced along the length of the rhizome (Jansen 1971, Wills et al. 1980). Rhizomes may develop into basal bulbs or tubers (Bendixen 1970b, Wills et al. 1980). At a 16 h photoperiod, Jansen (1971) observed that rhizomes had their greatest development into

basal buds, but when the photoperiod was less than 14 h, no new shoots were produced (Jansen 1971).

Rhizomes are produced from July to October in Illinois (Jordan-Molero and Stoller 1978). The rhizome consists of an endo and epidermal layer of cells that are separated by cortical cells (Wills et al. 1980). The vascular bundles in the rhizome have an endodermal layer that develops around them, and as the rhizome matures the vascular system is encompassed with lignified cells (Gifford and Bayer 1995, Wills et al. 1980). The lignification of the cells in the rhizome results in rhizomes that appear "wiry". Usually 5 to 6 vascular bundles are found in a rhizome cross-section with the xylem toward the center and phloem toward the outside (Gifford and Bayer 1995). Veins in the rhizomes are directly connected with the basal bulb, tubers, roots, and leaves (Bendixen 1973, Wills et al. 1980).

Inflorescence and Flowers. Yellow nutsedge inflorescence are present as early as July and flowering continues to late August (Hill et al. 1963, Jordan-Molero and Stoller 1978). Yellow nutsedge may produce up to 600,000 inflorescence ha^{-1} (Hill et al. 1963). The inflorescence grow from meristematic tissue located at the base of the involucral leaves (Jansen 1971). The flowering characteristics and seed biology of yellow nutsedge were reviewed (Doll 1983, Stoller and Sweet 1987). The inflorescence size and shape helps taxonomists differentiate between different varieties (Table 1) of yellow nutsedge (Schippers et al. 1995). Yellow nutsedge produces a compound umbel inflorescence on a stem or rachis that is triangular in shape. Spikelets are usually 1 to 3 cm long (Salzman et al. 1997). Yellow nutsedge requires a 12 to 14 h photoperiod once the plants reach six

weeks old (Jansen 1971) to induce flowering. The inflorescence may produce from 1227 to 6685 flowers per inflorescence, and 17% of the flowers produced seed that was viable approximately two weeks after anthesis (Thullen and Keeley 1979). However, some populations produced infertile inflorescence (Holt 1994).

Seed. Seed production is essential for the survival of several plant species; however, seed production is not considered a major factor in the spread of yellow nutsedge. Several researchers have reported viable seed production by yellow nutsedge (Hill et al. 1963, Justice and Whitehead 1946, Thullen and Keeley 1979). Yellow nutsedge produced over 90,000 seed plant^{-1}, and over half sprouted in a study by Hill et al. (1963). Seed germination may range from 1 to 78% and is best when a temperature greater than 21 C was achieved (Thullen and Keeley 1979). Seed was germinated by Hill et al. (1963) at 20/35 C temperature and with a 16/8 h photoperiod cycle. Yellow nutsedge seed weighed from 0.15 to 0.21 mg (Hill et al. 1963) or 0.13 to 0.31 mg (Thullen and Keeley 1979). As the seed weight increased, the percent germination also increased (Thullen and Keeley 1979).

Tubers. Yellow nutsedge tubers are enlargements produced at the terminal tip of a rhizome and are characterized by a cessation of rhizome growth and subsequent swelling of the rhizome (Bendixen 1970b, Bendixen 1973, Gifford and Bayer 1995, Tumbleson and Kommedahl 1961, Wills et al. 1980). Tubers consist of compressed nodes that are densely arranged at the terminal end of the rhizome with a lignified epidermis and leaves that are tightly packed against the tuber surface (Bendixen 1973). Scale leaves encompass the enlargement of a rhizome as the tuber develops from an immature white tuber to a mature

brown, tan, or black tuber (Gifford and Bayer 1995). The scale leaves encompass the tuber once it is mature for protection (Jansen 1971). Mature tubers have a lignified inner cortex and endodermis (Wills et al. 1980). During sprouting, roots are produced from the tubers (Wills et al. 1980) and act as part of the vascular system (Gifford and Bayer 1995). A primary thickening meristem that undergoes periclinal cell division is responsible for the enlargement of the tuber and establishing roots (Gifford and Bayer 1995).

Several studies have evaluated the chemical composition of yellow nutsedge tubers. Tubers have a diverse chemical composition which includes hormones and phenolic compounds like p-coumaric, ferulic, p-hydroxybenzoic, syringic, vanillic, salicylic, protocatechuic, eugenol, and caffeic acids. The total dry weight composition of yellow nutsedge includes 15% oil, 12% starch, 9% carbohydrate, 7.5% protein, 0.06% K, and 0.02% P (Stoller et al. 1972). Most of the stored reserves (60%) are consumed when a tuber sprouts the first time (Stoller et al. 1972). Several factors like light, fertility, and hormones influence the carbohydrate level in yellow nutsedge tubers. For instance, carbohydrate yield in the tubers under a 12 h photoperiod was less than that under a 15.5 h photoperiod (Garg et al. 1967). As temperature, gibberellic acid (GA), or nitrogen levels increased, tuber carbohydrate levels were reduced regardless of the photoperiod.

Tuber composition also depends on the specific ecotype. Stoller and Weber (1975) evaluated two yellow nutsedge ecotypes, one from Illinois and one from Georgia, for tuber composition differences. They determined that the tubers were composed of 47% starch, 3% sugar, 7% protein, and 5% fatty acids (Stoller and Weber 1975). Across six states, carbohydrate levels in yellow nutsedge varied from 45 to 75% of the total dry

weight (Matthiesen and Stoller 1978). Starch was the prevalent carbohydrate and triglycerides composed approximately 80% of the lipids (Matthiesen and Stoller 1978). The tuber lipid content was 5 to 7% on a dry weight basis (Stoller and Weber 1975). Fructose and sucrose were the sugars detected in tubers in the greatest quantity (Garg et al. 1967, Thullen and Keeley 1978). Bendixen (1973), Wills et al. (1980), and Tumbleson and Kommedahl (1961) reported that tuber parenchyma (Gifford and Bayer 1995) cells were also high in starch content.

Allelopathic compounds are the products of one plant that inhibit the growth of another plant. Tubers contain a sprouting inhibitor that affects nearby tubers and several crop species (Drost and Doll 1980, Jangaard et al. 1971, Muñiz and Tames 1982, Tumbelson and Kommedahl 1962). The extract was water soluble and nonvolatile (Tumbleson and Kommedahl 1962).

The identification of viable tubers is useful for evaluating tuber production when control mechanisms are implemented. Taylorson (1967) classified tubers into four groups: 1) black skinned and flacid which were usually dead; 2) black skinned and turgid which were usually older tubers; 3) brown and turgid; 4) white to tan colored. In other research, dead tubers were described as spongy, soft, and hollow (Thullen and Keeley 1975). Viable tubers were hard and fleshy with a white interior (Stoller and Wax 1973, Stoller et al. 1979). Young tubers are small and white colored with parenchyma cells that serve as an epidermal layer (Thullen and Keeley 1978, Wills et al. 1980) while mature tubers are large and brown to black colored (Gifford and Bayer 1995, Jansen 1971, Jordan-Molero and Stoller 1978). Other research has classified tubers based on size (Ghafar and Watson

1983a and 1983b, Keeley and Thullen 1970). Small tubers (157 mg) have a shorter life span compared to large (662 mg) tubers which is important for evaluating the longevity of tubers in the soil (Thullen and Keeley 1975); however, no differences in sprouting between tubers with different sizes have been reported (Thullen and Keeley 1975, Stoller et al. 1972).

Keeley et al. (1983) reported initial tuber counts of 449 to 553 0.1 m^{-3}, but maximum tuber densities were reported by Lapham (1985) which totaled 15,344 m^{-2} with 10,411 viable tubers m^{-2} in field research. Tumbleson and Kommedahl (1961) reported that over 99% of the tubers produced by yellow nutsedge were located in the top 25 cm of soil. Yellow nutsedge tubers may constitute up to 0.5% (w/w) of the top 15 cm of soil in the field (Drost and Doll 1980). Up to 10% of the total dry weight produced by yellow nutsedge was contributed to tubers (Holt and Orcutt 1991). Stoller and Sweet (1987), Bhowmik (1997), and Doll (1983) reviewed tuber formation, sprouting, longevity, dormancy, and the variability between biotypes.

Tubers are classified as the noxious perennial structure that allow the persistence of this troublesome weed, but in areas of Egypt, Spain, Burkina Faso, and the United States a cultivated variety of yellow nutsedge called chufa (*Cyperus esculentus* L. cv. Chufa) was raised (DeVries 1991). In the United States, chufa was described as a food source for turkeys (Daniel's Sons 1997). The primary morphological differences between the weed and chufa were tuber color, rhizome abundance, and the failure to flower (DeVries 1991). DeVries (1991) reviewed the characteristic differences between the yellow nutsedge weed and crop. Chufa is also called tiger nut and earth almond in Egypt

or paternoster in Spanish countries (DeVries 1991). The edible tubers are Mediterranean in origin with a hazelnut taste, and have a higher oil content than the weedy yellow nutsedge (DeVries 1991). The chufa tuber is grey orange, but the weed tuber is grey brown (DeVries 1991). Chufa tubers do not have the cold tolerance found in the weed and are more palatable due to reduced fiber coating the tuber surface (DeVries 1991).

For the weedy species, one tuber planted in the spring may produce greater than 1900 shoots and up to 6900 tubers in one year (Tumbleson and Kommedahl 1961). In other research, one plant produced over 100 tubers (Salzman et al. 1997) and with no crop one plant produced over 170 tubers (Lotz et al. 1991). Keeley and Thullen (1983) reported that yellow nutsedge tuber number (y) was directly related to the number of shoots (x) with the equation $y = 15.68x - 57.1$.

Tubers are usually less than 10 mm long (Bendixen 1973), but may range from 3 to 11 mm in length (Tumbleson and Kommedahl 1961). Larger tubers have a longer life span and produced uniform sprouts with a greater weight than smaller tubers (Thullen and Keeley 1975). Variability of tuber size has been reported by several researchers. For instance, tuber weight ranged from 157 to 662 mg tuber^{-1} dry weight (Thullen and Keeley 1975), 400 to 600 mg tuber^{-1} fresh weight (Wilen et al. 1996a, 1996b), 50 to 120 mg tuber^{-1} dry weight (Stoller and Wax 1973), 500 mg tuber^{-1} (Keeley and Thullen 1974), and 160 to 285 mg tuber^{-1} fresh weight with 40 to 60% moisture (Tumbleson and Kommedahl 1961). Tuber size has been related to the ecotype of nutsedge. For instance, populations in California differed in tuber weight from 6 to 25 mg tuber^{-1} (Holt 1994), and tubers from six states in the Midwest varied from 70 to 710 mg tuber^{-1} (Matthiesen and Stoller 1978).

Tuber fresh and dry weights for Illinois tubers were 255 and 135 mg tuber^{-1}, respectively, but a Georgian ecotype fresh and dry weights were 862 and 538 mg tuber^{-1}, respectively (Stoller and Weber 1975). Finally, Costa and Appleby (1976) reported that *C. esculentus* var. *esculentus* produced tubers with a fresh weight of 490 mg from the field and 330 mg from the greenhouse, while the variety *C. esculentus* var. *leptostachyus* yielded tubers with a fresh weight of 290 mg from the field an 90 mg from the greenhouse. Such differences in tuber size might affect early growth, the reproductive potential, and the control of this weed depending on the climate or latitude.

Several environmental factors affect yellow nutsedge tuber production and sprout. These factors were summarized in Table 2. In the field, temperature, relative humidity, soil depth, and light are the major environmental factors that affect tuber sprout. The production of tubers is related to day length. Short days, less than a 12 h photoperiod, stimulated tuber production. Tubers were observed as early as 7 d after exposure to short days, but do not reach maturity until 28 d after the short d treatment (Bendixen 1973). Temperature affects tuber survivability and sprouting. If tubers are subjected to freezing temperatures, winter kill may result. Warm (30 C) and moist conditions are most conducive for tuber sprout. Most tubers need to be stored in moist conditions because dessication reduces tuber sprout. Finally, the effect of hormones on tuber production and sprouting is dependent on the hormone. Several studies indicate that hormones have a large impact on yellow nutsedge tubers.

The survival of this perennial can result from the ability to produce more than one sprout per tuber even after mechanical removal of a shoot from the tuber (Stoller et al.

1972, Wills et al. 1980, Thullen and Keeley 1975). Stoller et al. (1972) produced plants from tubers that were sprouted up to three times; however, tuber sprout dropped from 80 to 52 to 27% for tubers that were sprouted one, two, and three times, respectively. Buds are concentrated at the terminal end of the tuber and range from 1 to 7 per tuber (Bendixen 1973, Thullen and Keeley 1975, Tumbleson and Kommedahl 1961), but usually number 4 to 5 (Thullen and Keeley 1975). The oldest and largest buds of a tuber usually sprout from the terminal end of the tuber cone first (Bendixen 1973). Tubers have survived up to 64 weeks even if the sprout was removed from the tuber (Thullen and Keeley 1975).

Tuber production may vary depending on the time of the year or location. In Georgia, tuber production was greatest in May and June and fell off in the summer months (Taylorson 1967), but in Minnesota tubers were produced from August to September (Tumbleson and Kommedahl 1961). In Illinois, tuber production began in August and yellow nutsedge needed to be controlled by August 1 to avoid tuber production (Jordan-Molero and Stoller 1978).

Yellow nutsedge varieties and their geographic origin were related according to Schippers et al. (1995). Horak and Holt (1986) determined that yellow nutsedge had limited genetic diversity which was characteristic of plants that have insignificant sexual reproduction. This is not surprising since underground tubers are the primary means of reproduction and propagation. Some genetic variation may occur among populations, but less variation occurs within populations (Horak et al. 1987). Holt (1994) evaluated the variability of yellow nutsedge phenotypic traits and reported high variability between

populations in California rather than within the populations. Quantitative differences such as emergence, flowering, height, dry weight production, rachis number and length, and spikelet characteristics were greater than isozyme differences, and isozyme differences provided a limited resource for identifying variability (Holt 1994). Thus, the possibilities for herbicide resistance by yellow nutsedge may be low due to the limited number of genotypes and the lack of viable seed. If a biotype was resistant to a herbicide, then the resistance should be widespread for that biotype due to the limited variability within populations.

Some field research has evaluated yellow nutsedge tuber production. Researchers have reported that tuber samples were taken from a 15 cm diameter by 30 cm deep (Keeley and Thullen 1983), two 10 cm deep by 1820 cm^2 samples (Keeley and Thullen 1975), and Neeser et al. (1997) sampled 13 cm diameter by 30 cm deep soil cores for purple nutsedge (*Cyperus rotundus* L.). Banks (1983) evaluated tuber production from herbicide treatments and samples were removed from the top 15 cm of soil. Gutman and Watson (1980) described plans for a soil sampler and washing device for tuber separation. The soil sampler was 15 cm^3 and tubers were separated with a 25 mm seed sieve (Ghafar and Watson 1983a, Gutman and Watson 1980). Since the tubers are concentrated in the top 20 cm of soil, samples that are taken to this depth would provide a good estimate of the tubers present in the soil.

Several management decisions in crop production may affect tuber production. When yellow nutsedge control is implemented, tuber production usually depends on the competitive nature of the crop. For example, an integrated approach to control yellow

nutsedge with a combination of cropping system, mechanical, and chemical control for at least two years reduced tuber production 97 to 99% in cotton (*Gossypium hirsutum* L.) (Keeley et al. 1983); however, tuber production may rapidly increase from 3 to 12 fold if not controlled in cotton (Keeley and Thullen 1975, 1983). In addition, Hauser et al. (1974) utilized cotton, corn and peanut rotations to reduce yellow nutsedge tuber production. In corn, herbicide treatments reduced tuber density more than the untreated control within one year after treatment (Stoller et al. 1979). By two and three years after similar treatments were applied, tuber density in the plots was reduced to 20 and 15% of the original density, respectively (Stoller et al. 1979). However, eradication was not achieved after 3 years of intensive management in corn (Stoller et al. 1979).

In other research, several herbicide treatments reduced tuber yield after two years in cotton or soybean (Banks 1983). Stoller et al. (1975) reported that bentazon killed the parent tubers of treated plants and no regrowth was observed with this treatment. Cultivation reduced purple nutsedge tuber density in the presence and absence of herbicides (Webster and Coble 1997). Cultivation caused tubers to be exposed and susceptible to dessication (Keeley et al. 1983). Soil fertility not only increases crop yield but may affect weed growth. At high nitrogen levels, tuber production was less, but shoot production increased especially under long photoperiods (Garg et al. 1967). Fertility levels may affect tuber yield since nitrogen applications to the *C. esculentus* var. *leptostachyus* increased tuber dry weight (Appleby and Paller 1978). Adjusting fertility and fertility placement may reduce the growth and depletion of nutrients by this weed.

Table 3 summarizes sprouting methods and environmental conditions that have been documented or employed for yellow nutsedge sprouting in controlled environments. Tuber collection for these experiments depended upon the tuber dormancy characteristics, resistance to dessication, and dormancy considerations. Keeley and Thullen (1974) and Banks (1983) collected tubers in March for use in greenhouse experiments to evaluate sprouting. For other evaluations, Jansen (1971) reported that leaf length, fresh weight of parent and peripheral shoots, shoot number, rhizome fresh weight, number of rhizome tip types, mature height, and number of flowering plants were suitable and repeatable measurements for yellow nutsedge research.

Breaking Dormancy. Tubers are dormant from summer to fall as low levels of carbohydrates were available for sprouting (Taylorson 1967). Thomas (1969) broke tuber dormancy by storing tubers at 4 C for 21 d while Stoller and Wax (1973) exposed the tubers to 2 C to break dormancy. Appleby and Paller (1978) stored tubers in plastic bags to maintain a humid environment at 5 C for 3 weeks to break dormancy. Research has been contradictory with respect to whether washing tubers affects sprouting (Taylorson 1967, Tumbleson and Kommedahl 1962). However, washed tubers in Minnesota research had 70 to 81% greater sprout than unwashed tubers (Tumbleson and Kommedahl 1961). Table 3 summarizes some of the sprouting methods and environmental conditions needed to effectively store and sprout yellow nutsedge tubers. Tubers sprouted at 25 to 30 C with or without a diurnal cycle has been utilized as an effective sprouting method. A petri dish with two filter papers surrounding the tubers and wrapped with parafilm has been used to make sure that the tuber with plenty of moisture available. A combination of washing and

cold storage in humid environments helps break tuber dormancy and ensure viable tubers for research purposes. Preventing the exposure of tubers to light or diurnal cycles do not appear to have a large influence on tuber sprout although several researchers have included this procedure in their experiments. These conditions will insure a dependable and repeatable environment that will encourage tuber sprout.

Internal tuber chemical composition appears to affect and regulate tuber sprouting. Hormone treatments may increase or prevent tuber sprouting. A treatment of 100 ppm N-6 benzyl adenine stimulated purple nutsedge sprouting (Zandstra and Nishimoto 1977). Also, GA promoted tuber sprouting within one week after planting, but had no effect 2 or 5 weeks after the tubers were planted (Tumbleson and Kommedahl 1962). Jangaard et al. (1971) inhibited tuber sprouting with high levels of phenols (eugenol and salicylic acid); however, limited salicylic acid was found in the tubers. Phenolic levels in the tubers were low in the spring and fall (Muñiz and Tames 1982). Jangaard et al. (1971) and Muñiz and Tames (1982) concluded that phenolic compounds had almost no effect upon tuber dormancy. Abscisic acid inhibited and delayed tuber sprouting depending on the rate applied to the tubers (Jangaard et al. 1971). Abscisic acid (ABA) levels were highest in November (6.81 ug g^{-1} fresh weight), but during sprouting (April) no ABA was detected (Muñiz and Tames 1982). Due to ABA fluctuations and its effect on sprouting, ABA could be a tuber dormancy regulating compound. Finally, 2,4-D reduced shoot growth 12 d after treatment (DAT) and was translocated to actively growing tissue, but was not metabolized (Bhan et al. 1970). Table 2 summarizes some of the effects of hormones on yellow nutsedge.

Sprouting and Longevity. Yellow nutsedge sprouts from the distal or terminal portion of the tuber (Tumbleson and Kommedahl 1962). When yellow nutsedge tubers sprout, one or two buds appeared as white protrusions on the tuber. Yellow nutsedge was considered sprouted when a bud protruded the scales of the tuber (Holt and Orcutt 1996). The subsequent sprout of other buds was observed when the growing bud was removed (Bendixen 1973). Taylorson (1967) reported that tuber sprouting was greatest from tubers harvested from February to March. However, yellow nutsedge usually emerged from April to July in Illinois fields (Jordan-Molero and Stoller 1978, Stoller and Wax 1973). Tubers may remain viable in the soil for at least two years (Stoller and Wax 1973). Tuber sprout was not affected by the tuber weight, but increased shoot biomass was produced from heavier tubers (Stoller et al. 1972).

Tuber ecotype differences were noted by Stoller and Weber (1975) with respect to winter survivability. An Illinois ecotype had excellent sprouting (100%) after a cold treatment, over winter (87%), or fresh after harvest in October (91 to 94%); however, the Georgia ecotype had only 8% sprout after overwintering and 67 to 78% after cold treatment or fresh out of the field (Stoller and Weber 1975). Stoller and Weber (1975) reported that starch, sugar, and lipid levels increased in the tuber during cold treatment; however, they concluded that differences in several tuber constituents may be related to tuber cold tolerance. The adaption of an ecotype and environmental factors may significantly affect yellow nutsedge tuber survival. Table 2 summarizes some of the environmental effects on tubers which may directly or indirectly affect tuber production or sprouting. Mechanical soil disturbance may increase tuber sprouting in the field

(Taylorson 1967); however, tubers are susceptible to desiccation and low temperatures (Thomas 1969).

The temperature threshold for tuber sprouting was 6 C for yellow nutsedge while for purple nutsedge it was 11.2 C (Holt and Orcutt 1996). This would imply that yellow nutsedge has an excellent chance for early sprouting and establishment, and would help explain why purple nutsedge was not found in northern latitudes. Yellow nutsedge also had the broadest sprouting temperature range compared to purple nutsedge and johnsongrass (Holt and Orcutt 1996). The LD_{50} for temperature was -7 C, but tubers could easily survive temperatures of -4 C (Stoller and Wax 1973). Thomas (1969) reported that tubers had the greatest survivability at 90% relative humidity and room temperature. Under alternating temperature regimes tubers readily sprouted and stimulated quick basal bulb development in contrast to constant temperatures (Stoller and Woolley 1983), and as storage time increased, the number of sprouts that emerged from a tuber increased (Thullen and Keeley 1975).

The depth of tubers in the soil has a large impact upon sprouting. Tubers at 2.5 cm depth had 1% sprouting the following year while sprouting from 10 to 20 cm deep was 55 and 84%, respectively (Stoller and Wax 1973). No shoots emerged from tubers planted 30 cm deep in Minnesota (Tumbleson and Kommedahl 1961). Tuber sprout ranged from 49 to 57% 3 to 9 weeks after planting (Dixon and Stoller 1982), 80 to 100% for tubers in Illinois (Stoller et al. 1972, Stoller et al. 1979, Stoller and Wax 1973) and 100% for tubers in California (Holt and Orcutt 1996, Keeley et al. 1979). Black skinned

turgid tubers usually had 15% or less sprouting compared with brown tubers (Taylorson 1967).

Recently, researchers have tried to predict yellow nutsedge emergence using environmental parameters in models to help predict cultivations (Wilen et al. 1996a). However, emergence patterns for California and Arizona genotypes were different and under dry conditions these models had difficulty predicting emergence (Wilen et al. 1996a, 1996b). For the California genotype, emergence was reported 47 to 61 d after planting in dry soil (-0.06 MPa) and 33 to 49 d after planting in wet soil (-0.03 MPa); however, emergence for the Arizona genotype emerged 51 to 76 d after planting in dry soil (-0.06 MPa) and 43 to 61 d after planting in wet soil (-0.03 MPa) (Wilen et al. 1996a). Both genotypes emerged in California earlier than in Arizona even under similar growing conditions (Wilen et al. 1996a). Predictions of yellow nutsedge emergence could be made with a 2 d accuracy under adequate rainfall conditions (Wilen et al. 1996b). These methods may help producers make economical decisions for cultivation timings to control this weed.

COMPETITION WITH CROPS

Yellow nutsedge is competitive with several crop species early in the growing season (Doll 1983, Ghafar and Watson 1983a, Keeley 1987). Yellow nutsedge densities from 40 to 1000 plants m^{-2} have reduced agronomic and horticultural crop yields (Keeley 1987). Based on the leaf area index (LAI), yellow nutsedge was not considered a big competitor for light late in the season (Ghafar and Watson 1983a). However, yellow nutsedge does compete for moisture, nutrients and light, and may exert allelopathic effects

on crop species early in the season (Keeley 1987). Holt and Orcutt (1991) reported that yellow nutsedge was more competitive than johnsongrass, purple nutsedge, and cotton when the above-ground yield and aggressiveness was measured and compared. Nonetheless, tuber production may be reduced up to 96% depending on the cropping system and the adopted herbicide management program (Keeley et al. 1979). Although yellow nutsedge plants rarely grow taller that 20 to 60 cm (Salzman et al. 1997), a dense stand may intercept light and deplete soil moisture and nutrients before the crop is able to grow above this aggressive perennial.

Crop selection has an important effect on the ability of yellow nutsedge to compete. Canopy development differences between crops caused bush bean to be more competitive than corn, followed by bell pepper (*Capiscum annum* L.) with purple nutsedge for incident PAR (Neeser et al. 1997). Competition for water and nutrients is usually greater than for light due to the low growth habit of nutsedge. However, crops may require 8 to 9 weeks before they reach 90% light interception (LI) (Keeley and Thullen 1978). Shading yellow nutsedge effectively reduced shoot number, height, and tuber and shoot biomass in field and greenhouse research (Keeley and Thullen 1978, Santos et al. 1997). Flowering was also reduced when yellow nutsedge was in 30 to 47% shade (Keeley and Thullen 1978). A competitive crop like hemp (*Cannabis sativa* L.) reduced tuber and shoot production by yellow nutsedge from 99 to 100% (Lotz et al. 1991). However, tuber production with barley or rye was only reduced 40% (Lotz et al. 1991). Fallow treatments reduced tuber sprout 44% and tuber yield 99% (Tumbleson and Kommedahl 1961) and glyphosate treatments on fallow areas reduced tubers by 98%

(Keeley et al. 1979). In one year, viable tubers m^{-2} were reduced by about 75% with one year of control (Lapham 1985).

Keeley and Thullen (1978) reported that there was a strong correlation between yellow nutsedge growth, tuber production, and shade levels. The amount of light available to yellow nutsedge decreased as corn population increased (Ghafar and Watson 1983a). Yellow nutsedge compensates for low light by increasing shoot growth and reducing root and tuber production (Santos et al. 1997). Lotz et al. (1991) reported that adequate light was important for tuber production. Yellow nutsedge may survive under reduced light intensities because of its low light compensation point (where the CO_2 fixed equals CO_2 respired) (Santos et al. 1997). Tuber production was reduced under 30 and 70% shade by approximately 20 and 75%, respectively, when grown under shade cloth (Jordan-Molero and Stoller 1978). However, yellow nutsedge aboveground growth at 30% shade was similar to plants grown in full sunlight (Jordan-Molero and Stoller 1978). Similarly, reducing the light intensity by 43 and 18% reduced tuber production 51 and 3% as well as tuber weight 56 and 30%, respectively, in the greenhouse 80 d after planting (Lotz et al. 1991). Tuber production was reduced by 95% under 80% shade, and with no shade each plant averaged 62 tubers (Santos et al. 1997). Under 0% shade, yellow nutsedge produced from 400 to 1100 tubers, but under 73% shading only 100 to 300 tubers were produced (Jordan-Molero and Stoller 1978). Keeley and Thullen (1978) reported that 94% continuous shade reduced tuber production by 94%, but under 80 to 94% continuous shade there was still a net increase in tubers. Tuber dry weight (y) was reduced linearly in controlled environments by shading treatments (x): $y = 12.46 - 0.16x$.

Several cultural methods like crop planting date, density, selection, rotations, cultivation, fertility, and weed-free periods may be adopted to manage yellow nutsedge (Ghafar and Watson 1983a and 1983b, Glaze 1987, Jordan-Molero and Stoller 1978, Keeley 1987). Crop selection and crop density are key factors that help suppress yellow nutsedge (Neeser et al. 1997, Keeley 1987, Keeley and Thullen 1978, Keeley et al. 1979). For instance, alfalfa reached 90% LI in 2 to 3 weeks, but onion required 26 weeks before light interception reached 95% (Keeley and Thullen 1978). Based on light interception data, alfalfa, barley, corn, potatoes, and safflower were the best competitors with yellow nutsedge (Keeley and Thullen 1978). At high corn populations (133,300 plants ha^{-1}), the number of tubers and tuber size was reduced compared to lower corn populations (33,300 plants ha^{-1}) (Ghafar and Watson 1983a). Tuber production was reduced 97 to 99% when a combination of cropping systems, mechanical and chemical control methods were used in cotton (Keeley et al. 1983). This was also evident when herbicides were applied in a cotton, corn, and peanut rotation in Georgia which killed 78 to 99% of the tubers depending upon the herbicide use intensity (Hauser et al. 1974). Several herbicide treatments in cotton and soybean reduced tuber yield after two years of use (Banks 1983). Intense cultivation alone reduced tuber production from 97 to 99% (Hauser et al. 1974). Cultivation helps remove early flushes of yellow nutsedge in cotton and corn, but does not always provide excellent yellow (Keeley and Thullen 1978) or purple nutsedge control (Webster and Coble 1997). By delaying planting time, yellow nutsedge growth, dry matter production, tuber number and weight were reduced (Jordan-Molero and Stoller 1978). Late planted corn had smaller tubers produced in the corn row while large ones

were produced between the corn rows (Ghafar and Watson 1983b). The planting date with the highest corn yield and the lowest yellow nutsedge growth in Canada was the third week of May (Ghafar and Watson 1983b).

Holt and Orcutt (1991) measured yellow nutsedge competition as the relative "agressiveness" (A) in terms of dependent variables [A=1.86 - 1.5(relative growth rate) + 200.96(unit leaf rate) + 0.71(ln height) - 0.28(initial propagule weight)]. Yellow nutsedge was more aggressive than cotton (Holt and Orcutt 1991). The unit leaf rate (mg $cm^{-2} \cdot d^{-1}$) was the best variable that described the relative competitive difference between yellow nutsedge, cotton, johnsongrass, and purple nutsedge (Holt and Orcutt 1991).

Allelopathic effects of yellow nutsedge residue reduced corn and soybean growth in greenhouse research, but the effect did not interact with soil texture (Doll 1983, Drost and Doll 1980). The placement of yellow nutsedge residue beside the seed reduced corn and soybean growth more than the absence of the residue (Drost and Doll 1980). Soybean were more susceptible than corn to increased rates (0 to 0.675%) of yellow nutsedge residue (Drost and Doll 1980). Similarly, Tumbelson and Kommedahl (1962) reported that yellow nutsedge tuber extract inhibited or delayed red clover, barley, oats, pea, alfalfa, and soybean germination and inhibited root development of some species. However, Tumbelson and Kommedahl (1962) reported that tuber extracts had no effect on the germination of wheat or corn unless high rates were present with the corn seeds. Tumbelson and Kommedahl (1962) developed an alfalfa germination bioassay that could detect the tuber extract.

Corn. Yellow nutsedge densities in corn have been recorded up to 2580 to 3875 plants m^{-2} (Tumbleson and Kommedahl 1961), and untreated yellow nutsedge in corn produced over 1200 tubers m^{-2} (Stoller et al. 1979). Yellow nutsedge reduced corn biomass 7% in container-grown plants 6 weeks after planting (Campbell and Hartwig 1982). In the field, yellow nutsedge densities of 300 and 1200 tubers m^{-2} reduced corn yields by 17 and 41%, respectively (Stoller et al. 1979). However, silage corn reduced tuber production from 4 to 15% (Lotz et al. 1991). Corn intercepted 70% of the incident photosynthetically active radiation (PAR) at the ground level by June 12, and at July 18, 95% of the incident PAR was intercepted by corn (Lotz et al. 1991). Tuber residue was evaluated from 0 to 0.075% (w/w) which reduced shoot growth of corn and soybean more than foliage residue (Drost and Doll 1980). However, corn shoot and root growth were reduced by tuber residue by 46 and 45%, respectively (Drost and Doll 1980). Finally, corn yield was reduced 8% for every 100 shoots m^{-2} and tubers were produced under the corn canopy (Stoller et al. 1979).

Herbicides may reduce yield loss from yellow nutsedge in corn depending on the level of yellow nutsedge infestation. For instance, alachlor preplant incorporated (PPI) prevented yield loss from yellow nutsedge (Stoller et al. 1979). Yield of corn treated with no postemergence herbicides equaled yield of corn treated with postemergence bentazon or cultivated, and tuber production in cultivated corn was similar to tuber production in corn treated with a postemergence application of bentazon (Stoller et al. 1979). Corn treated with postemergence and postemergence-directed herbicide applications had from 7 to 8% yield loss when a PPI weed control program was not applied (Stoller et al. 1979).

Moderate herbicide use controlled yellow nutsedge from 70 to 76% in corn which was similar to the 70% control with cultivation alone, while intense herbicide use provided 91% control (Hauser et al. 1974).

Soybean. Soybean had greater susceptibility to tuber residue than corn (Drost and Doll 1980). Soybean shoot and root growth was reduced by tuber residues 42 and 55%, and shoot residues 35 and 61%, respectively (Drost and Doll 1980). However, the leachate reduced shoot growth by only 16% (Drost and Doll 1980). Soybean seed yield was reduced by densities from 200 to 300 shoots m^{-2} when poor yellow nutsedge control was attained with herbicides (Banks 1983). Untreated soybean had 379 viable tubers m^{-2} (Banks 1983). Limited research has been conducted on yellow nutsedge control in soybean.

Cotton. Variable effects of yellow nutsedge on cotton yield have been reported. Holt and Orcutt (1991) reported that yellow nutsedge had superior growth characteristics as measured by several growth parameters compared to cotton. Yellow nutsedge reduced cotton seed yield 41% and stem diameter 20% two of three years, but had no effect one year when it was allowed to compete for 25 weeks (Patterson et al. 1980). Cotton seed and lint yield were reduced by densities from 200 to 300 shoots m^{-2} when poor yellow nutsedge control was reported (Banks 1983). Untreated cotton had 307 viable tubers m^{-2} after two years (Banks 1983). Yellow nutsedge reduced cotton seed yield approximately 18 kg ha^{-1} for each plant m^{-2} (Patterson et al. 1980). Patterson et al. (1980) recommended that yellow nutsedge should be controlled between 10 and 25 weeks after planting, but after 25 weeks it was too late. However, four weeks of competition reduced cotton seed

yields, delayed maturity, and reduced cotton height in research by Keeley and Thullen (1975). Keeley and Thullen (1983) reported that a 2 to 6 week weed free period reduced tuber production and had no effect on cotton yield. Cotton seed yield was reduced 0 to 50% by yellow nutsedge in the untreated control, but had no effect on the quality or quantity of lint produced (Keeley and Thullen 1983). However, a more drastic effect was reported where cotton seed yield was reduced 34% in irrigated soil by yellow nutsedge, and quality was reduced during one year (Keeley and Thullen 1975).

A combination of cropping system, and mechanical and chemical control for at least two years reduced tuber production 97 to 99% in cotton (Keeley et al. 1983). Similarly, in a corn-cotton rotation, cultivation plus halosulfuron and ametryn reduced purple nutsedge tuber populations (Webster and Coble 1997). Moderate herbicide use in cotton controlled yellow nutsedge 37% and cultivation provided 97% control, while intense herbicide usage provided 92% control (Hauser et al. 1974). Cotton germination threshold was from 11 to 44 C compared to yellow nutsedge which sprouted from 3 to 45 C (Holt and Orcutt 1996).

YELLOW NUTSEDGE RESPONSE TO HERBICIDES AND HORMONES

Herbicides are utilized as a primary yellow nutsedge control mechanism in crop production. In 1968, Hauser et al. pointed out in a review that there was a dire need for the development of herbicides that effectively control yellow nutsedge. In 1987, Pereira et al. reviewed the efficacy of several herbicides used to control yellow nutsedge. Herbicides included in the review were 2,4-D, atrazine, bromacil, linuron, bentazon, amitrol, fluoridone, norflurazon, paraquat, dichlobenil, alachlor, metolachlor, nitrofen, oxyfluorfen,

fluorodifen, EPTC, vernolate, butylate, arsenicals, fumigants, and glyphosate (Pereira et al. 1987). Additives that increased yellow nutsedge control with some of the herbicides included oils and nitrogen sources like ammonium sulfate (Pereira et al. 1987). Much of the research evaluating yellow nutsedge control was conducted before the 1980's. Numerous papers evaluated yellow nutsedge control in agronomic crops prior to the introduction of bentazon. The decline in yellow nutsedge research was strongly related to the introduction of this effective postemergence herbicide used in corn and soybean. The only thing bentazon lacks is residual control; therefore, late sprouting tubers may still produce enough tubers under the low light intensities of the crop canopy to maintain a competitive population under stressful environmental conditions.

Several herbicides suppress the growth and development of yellow nutsedge. Table 4 reviews the effects of several herbicides and control methods on yellow nutsedge tubers. Herbicides have reduced yellow nutsedge shoot density 77 to 97% (Keeley et al. 1979). For instance, EPTC applied in alfalfa reduced nutsedge shoot counts from 5.5 to 6.4 m^{-2}. Butylate applied in corn reduced nutsedge shoot number 80 to 85%, but yellow nutsedge regrew late in the season; and MSMA applied in cotton reduced shoot number 80% (Keeley et al. 1979). Again, yellow nutsedge regrew late in the season (Keeley et al. 1979). Herbicide applications of imazethapyr, metolachlor, or imazethapyr plus metolachlor reduced yield loss in peanuts to yellow nutsedge (Grichar et al. 1992). However, one important goal is to reduce the reproductive potential of this weed which means a reduction in tuber production. A lack of residual herbicide activity is a common theme for producers trying to control yellow nutsedge since tubers may sprout after a

herbicide application or shoots may arise from underground rhizomes. The exposure of tubers to herbicide treated soil can reduce growth and daughter tuber production (Banks 1983). A combination of cropping system management and herbicide selection can effectively reduced tuber production to 5 to 17% of the original population and reduce tuber viability to 3 to 9% (Keeley et al. 1979). The recent introduction of Roundup Ready® crops and the acetolactate synthase-inhibiting (ALS, E.C. 4.1.3.18) herbicides has made more options for yellow nutsedge control in agronomic crops.

Glyphosate. Glyphosate has been utilized as an effective non-selective herbicide for the control of perennial weeds like yellow nutsedge. Table 5 summarizes the effect of glyphosate on yellow nutsedge. Pereira et al. (1987) reported that glyphosate was a superior herbicide for preventing yellow nutsedge regrowth and tuber production. Glyphosate symptoms exhibited by yellow nutsedge included leaf apex, basal bulb, and vein tissue necrosis and chlorosis (Keeley et al. 1979), height reduction, reduced fresh weight, and rhizome, tuber, and secondary shoot growth inhibition (Cañal et al. 1990, Villaneuva et al. 1985, Stoller et al. 1975). Glyphosate also reduced chlorophyll content 6 DAT when it was applied to yellow nutsedge (Pereira and Crabtree 1986). Similarly, Villaneuva et al. (1985) reported that chlorophyll pigments were as susceptible to glyphosate as carotenoid pigments. This may account for the observed symptoms exhibited by yellow nutsedge to glyphosate.

Variable yellow nutsedge control from glyphosate was usually related to the application rate. The yellow nutsedge shoot number following an application of glyphosate to fallow ground remained high through the fallow period, but the shoot number

decreased by 95% by the next year (Keeley et al. 1979). However, yellow nutsedge plants treated with 1100 or 2200 g ha^{-1} of glyphosate regrew from secondary shoots (Pereira and Crabtree 1986) and 0 to 3% of the parent tubers were not killed (Stoller et al. 1975). Appleby and Paller (1978) reported 10 to 12% tuber sprout and up to 42% nonviable tuber production from 1100 and 2200 g ha^{-1} glyphosate. Regrowth was less when plants were treated at 70 compared with 40 d after emergence (Pereira and Crabtree 1986). Young plants (30 d old) absorbed more ^{14}C-glyphosate than 60 d old plants, but translocation to underground plant parts was greater in the 60 d old plants (Pereira and Crabtree 1986) which could account for differential control. In other research, glyphosate translocation to the tubers decreased as the plant aged (Keeley et al. 1985). Appleby and Paller (1978) reduced the number of tubers produced per plant and increased the number of nonviable tubers as the rate of glyphosate increased from 300 to 2200 g ha^{-1}.

Careful selection of herbicide tank mixtures with glyphosate should identify combinations that synergistically control yellow nutsedge since antagonistic combinations with glyphosate have been reported on difficult-to-control weeds like velvetleaf (*Abutilon theophrasti* Medicus) (Lich et al. 1997), ivyleaf morningglory [*Ipomoea hederacea* (L.) Jacq.] (Lich et al. 1997), purple nutsedge (Rao and Reddy 1999), and sicklepod (*Cassia obtusifolia* L.) (Rao and Reddy 1999). Imazaquin plus glyphosate was the most antagonistic tank mixture for purple nutsedge control (Rao and Reddy 1999). Interactions could be due to reduced absorption or translocation of glyphosate in the presence of the ALS-herbicides. The ALS-herbicides may be translocated more readily than glyphosate. However, Starke and Oliver (1998) reported that chlorimuron did not affect the

absorption and translocation of ^{14}C-glyphosate by velvetleaf or pitted morningglory, but glyphosate increased the absorption of ^{14}C-chlorimuron or ^{14}C-imazethapyr by velvetleaf.

An application of naptalam followed by glyphosate synergistically controlled yellow nutsedge due to the stimulation of shoot growth and inhibited tuber formation from the naptalam (Appleby and Paller 1978). Such treatments with hormones might provide greater yellow nutsedge control and should be explored. Pereira and Crabtree (1986) synergistically controlled 30 d old yellow nutsedge when 1500 g ha^{-1} glyphosate was tank mixed with 1500 g ha^{-1} oxyfluorfen due to increased absorption and translocation in young plants. Similarly, 2,4-D and amitrol increased purple nutsedge control with glyphosate (Suwunnamek and Parker 1975). However, photosynthesis inhibitors (diuron, atrazine, and terbacil) antagonized purple nutsedge control with glyphosate (Suwunnamek and Parker 1975). The addition of ammonium sulfate from 1.25 to 10 kg ha^{-1} and other ammonium salts to glyphosate increased purple nutsedge control four fold (Suwunnamek and Parker 1975) while monovalent inorganic salts (NH_4, K, and Na) increased purple nutsedge control compared to untreated, divalent (Zn), and trivalent (Fe) cations (Wills and McWorter 1985). However, an organosilicone surfactant plus glyphosate (formulated as Roundup Ultra®)[22] did not affect control, absorption, or translocation of ^{14}C-glyphosate (Bariuan et al. 1999).

Glyphosate is readily absorbed by and translocated in yellow nutsedge. From one to nine d after yellow nutsedge was spotted with ^{14}C-glyphosate, absorption and translocation increased with time (Pereira and Crabtree 1986). In non-resistant soybean,

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[22]Roundup Ultra, Monsanto Co., 800 N. Lindbergh Blvd., St. Louis, MO 63167.

glyphosate caused severe injury to soybean at 0.3 kg ha^{-1}, while 2.2 kg ha^{-1} was required for yellow nutsedge control (Stoller et al. 1975). Postemergence applications of 600 g ha^{-1} glyphosate to 2 to 4 week old plants reduced parent tuber resprouting 14 to 32% with a visual control of 43 to 45% (Keeley et al. 1985). Similarly, Appleby and Paller (1978) reported control from 300, 600, 1100, and 2200 g ha^{-1} was 27, 52, 97, and 100%, respectively. One could estimated from this research that 600 g ha^{-1} would probably provide 50% yellow nutsedge growth reduction in greenhouse research (Appleby and Paller 1978). When a single twin shoot was treated with glyphosate the growth of the other shoot was reduced (Keeley et al. 1985). Table 6 summarizes research on the absorption and translocation of glyphosate by yellow nutsedge.

At high rates of glyphosate (10mM), vacuolar phenol levels were reduced, vascular bundles developed necrotic areas, and sclerenchyma cells were formed between the cortical parenchyma cells that separate the root and rhizome of yellow nutsedge plants (Cañal et al. 1990). Glyphosate may act as a sprouting inhibitor since 300 to 600 g ha^{-1} increased the number of dormant tubers produced in greenhouse research (Appleby and Paller 1978). Catalase activity in purple nutsedge was reduced 70% by glyphosate (Abu-Irmaileh and Jordan 1978). Glyphosate at 4000 g ha^{-1} killed the tubers of young purple nutsedge (Zandstra and Nishimoto 1977). Young purple nutsedge tubers were a strong sink for ^{14}C-glyphosate, but tubers from older plants were not a strong sink (Zandstra and Nishimoto 1977). Glyphosate reduced respiration in purple nutsedge tubers as denoted by the pink color from the tetrazolium test (Zandstra and Nishimoto 1977), and glyphosate

was not metabolized by purple nutsedge (Zandstra and Nishimoto 1977). One of the major problems with glyphosate is consistently killing the parent tubers.

Control of purple nutsedge with glyphosate has been evaluated by several researchers. Glyphosate at 4000 g ha^{-1} reduced purple nutsedge fresh weight and sprouts per tuber in the greenhouse, and in the field purple nutsedge stands were reduced by 26 and 67% with 2 and 4 kg ha^{-1} glyphosate, respectively (Zandstra et al. 1974). Glyphosate at 2.24 kg ha^{-1} and 4.48 kg ha^{-1} controlled 17 d and 10 week old plants (Bariuan et al. 1999). In the top 13 cm of soil, tuber production was reduced 92% with 2 to 4 kg ha^{-1} glyphosate and 89% with MSMA, but neither dicamba nor paraquat reduced tuber production (Zandstra et al. 1974). Tuber sprouting was reduced by 49 to 52% with glyphosate at 2.24 and 4.48 kg ha^{-1} (Bariuan et al. 1999), and 55% with MSMA (Zandstra et al. 1974). At high humidity (90%) and low water stress (-2 bars), glyphosate at 2000 g ha^{-1} controlled purple nutsedge 73 to 77% due to increased translocation at these environmental conditions (Chase and Appleby 1979). Bariuan et al. (1999) reported that the most glyphosate was translocated in the shoot 168 h after treatment. However, translocation of glyphosate to the underground growth, was less than 1% (Chase and Appleby 1979). The addition of additives to glyphosate may affect yellow nutsedge control. Limited research has evaluated other adjuvants commonly used for weed control with other herbicides. Tank mixture combinations of glyphosate with other herbicides may be related to increased control due to the addition of additional adjuvant.

ALS-inhibitors. The ALS-inhibiting herbicides inhibit acetolactate synthase which is essential for the production of the plant essential amino acids leucine, valine, and

isoleucine (Claus 1987). ALS-inhibiting herbicides are translocated to the actively growing meristematic tissue in plants (Claus 1987). The ALS-inhibiting herbicides include the sulfonylurea, imidazolinone, triazolopyrimidine sulfonanilide, and pyrimidinyl thiobenzoate herbicide families. These herbicides may cause yellow nutsedge necrosis and death, suppression, or no control depending upon the herbicide. The use of these herbicides encompasses a wide spectrum of agronomic and horticultural crops. Several studies have evaluated the sight of uptake for the ALS-inhibiting herbicides. Many of these herbicides have the best yellow nutsedge control when applied postemergence which allows for both foliar and soil uptake. Several of these herbicides provide residual control of yellow nutsedge unlike glyphosate. Table 6 summarizes the absorption and translocation research conducted to date with these herbicides.

Ackley et al. (1996) evaluated yellow nutsedge control with several ALS-inhibiting herbicides and reported that halosulfuron provided 88 to 94% control, chlorimuron 78 to 91%, imazethapyr 28 to 54%, nicosulfuron 50 to 58%, primisulfuron 60 to 73%, rimsulfuron 51 to 89%, and pyrithiobac 41 to 74%. CGA-152005 (proposed prosulfuron) or thifensulfuron did not control yellow nutsedge (Ackley et al. 1996). In greenhouse research, halosulfuron controlled yellow nutsedge greater than chlorimuron, while imazethapyr had increased activity in the greenhouse compared with other herbicides in the field (Ackley et al. 1996). Richburg III et al. (1993a) reported that nicosulfuron provided less than 43% yellow nutsedge control, but nicosulfuron tank-mixed with bentazon provided greater control than either herbicide applied alone. The interaction between glyphosate, chlorimuron, imazamox, imazaquin, and halosulfuron was evaluated

for purple nutsedge control (Rao and Reddy 1999). At 50% the recommended rate, control was greatest with chlorimuron followed by halosulfuron > imazaquin > imazamox > pyrithiobac of three week old yellow nutsedge and control was at least 27% less compared with the full rate (Rao and Reddy 1999).

Postemergence applications of halosulfuron at 30 to 280 g ha^{-1} provided 34 to 89% yellow nutsedge control 63 DAT (Derr et al. 1996). Yellow nutsedge control with halosulfuron was linearly related to the rate applied preemergence (PRE) (Derr et al. 1996). Chlorimuron at 10 g ha^{-1} provided 33% control 63 DAT which was similar to 3360 g ha^{-1} glyphosate (Derr et al. 1996). Control of yellow nutsedge with halosulfuron was greater than control with 1120 g ha^{-1} bentazon (Derr et al. 1996). Similarly, halosulfuron provided greater yellow nutsedge control than bentazon or imazaquin in turfgrass (Czarnota and Bingham 1997). Below tuber placement treatments of halosulfuron reduced yellow nutsedge regrowth by 95% in the greenhouse, while control decreased when halosulfuron was placed at other locations in the soil (Vencill et al. 1995). A postemergence foliar, soil, or foliar plus soil treatment of halosulfuron reduced yellow nutsedge dry weight 54 to 65% 30 DAT, and regrowth dry weight was reduced by more than 96% (Vencill et al. 1995). Halosulfuron reduced purple nutsedge tuber population and shoot density in a corn followed by corn rotation in the presence and absence of cultivation (Webster and Coble 1997). In turfgrass, halosulfuron had no effect on yellow nutsedge tuber density or weight, but reduced tuber viability with two applications (Molin et al. 1999). As the rate of halosulfuron increased, tuber viability was reduced (Molin et al. 1999). Similarly, yellow nutsedge control 6 weeks after treatment (WAT) was 77 to

100% with halosulfuron at 0.14 kg ha^{-1} and limited regrowth (Czarnota and Bingham 1997). No research has evaluated the absorption or translocation of halosulfuron to underground tubers.

Imazethapyr, an imidazolinone herbicide, at 60 and 120 g ha^{-1} reduced yellow nutsedge shoot fresh weight 31 to 83% PRE and 70 to 72% postemergence with a 0 to 90% reduction in regrowth which was dependent on the application rate and timing (Derr and Wilcut 1993). Imazethapyr postemergence at 70 g ha^{-1} had variable control (40 to 66%) 46 to 142 DAT, but control with a PPI application was 55 to 99% (Grichar et al. 1992). Richburg III et al. (1993b) reported that soil and foliar plus soil applied imazethapyr provided the best yellow nutsedge control. Imazethapyr at 70 g ha^{-1} applied above or below the tuber stimulated growth 28 DAT and stimulated regrowth when applied below the tuber 42 DAT (Richburg III et al. 1993b). Above and below tuber treatments with imazethapyr reduced shoot number, dry weight 28 DAT, and regrowth 42 DAT (Richburg III et al. 1993b). A foliar postemergence application of imazethapyr plus NIS provided similar control at between an early postemergence and postemergence timing 28 DAT; however, 42 DAT regrowth was reduced by the postemergence greater than the early postemergence timing, but no difference between timings was reported for below ground root and tuber control (Richburg III et al. 1993b).

Imazaquin at 250 and 500 g ha^{-1} reduced yellow nutsedge fresh weight by 83 to 89% PRE and by 72 to 77% postemergence with an 85 to 95% reduction in regrowth (Derr and Wilcut 1993). Imazaquin at 0.1 to 0.5 ppmw prevented yellow nutsedge shoot growth and provided the best control when soil-applied (Nandihalli and Bendixen 1988).

Imazaquin may have a regulatory effect on the apical dominance of yellow nutsedge since tuber sprouting (y) was linearly enhanced ($y = 2.53 + 3.62x$) by increasing imazaquin rates (x) (Nandihalli and Bendixen 1988). Absorption was primarily by the shoot with rapid initial absorption by the shoot compared with the roots (Nadihalli and Bendixen 1988). Shoot uptake was similar to the acetanilide herbicides and provided greater control than root uptake (Nadihalli and Bendixen 1988). Acropetal and basipetal movement of ^{14}C-imazaquin was observed in yellow nutsedge (Nadihalli and Bendixen 1988).

Chlorimuron PRE reduced yellow nutsedge fresh weight 21 to 62%, and 81 to 83% when applied postemergence with regrowth less than 5% (Derr and Wilcut 1993). Rates as low as 5 g ha^{-1} injured yellow nutsedge (Reddy and Bendixen 1988). Chlorimuron controlled purple nutsedge better when COC was added as compared with a nonionic surfactant or organosilicone adjuvant (Jordan 1996). Yellow nutsedge treated with chlorimuron sprouted 13 to 68% by 28 DAT depending on the chlorimuron rate, and 20 to 25% of the plants sprouted when exposed to soil treated with chlorimuron (Reddy and Bendixen 1989). A strong linear relationship between tuber sprouting (y) and chlorimuron rate (x) was determined ($y = 88.7 - 1.3x$) by Reddy and Bendixen (1989).When tubers were sprouted in soil treated with chlorimuron at 10 g ha^{-1}, yellow nutsedge dry weight was reduced by 98% 28 DAT (Reddy and Bendixen 1989). Foliar and soil-applied chlorimuron was readily absorbed by the shoot and 53% was recovered from the shoot within 48 h after treatment (Reddy and Bendixen 1989). When sprouting tubers were evaluated, less than 2% of the shoot and root-applied chlorimuron was translocated to the tubers (Reddy and Bendixen 1989). Purple nutsedge control was 20 to

28% greater with chlorimuron compared with imazethapyr regardless of the adjuvant and control was greater with both herbicides when plant size was 2 to 6 cm compared to 8 to 10 cm tall (Jordan 1996). Chlorimuron was slowly degraded by yellow nutsedge (Reddy and Bendixen 1988). Using thin-layer chromatography to evaluate metabolites, the Rf 0.57 corresponded to the chlorimuron standard, but Rf 0 was considered a polar metabolite (Reddy and Bendixen 1988).

Limited research has evaluated yellow nutsedge control with the triazolopyrimidine sulfonanilide or pyrimidinyl thiobenzoate herbicides. Cloransulam-methyl, a triazolopyrimidine sulfonanilide, did not control yellow nutsedge (Askew et al 1999). Cloransulam-methyl, applied postemergence following trifluralin, resulted in 72% control 1 to 2 weeks after treatment (Askew et al. 1999). Chlorimuron reduced yellow nutsedge dry weight by 94% compared to a 50% dry weight reduction with cloransulam-methyl 28 DAT in the greenhouse (Nelson and Renner 1998). Pyrithiobac, a pyrimidinyl thiobenzoate, at 72 g ha^{-1} reduced yellow nutsedge shoot number by 29% 50 DAT (Wilcut 1999). When the placement of pyrithiobac was evaluated in the soil compared to postemergence treatments, foliar plus soil-applied pyrithiobac provided greater yellow nutsedge control than either placement applied alone (Wilcut 1999).

Other Herbicides. Several other herbicides have exhibited suppressive or inhibitory capabilities on yellow nutsedge growth. A summary of the effects of these herbicides on yellow nutsedge tubers is presented in Table 4. Differences in yellow nutsedge control may be related to the variety treated. Costa and Appleby (1976) reported that the variety *C. esculentus* var. *leptostachyus* was less susceptible to 2,4-D and more susceptible to

atrazine and metribuzin PRE and PPI than *C. esculentus* var. *esculentus*. 2,4-D stimulated shoot initiation from the tubers and reduced rhizome development of *C. esculentus* var. *leptostachyus* (Costa and Appleby 1976). Translocation of materials to the tubers is greater when the tuber is young than in a mature tuber. When plants were treated with ^{14}C from urea or naphthaleneacetic acid young tubers accumulate the highest levels of ^{14}C while parent tubers accumulate only 8% (Thullen and Keeley 1978). Holt et al. (1967) reported the translocation of arsenic to purple nutsedge tubers. Tuber viability was not related to the presence of arsenic, but was related to the depletion of tuber reserves (Holt et al. 1967).

Amine methylarsonate affected the apical dominance of purple nutsedge tubers and stimulated shoot initiation after treatment (Holt et al. 1967). Dichlobenil provided excellent purple nutsedge control and killed tubers according to sprout tests (Waters and Burgis 1968). PPI applications in the greenhouse of the acetanilide, thiocarbamate, and amide herbicides delayed sprouting, but the photosynthesis inhibitors killed tubers after emergence by causing excess consumption of stored tuber reserves in the tubers (Keeley and Thullen 1974). Incorporated herbicides prevented tuber production and reduced tuber sprout under normal conditions, but under cool conditions exposure to treated soil did not affect tuber sprouting (Keeley and Thullen 1974). Soil-applied fluridone and norflurazon controlled yellow nutsedge eight weeks after treatment and no new tubers were produced in the greenhouse (Banks 1983). Naptalam increased shoot growth and inhibited tuber formation (Appleby and Paller 1978). Bentazon provided good yellow nutsedge control at 1100 g ha^{-1}, but plants regrew after eight weeks (Derr and Wilcut 1993). However,

bentazon at 0.8 and 1.7 kg ha^{-1} killed 86 and 96% of the parent tubers that were treated, but basipetal translocation to the tuber did not occur (Stoller et al. 1975). The arsenical herbicides (DSMA and MSMA) reduced the number of small tubers that sprouted and killed some tubers (Keeley and Thullen 1970). Smaller and younger plants were easier to control with the arsenicals than older larger plants (Keeley and Thullen 1970).

Sulfentrazone applied postemergence at 56 g ha^{-1} controlled yellow nutsedge 45 to 75% 7 DAT depending on the surfactant system utilized, but caused from 30 to 55% injury to soybean (Dayan et al. 1996). Sulfentrazone controlled yellow nutsedge more effectively at pH 6.2 than 4.2 when exposed to the root and sulfentrazone was translocated from the roots to the leaf tissue (Wehtje et al. 1997). Yellow nutsedge was more susceptible to sulfentrazone than purple nutsedge which was probably related to increased tuber uptake of ^{14}C-sulfentrazone by yellow nutsedge compared to purple nutsedge (Wehtje et al. 1997). Postemergence applications of sulfentrazone were not effective in controlling yellow nutsedge, but as soil applications effectively controlled yellow nutsedge (Wehtje et al. 1997).

Alachlor was taken up in the rhizome shoot of yellow nutsedge and the best control was observed when treated soil was placed above the tuber (Armstrong et al. 1973). Armstrong et al. (1973) reported that yellow nutsedge rapidly metabolizes alachlor to a polar metabolite (Rf 0.1). In the field, alachlor did not decrease tuber numbers in soybean while other PPI treatments effectively reduced tuber yield (Banks 1983).

Metolachlor applied 20 d after emergence controlled yellow nutsedge, while bentazon plus metolachlor applied 30 d after emergence provided 92% yellow nutsedge

control (Grichar et al. 1996). Metolachlor at 1.7 kg ha^{-1} gave 72 to 96% control of yellow nutsedge depending on the application timing, and tank mixtures with imazethapyr PPI resulted in 85 to 96% control (Girchar et al. 1992). Dixon and Stoller (1982) reported that metolachlor had no effect on tuber sprout, 4 ppmw did not kill tubers, and less than 3% of the ^{14}C-metolachlor was translocated to the tuber. Metolachlor at 1 ppmw around the tuber reduced growth more than placement above or below (Dixon and Stoller 1982). ^{14}C-metolachlor was translocated from the root to shoot when it was applied to the roots, but basipetal movement was reported when foliar applied (Dixon and Stoller 1982). Yellow nutsedge metabolized metolachlor more slowly than corn (Dixon and Stoller 1982). Metolachlor reduced yellow nutsedge dry weight more than alachlor (Dixon and Stoller 1982). Yellow nutsedge metabolized metolachlor to approximately ten metabolites that were similar to corn, but differential susceptibility between corn and yellow nutsedge was related to slower metabolism by yellow nutsedge compared with corn (Dixon and Stoller 1982). Dixon and Stoller (1982) also observed that ^{14}C-metolachlor was exuded from the yellow nutsedge roots.

Hormones. Hormones also affect the differentiation of rhizomes and the growth of yellow nutsedge. Research has evaluated GA and auxins. GA is a plant hormone that stimulates cell division and elongation along with increased starch degradation in cereal grain seeds (Taiz and Zeigler 1991). Auxins are plant hormones that regulate apical dominance, inhibit abscission, stimulate ethylene synthesis, affect flowering, stimulate fruit development, and stimulate root development on cuttings (Taiz and Zeigler 1991). After GA was applied three times to the youngest shoot tip, yellow nutsedge rhizomes grew

erect under several environmental conditions (Bendixen 1970a). 1000 ppm GA reduced tuber differentiation of plants in a 12.5 h photoperiod, but promoted tuberization under long photoperiods (Garg et al. 1967). At low temperatures and under a short photoperiod, GA had limited effects on rhizome suppression (Bendixen 1970a). Indoleacetic acid (IAA) and triiodobenzoic acid (TIBA), synthetic auxins, alone or with GA had no effect upon rhizome growth, but under high levels of naptalam or TIBA shoot initiation was stimulated from the rhizomes (Bendixen 1970a). If one could increase rhizome differentiation into above ground growth, then treatment and control of yellow nutsedge may be similar to an annual weed.

BIOLOGICAL CONTROL

Biological control methods were reviewed by Phatak et al. in 1987. Over 66 insects and 10 pathogens were associated with yellow nutsedge (Phatak et al. 1987). Other organisms like insects, nematodes, bacteria, and birds had been evaluated as biocontrol methods (Daniel's 1997, Neeser et al. 1997, Phatak et al. 1987). The infection of the sedge, *Cyperus virens*, with a fungus caused flower abortion which was an effective method of control since the sedge did not produce rhizomes. However, the success of biocontrol methods for yellow nutsedge control has been limited. For example, Keeley et al. (1970) reported that the infestation of the insect *Bactra verutana* Zeller was variable. The insect had little effect on tuber production and showed little promise as a biocontrol agent since the insect infested yellow nutsedge in the fall (Keeley et al. 1970).

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Table 1. Taxonomic key from Schippers et al. (1995) for *Cyperus esculentus* varieties based primarily on flower characteristics of plants grown in the field and over different fertility, temperature, and day length conditions in the greenhouse.

I. a) Angle between spikelet and rachis is < 35°.
b) Spikelet density is > 2 mm^{-2}.
c) Ratio of ray number to bracts is < 0.75 or > 15 flowers $spikelet^{-1}$. Variety ..... *heermannii*

I. Other characteristic combinations.
a) Floral scale length mean is ≥ 3.4 mm. Variety ..... *macrostachyus*
b) Floral scale length mean is < 3.4 mm.

II. a) Floral scale length mean is ≥ 2.8 mm.
b) Height of the floral scale maximum width is ≥ 1.5 mm. Variety ..... *macrostachyus*

II. a) Floral scale length mean is < 2.8 mm.
b) Height of the floral scale maximum width is < 1.5 mm.

1. a) Floral scale length mean is ≥ 2.5 mm.
   b) Floral scale reaches maximum width at ≥ 1.4 mm.
   c) Floral scale width maximum is > 1.8 mm.
   d) Ratio of the top floral scale to the mean length of the spikelet floral scales is > 0.9 or number for floral scales $spikelet^{-1}$ is ≥ 20. Variety .. *macrostachyus*
2. Other characteristic combinations.

III. Style length with branches is ≥ 4.2 mm. Variety ..... *esculentus*

III. Style length with branches is < 4.2 mm.
a) Maximum floral scale width is ≤ 1.5 mm. Variety ..... *leptostachyus*
b) Maximum floral scale width is > 1.5 mm.

IV. Floral scales spikelet is > 23. Variety ..... *leptostachyus*

IV. Floral scales spikelet is ≤ 23.
a) Top floral scale length:floral scale length > 0.94. Variety ..... *leptostachyus*
b) Top floral scale length:floral scale length ≤ 0.94.

V. Floral scale width is ≤ 1.8 mm and style length is ≤ 3.4 mm. Variety ..... *leptostachyus*

V. Floral scale width is ≤ 1.8 mm and style length is ≤ 3.4 mm.
a) Floral scale width is ≤ 1.9 mm, floral scale length mean is ≤ 2.3 mm, and anther length is ≤ 1.4 mm. Variety ..... *leptostachyus*
b) Floral scale width is > 1.9 mm, floral scale length mean is > 2.3 mm, and anther length is > 1.4 mm.

VI. a) Floral scale width is ≤ 2.0 mm, floral scale length mean is ≥ 2.6 mm, and style length is ≤ 3.5 mm or the angle between the floral scale and spikelet axis is < 17°. Variety ..... *leptostachyus*
b) Other characteristic combinations. Variety ..... *esculentus*

Table 2. Environmental stimulus and the effect on yellow nutsedge tuber production and sprouting.

Environmental stimulus[a]	Effect on tubers	Source
Temperature		
Increase from 21 to 27 C	At 12.5 h photoperiod, tuber production increased.	Garg et al. 1967
6 and 43 C	Threshold temperatures for tuber sprout.	Holt and Orcutt 1996
Alternating	Faster and improved tuber sprout.	Miles et al. 1996
-7 C	LD_{50} temperature.	Stoller and Wax 1973
12 C	Minimum temperature required for sprouting.	Stoller and Wax 1973
22 C vs 4 C	Increased survival and weight loss at higher temperature.	Thomas 1969
Cold treatment	Increased sprouting from 12% in the fall to 95% in the spring.	Tumbleson and Kommedahl 1962
Degree-days California genotype	114 degree-day units were needed before predicting emergence.	Wilen et al. 1996b
Degree-days Arizona genotype	111 to 115 degree-day units were needed before predicting emergence.	Wilen et al. 1996b
Relative Humidity		
30 vs 90%	Increased survival and reduced tuber weight loss at high relative humidity.	Thomas 1969
Dessication	Reduced tuber sprouting.	Thomas 1969
Soil Depth		
2.5 to 5.1 cm	Suffered winter kill.	Stoller and Wax 1973
10.2 to 20.3 cm	Greatest tuber emergence was observed.	Stoller and Wax 1973
Top 25 cm of soil	Location of over 99% of the tubers.	Tumbleson and Kommedahl 1961
Scarification		
Cut tuber in half	Increased tuber sprouting.	Tumbleson and Kommedahl 1962
Wash with water	Increased sprouting.	Tumbleson and Kommedahl 1961, 1962
Light		
Day length	Long d had limited tuber production and short d induced tuber formation.	Appleby and Paller 1978

Table 2 (cont'd).

Photoperiod	Reduced photoperiod to 10 h induced tuberization.	Bendixen 1973
Photoperiod	Reduced photoperiod from 14.5 to 12 h to stimulate tuber production.	Costa and Appleby 1976
Photoperiod	Short d (12.5) stimulated greater tuber production than 14 and 15.5 h d.	Garg et al. 1967
Photoperiod	8 to 12 h had the greatest tuber development from rhizome tips and tubers were produced by 3 months after planting.	Jansen 1971
80 to 94% continuous LI	Net gain of tubers.	Keeley and Thullen 1978
30% reduction in PAR	Reduced tuber production 32%.	Keeley and Thullen 1978
94% reduction in PAR	Reduced tuber production 95%.	Keeley and Thullen 1978
Crop selection	Tuber production was reduced 18 to 43% under reduced light intensities. Hemp had early canopy closure.	Lotz et al. 1991
80% incident PAR	Reduced tuber dry weight to 100 mg $plant^{-1}$ and production to 3 $plant^{-1}$.	Santos et al. 1997
Photoperiod	12 h was needed for tuber production.	Santos et al. 1997
Hormones		
2,4-D	Shoot, rhizome, and rhizome number were reduced 12 DAT.	Bhan et al. 1970
Gibberillic acid	Reduced tuberization and increased shoot production under short photoperiods. Increased tuberization and reduced shoot production under long photoperiods.	Garg et al. 1967
ABA	Inhibited and delayed tuber sprouting.	Jangaard et al. 1971
Gibberillic acid	Promoted tuber sprouting one week after planting.	Tumbleson and Kommedahl 1962
N-6 Benzyl adenine	100 ppm stimulated tuber sprouting.	Zandstra and Nishimoto 1977

[a]Abbreviations: ABA, abscisic acid; LI, Light interception; and PAR, photosynthetically active radiation.

Table 3. Yellow nutsedge sprouting methods and environmental conditions for yellow nutsedge sprouted in controlled environments.

Sprouting environment	Sprouting mechanism and media	Storage or collection characteristics	Source
30 C over a 3 week period		plastic bags at 5 C for 3 weeks	Appleby and Paller 1978
		collected tubers in March	Banks 1983, Keeley and Thullen 1974
27 C with a 16 h photoperiod	growth chamber		Bendixen 1973
darkness with 14 h at 30 C and 10 h at 15 C			Dixon and Stoller 1982
3 to 6 d with an alternating 8/16 h light at 30 C	absorbent paper		Jordan-Molero and Stoller 1978
	moist vermiculite	2 C to break dormancy	Keeley and Thullen 1970
	soil	2 C for 4 weeks	Keeley et al. 1979, 1983
30 C and 100% relative humidity		3 C for 6 weeks	Reddy and Bendixen 1989
Dark at 25 C	absorbent towels		Stoller et al. 1972
25 C in the dark for 14 d			Stoller et al. 1979
30 C for 8 h and 20 C for 16 h	germinator		Stoller and Wax 1973

Table 3 (cont'd).

14 d at 30 C for 8 h and 20 C for 16 h		stored in plastic bags at high humidity for 6 weeks at 2C	Stoller and Weber 1975
28 C			Taylorson 1967
28 C	germinator in petri dishes		Tumbleson and Kommedahl 1961
28 C			Tumbleson and Kommedahl 1962
	paper towels in aluminum foil covered trays in the greenhouse		Vencill et al. 1995
48 h at 30 C with 90% relative humidity	moist towels in a growth chamber		Wehtje et al. 1997
Tubers collected from dormant plants		6 weeks at 4 C before planting	Wilen et al. 1996a

Table 4. Herbicide efficacy or yellow nutsedge control in the field or greenhouse and the effect on yellow nutsedge tubers.[a]

Control method	Field/greenhouse conditions and/or the control level in the respective crop.	Effect on tubers	Source
Acetachlor	Soybean 68 to 93% 90 DAT	Reduced viable and nonviable tubers 92 and 87%[c].	Banks 1983
Alachlor	Soybean 50 to 67% 90 DAT	Reduced viable and nonviable tubers 51 and 59%[c].	Banks 1983
Alachlor PPI[b]	Greenhouse 16 to 49% of the control 12 WAT.	54 to 89% tuber sprout and 5 to 7 % soft tubers.	Keeley and Thullen 1974
Alachlor PPI	Did not kill tubers in the greenhouse.	No effect on tuber sprouting.	Dixon and Stoller 1982
Atrazine PPI[b]	Greenhouse 5 to 14% control 12 WAT.	94 to 97% tuber sprout and 83 to 87% soft tubers.	Keeley and Thullen 1974
Bentazon	Greenhouse and soybean	86 to 96% tuber death at 0.8 and 1.7 kg ha^{-1}, respectively.	Stoller et al. 1975
Bromacil PPI[b]	Greenhouse 0% control 12 WAT.	93 to 96% tuber sprout and 92 to 97% soft tubers.	Keeley and Thullen 1974
Butachlor PPI[b]	Greenhouse, 16 to 31% control 12 WAT.	77 to 68% tuber sprout and 0 to 3% soft tubers.	Keeley and Thullen 1974
Butylate	Barley-corn rotation for 3 years	92% reduction in firm and 96% reduction in viable tubers.	Keeley et al. 1979
Chlorimuron	Greenhouse, 84% control 28 DAT at 20 g ha^{-1}	13% sprouted at 5 g ha^{-1}.	Reddy and Bendixen 1988
Chlorimuron	Greenhouse	Treated soil reduced sprouting 20 to 25%.	Reddy and Bendixen 1989

Table 4 (cont'd).

Cultivation		Reduced tuber production from 97 to 99%.	Hauser et al. 1974
Cultivation	Corn	Reduced tubers m^{-2} 53%.	Stoller et al. 1979
Cycloate PPI[b]	Greenhouse, 3 to 19% of the control 12 WAT.	37 to 76% tuber sprout and 4 to 5% soft tubers.	Keeley and Thullen 1974
Diethatyl	Soybean 73 to 85% 90 DAT	Reduced viable and nonviable tubers 74 and 66%[c]	Banks 1983
DSMA	Greenhouse	Reduced small tuber sprouting and weight was reduced 50%.	Keeley and Thullen 1970
EPTC PPI[b]	Greenhouse, 31 to 71% control 12 WAT.	66 to 87% tuber sprout and 2 to 5% soft tubers.	Keeley and Thullen 1974
EPTC PPI	Corn at 5.6 kg ha^{-1}	Reduced tubers m^{-2} 94%.	Stoller et al. 1979
EPTC	Alfalfa for 3 years	95% reduction in firm and 97% reduction in viable tubers.	Keeley et al. 1979
Fallow treatments		Reduced tuber sprout 44% and tuber yield 99%.	Tumbleson and Kommedahl 1961
Fluridone	Cotton 93 to 95% 90 DAT	Reduced viable and nonviable tuber 97 and 89%[c].	Banks 1983
Halosulfuron	Greenhouse, 54 to 65% dry weight reduction 30 DAT.	Reduced tuber regrowth greater than 96% and root-tuber dry weight 82 to 87%.	Vencill et al. 1995
Handhoeing	Timely hoeing with 100% control in cotton.	Reduced tuber number by 70 to 90%.	Keeley and Thullen 1983
Handhoeing	Cotton	24% reduction in tuber production.	Keeley and Thullen 1975

Table 4 (cont'd).

Methazole PPI[b]	Greenhouse, 0 to 1% control 12 WAT.	99% tuber sprout and 91 to 98% soft tubers.	Keeley and Thullen 1974
Metolachlor	Did not kill the tubers in the greenhouse.	No effect on tuber sprouting.	Dixon and Stoller 1982
Metolachlor	Soybean 88 to 92% 90 DAT	Reduced viable and nonviable tubers 87 and 79%[c].	Banks 1983
Metolachlor	Greenhouse	No effect on tuber sprouting.	Dixon and Stoller 1982
MSMA	Cotton for 3 years	83% reduction in firm and 91% reduction in viable tubers.	Keeley et al. 1979
MSMA	Greenhouse	Reduced small tuber sprouting and arsenic level was higher than with DSMA.	Keeley and Thullen 1970
Napropamide PPI[b]	Greenhouse, 1% control 12 WAT.	2 to 9% tuber sprout and 2 to 3% soft tubers.	Keeley and Thullen 1974
Naptalam	Greenhouse stimulated shoot growth from rhizomes.	Reduced tuber production.	Appleby and Paller 1978
Norflorazon	Cotton 75% 90 DAT	Reduced viable and nonviable tubers 74 and 57%[c], respectively.	Banks 1983
Perfluidone	Cotton 60 to 90% 90 DAT	Reduced viable and nonviable tubers 87 and 63%[c], respectively.	Banks 1983
Population	Increase corn from 66,700 to 133,000 plants ha^{-1}	Reduced tuber number 71%.	Ghafar and Watson 1983a

Table 4 (cont'd).

Prometryne PPI[b]	Greenhouse, 1 to 11% control 12 WAT.	90 to 97% tuber sprout and 69 to 97% soft tubers, respectively.	Keeley and Thullen 1974
Pyrithiobac at 36 and 72 g ha^{-1}	Greenhouse, -22 and 17% dry weight reduction 50 DAT, respectively	-26 and 27% root and tuber dry weight reduction, respectively.	Wilcut 1998
San-6706 PPI[b]	Greenhouse, 2 to 10% control 12 WAT.	98 to 99% tuber sprout and 88 to 90% soft tubers.	Keeley and Thullen 1974
Terbacil PPI[b]	Greenhouse, 0% control 12 WAT	98% tuber sprout and 95 to 96% soft tubers.	Keeley and Thullen 1974
U-27267 PPI[b]	Greenhouse, 7 to 10% control 12 WAT	49 to 66% tuber sprout and 3 to 8% soft tubers.	Keeley and Thullen 1974
UBI-S-734	Cotton 87 to 97% 90 DAT	Reduced viable and nonviable tuber 83 and 40%[c].	Banks 1983
Vernolate	Soybean 78 to 85% 90 DAT	Reduced viable and nonviable tubers 79 and 68%[c].	Banks 1983

[a]Abbreviations: DAT, days after treatment; PPI, preplant incoporated; PRE, preemergence; and WAT, weeks after treatment.
[b]Under cool conditions, exposure to treated soil did not affect tuber sprouting.
[c]Evaluated after the second year of the same treatment.

Table 5. Yellow nutsedge control and the effect on tubers with glyphosate.[a]

Glyphosate rate	Control or weight reduction	Effect on tubers	Source
(g ai ha^{-1})	(%)		
300		43% sprouted, 57% remained dormant, and 0% were nonviable.	Appleby and Paller (1978)
600	59 to 79% control, 43 to 45% fresh weight reduction 10 DAT	reduced resprouting from tubers 14 to 32%, viability was reduced.	Keeley et al. 1985
600		27% sprouted, 68% remained dormant, and 5% were nonviable.	Appleby and Paller (1978)
750 treated 40 DAE	44% fresh weight reduction		Pereira and Crabtree (1986)
750 treated 70 DAE	93% fresh weight reduction		Pereira and Crabtree (1986)
1100		12% sprouted, 47% dormant, 42% nonviable.	Appleby and Paller (1978)
1100 or 2200 on 4 to 5 leaf plants	100% control.	0 to 3% of parent tubers were killed.	Stoller et al. 1975
1100 or 2200 on 5 to 7 leaf plants	100% control.		Stoller et al. 1975
1100 or 2200 on 7 to 9 leaf plants	97 and 100% control, respectively.		Stoller et al. 1975
1500	57% root and rhizome dry weight reduction, 0% regrowth	56% reduction in tubers produced $plant^{-1}$ and 52% reduction in tuber fresh weight $plant^{-1}$.	Pereira and Crabtree (1986)

Table 5 (cont'd).

1500 treated 40 DAE	99% fresh weight reduction		Pereira and Crabtree (1986)
1500 treated 70 DAE	98% fresh weight reduction		Pereira and Crabtree (1986)
2200		10% sprouted, 52% dormant, and 38% were nonviable.	Appleby and Paller (1978)
2240 on fallow barley		12% of the tubers were firm and 2% were viable after 3 years.	Keeley et al. 1979
3400	87% fresh weight reduction and 94% regrowth fresh weight reduction		Derr and Wilcut 1993
0.1 mM	5% fresh weight reduction, 7% dry weight reduction	13% dry weight reduction of underground plant parts.	Villaneuva et al. 1985
1.0 mM	9% fresh weight reduction, 3% dry weight reduction	33% dry weight reduction of underground plant parts.	Villaneuva et al. 1985
5.0 mM	19% fresh weight reduction, 2% dry weight reduction	60% dry weight reduction of underground plant parts.	Villaneuva et al. 1985
10.0 mM	40% fresh weight reduction, 10% dry weight reduction	80% dry weight reduction of underground plant parts.	Villaneuva et al. 1985

[a]Abbreviations: DAE, d after emergence; and DAT, d after treatment.

Table 6. Absorption and translocation of glyphosate, ALS-inhibitors, and other herbicides in yellow nutsedge.

Glyphosate

Absorbed	Translocated	Source
32% of the applied amount (1000 g ha^{-1} application rate).	28% of the applied amount. 22% of the translocated herbicide was moved to below ground plant parts.	Pereira and Crabtree 1986
	83% in shoot 15% to underground tissue 2% in the tubers	Keeley et al. 1985
	20% was translocated from the treated leaf of purple nutsedge	Zandstra and Nishimoto 1977

ALS-Inhibitors

Absorbed	Translocated	Source
Imazaquin		
83% in the shoot and 1% from the roots after 48 h	Acropetal and basipetal movement.	Nadihalli and Bendixen 1988
Chlorimuron		
12% foliar	15% in 1 d, 78% remained in the leaf, acropetal and basipetal, 0.2% in the tuber	Reddy and Bendixen 1988
47% foliar in 12 h 1.3% root	< 1% translocated to the tubers	Reddy and Bendixen 1989

Other herbicides

Absorbed	Translocated	Source
2,4-D		
<19% after 24 h	<1% translocated, but not metabolized	Bhan et al. 1970
Metolachlor		
roots absorbed 8%	53% after 13 d, acropetal	Dixon and Stoller 1982
shoots	basipetal movement and was exuded	Dixon and Stoller 1982
Alachlor		
uptake is above the tuber	shoots had limited acropetal movement	Armstrong et al. 1973
Sulfentrazone		
43% was absorbed	<1% in the tubers	Wehtje et al. 1997

CHAPTER 6

YELLOW NUTSEDGE (*Cyperus esculentus*) CONTROL AND TUBER PRODUCTION WITH GLYPHOSATE AND ALS-INHIBITING HERBICIDES

Abstract: Greenhouse and field research evaluated yellow nutsedge growth, vegetative control, and tuber production following an application of glyphosate, acetolactase synthase (ALS)-inhibiting herbicides, and tank mixtures thereof. In the greenhouse or field, glyphosate at 840 g ae ha^{-1} did not control yellow nutsedge. Chlorimuron and imazethapyr/imazapyr provided 90% or greater yellow nutsedge control, prevented panicle formation, and reduced tuber density and fresh weight by 90% or more 14 weeks after treatment (WAT) in the greenhouse. The addition of glyphosate to cloransulam or imazethapyr increased yellow nutsedge control and reduced tuber density and fresh weight more than either treatment or glyphosate applied alone. Imazamox at 45 g ai ha^{-1} applied alone stimulated tuber production. In the field, halosulfuron and chlorimuron provided greater than 85% yellow nutsedge control and reduced tuber density and fresh weight more than 80 and 85%, respectively. All treatments reduced tuber fresh weight 45 to 91% when compared to the untreated control, and tuber density was reduced 33 to 90% by all herbicide treatments except imazamox and rimsulfuron applied alone compared to the untreated control. Tank mixtures of glyphosate with the ALS-inhibiting herbicides increased visual control, but did not reduce tuber production compared to the ALS-inhibiting herbicides applied alone. Tuber sprouting was reduced 19% in plots treated with halosulfuron and pyrithiobac compared to untreated yellow nutsedge 42 WAT. Yellow nutsedge control by the herbicides evaluated and applied at recommended use

rates was ranked: halosulfuron and chlorimuron (> 80% control and reduction in tuber density); imazethapyr/imazapyr, imazethapyr, and glyphosate (50 to 80% control and reduction in tuber density); and cloransulam, rimsulfuron, and imazamox (20 to 50% control and reduction in tuber density). However, pyrithiobac controlled yellow nutsedge 48% and reduced tuber density 60%. Long-term yellow nutsedge management may be achieved with treatments that reduce tuber production.

Nomenclature: chlorimuron, 2-[[[[(4-chloro-6-methoxy-2-pyrimidnyl)amino]carbonyl]amino]sulfonyl]benzoic acid; cloransulam, 3-chloro-2-[[(5-ethoxy-7-fluoro[1,2,4]triazolo[1,5-*c*]pyrimidin-2yl)sulfonyl]amino]benzoic acid; glyphosate, *N*-(phosphonomethyl)glycine; imazapyr, (±)-2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1*H*-imidazol-2-yl]-3-pyridinecarboxylic acid; MON 12000 or MON 12037 (proposed halosulfuron), methyl 5-[[(4,6-dimethoxy-2-pyrimidinyl)amino]carbonylaminosulfonyl]-3-chloro-1-methyl-1-*H*-pyrazole-4-carboxylate; imazethapyr, 2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1*H*-imidazol-2-yl]-5-ethyl-3-pyridinecarboxylic acid; imazamox, 2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1*H*-imidazol-2-yl]-5-(methoxymethyl)-3-pyrdinecarboxyilic acid; pyrithiobac, 2-chloro-6-[(4,6-dimethoxy-2-pyrimidinyl)thio]benzoic acid; rimsulfuron, *N*-[[(4,6-dimethoxy-2-pyrimidinyl)amino]carbonyl]-3-(ethylsulfonyl)-2-pyridinesulfonamide; yellow nutsedge, *Cyperus esculentus* L. #[23] CYPES.

Additional index words: acetolactase synthase inhibitor, plant height, postemergence, shoot production, tubers, tuber production.

Abbreviations: ALS, acetolactate synthase; AMS, ammonium sulfate ($(NH_4)_2SO_4$); COC, crop oil concentrate; MSO, methylated seed oil; NIS, nonionic surfactant; WAT, weeks after treatment.

[23]Letters following this symbol are a WSSA-approved computer code from Composite List of Weeds, Revised 1989. Available from WSSA, 1508 West University Ave., Champaign, IL 61821-3133.

INTRODUCTION

Yellow nutsedge is a problematic perennial weed found throughout the world (Bendixen and Nandihalli 1987, Lapham 1985, Schippers et al. 1995). Yellow nutsedge is prolific and aggressive due to its rapid growth (Holt and Orcutt 1991) and ability to propagate through rhizomes and tubers. Several buds on each tuber can sprout and cause reinfestation after tillage or selective herbicide treatments. Yellow nutsedge competes with crops early in the growing season (Ghafar and Watson 1983a, Keeley 1987), and densities from 40 to 1000 plants m^{-2} have reduced agronomic and horticultural crop yields (Keeley 1987). In reviews by Glaze (1987) and William and Bendixen (1987), prevention, crop selection and rotation, row spacing, tillage, fumigation, and herbicides were some of the strategies available to manage this weed.

Yellow nutsedge tubers are enlargements produced at the terminal tip of the rhizome (Bendixen 1970, Bendixen 1973, Gifford and Bayer 1995, Tumbleson and Kommedahl 1961, Wills et al. 1980). Tubers consist of compressed nodes with a lignified epidermis and scale leaves that are tightly packed against the tuber surface (Bendixen 1973). Scale leaves encompass the tuber for protection as it develops from an immature white tuber to a mature brown, tan, or black tuber (Gifford and Bayer 1995, Jansen 1971). Tubers are usually less than 10 mm long (Bendixen 1973), but may range from 3 to 11 mm in length (Tumbleson and Kommedahl 1961). Larger tubers have a longer life span and produce uniform sprouts with a greater weight than smaller tubers (Thullen and Keeley 1975). One tuber planted in the spring may produce 1900 shoots and 6900 tubers in one year (Tumbleson and Kommedahl 1961). Up to 10% of the total dry weight produced by

yellow nutsedge is partitioned into the tubers (Holt and Orcutt 1991). Tumbleson and Kommedahl (1961) reported that over 99% of the tubers produced by yellow nutsedge were located in the top 25 cm of soil. Tubers are the primary means of propagation and may have allelopathic effects on the crop (Drost and Doll 1980, Tumbleson and Kommedahl 1962). The long-term control of yellow nutsedge depends on killing the parent tuber and preventing daughter tuber production.

Crop management used in conjunction with herbicide treatments may reduce tuber viability, germination, and yield. For instance, tuber yield, growth, and daughter tuber production were reduced following two years of several herbicide treatments in cotton (*Gossypium hirsutum* L.) or soybean (Banks 1983). Stoller et al. (1975) reported that bentazon killed the parent tubers of treated plants and no regrowth was observed. In other research, cultivation reduced yellow (Stoller et al. 1979) and purple nutsedge (*Cyperus rotundus* L.) (Webster and Coble 1997) tuber density in the presence and absence of herbicides. The integration of crop rotation and herbicide selection effectively reduced yellow nutsedge tuber production to 5 to 17% of the original population and reduced tuber viability 3 to 9% (Keeley et al. 1979, Keeley et al 1983). Yellow nutsedge tubers can resprout after a herbicide application or new shoots may arise from underground rhizomes if the herbicide has no residual activity.

The introduction of glyphosate-resistant crops and the acetolactate synthase-inhibiting (ALS, E.C. 4.1.3.18) herbicides has provided more postemergence perennial weed management options for crop producers. As glyphosate resistant crops become more widespread, producers may select for weeds like yellow nutsedge that are not

completely controlled at a typical use rate of glyphosate. Variable yellow nutsedge control with glyphosate has been reported and was related to the application rate and the size of the plants at application. A single application of glyphosate at 430, 630 or 840 g ha^{-1} in glyphosate-resistant corn did not control yellow nutsedge (Fischer and Harvey 1998). Yellow nutsedge required at least 2200 g ha^{-1} of glyphosate for control, but parent tubers were not killed (0 to 3%) with 1100 or 2200 g ha^{-1} application regimes (Stoller et al. 1975). However, Appleby and Paller (1978) reduced the number of tubers produced per plant and increased the number of nonviable tubers as the rate of glyphosate increased from 300 to 2200 g ha^{-1}. Poor yellow nutsedge control with glyphosate has been related to slow absorption and limited translocation (Periera and Crabtree 1986), plant size at the time of application (Keeley et al 1985, Pereira and Crabtree 1986), and environmental conditions (Chase and Appleby 1979).

Studies have evaluated yellow nutsedge control with the ALS-inhibiting herbicides including the sulfonylurea (Ackley et al. 1996, Derr et al. 1996, Molin et al. 1999, Reddy and Bendixin 1988 and 1989), imidazolinone (Ackley et al. 1996, Czarnota and Bingham 1997, Derr and Wilcut 1993, Grichar et al. 1992, Nadihalli and Bendixen 1988, Richburg III et al. 1993), triazolopyrimidine sulfonanilide (Askew et al. 1999, Nelson and Renner 1998), and pyrimidinyl thiobenzoate (Ackley et al. 1996, Wilcut et al. 1999) herbicide families. Several of the ALS-inhibiting herbicides controlled yellow nutsedge when there was both foliar and soil uptake (Reddy and Bendixen 1989, Richburg III et al. 1993, Vencill et al. 1995, Wilcut 1999). A postemergence application of glyphosate used in

combination with an ALS-inhibiting herbicide with residual activity may provide consistent yellow nutsedge control.

The interactions between glyphosate, chlorimuron, imazamox, imazaquin, and halosulfuron were evaluated for purple nutsedge control in greenhouse research (Rao and Reddy 1999). All tank mixture treatments were additive when applied to purple nutsedge that was 6 weeks old, but 1120 g ha^{-1} of glyphosate alone reduced purple nutsedge fresh weight 100% (Rao and Reddy 1999). Imazaquin and pyrithiobac applied at reduced rates and tank mixed with glyphosate at 560 g ha^{-1} antagonized purple nutsedge control (Rao and Reddy 1999). Antagonistic combinations with glyphosate plus ALS-inhibiting herbicides have been reported on difficult-to-control weeds like velvetleaf (*Abutilon theophrasti* Medicus) (Lich et al. 1997), ivyleaf morningglory [*Ipomoea hederacea* (L.) Jacq.] (Lich et al. 1997), purple nutsedge (Rao and Reddy 1999), and sicklepod (*Cassia obtusifolia* L.) (Rao and Reddy 1999). However, Starke and Oliver (1998) reported that chlorimuron did not affect the absorption and translocation of ^{14}C-glyphosate by velvetleaf or pitted morningglory (*Ipomoea lacunosa* L.), but glyphosate increased the absorption of ^{14}C-chlorimuron or ^{14}C-imazethapyr by velvetleaf. ALS-inhibiting herbicides and glyphosate are translocated to actively growing tissue where they inhibit amino acid synthesis. Since both herbicides are translocated, these herbicide tank mixtures could provide greater yellow nutsedge control than either herbicide applied alone.

Additives may also increase yellow nutsedge control with glyphosate. The addition of ammonium sulfate from 1.25 to 10 kg ha^{-1} and other ammonium salts to glyphosate increased purple nutsedge control four fold (Suwunnamek and Parker 1975).

However, yellow and purple nutsedge control was not increased by adding ammonium sulfate (Fischer and Harvey 1998) or organosilicone surfactants (Bariuan et al. 1999) to glyphosate (formulated as Roundup Ultra®)[24], respectively. Similarly, the addition of crop oil concentrate, methylated seed oil, or nonionic surfactant to glyphosate did not affect control (Nelson and Renner 1999).

Yellow nutsedge control depends on reducing the propagative potential of this weed. This research was initiated to evaluate yellow nutsedge growth suppression and tuber production following applications of glyphosate, ALS-inhibiting herbicides, and tank mixtures of glyphosate with the ALS-inhibiting herbicides. In addition, the growth rate of yellow nutsedge during the season was determined. Rapid growth rate of a perennial weed like yellow nutsedge early in the season may result in a competitive advantage to this weed, and understanding yellow nutsedge growth will improve the timing of weed management practices.

MATERIALS AND METHODS

Greenhouse Methods.

Locally collected tubers were stored at 4 C. Tubers were germinated in a Freas 815 Incubator[25] and two sprouted tubers were planted in 4-L pots of Spinks loamy sand (mixed, mesic Psammentic Hapludalfs) with pH 5.2 and 1.1% organic matter. Plants were grown in a 16-h photoperiod of natural and supplemental sodium vapor lighting that

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[24]Roundup Ultra, Monsanto Co., 800 N. Lindbergh Blvd., St. Louis, MO 63167.

[25]Precision Scientific Co., 3737 West Cortland St., Chicago, IL 60647.

provided a photosynthetic photon flux density[26] of 120 μE $m^{-2} \cdot s^{-1}$, fertilized with 0.1 g of water soluble fertilizer (20% N, 20% P_2O_2, 20% K_2O) every two weeks, and surface irrigated as needed. Environmental conditions were maintained at 27 $\pm$ 5 C. Yellow nutsedge had 6 to 8 leaves per shoot and was 13 to 18 cm tall at the time of herbicide application. Herbicide treatments included glyphosate (formulated as Roundup Ultra) at 840 g ae ha^{-1} plus spray grade AMS at 20 g L^{-1} applied alone and in combination with ALS-inhibiting herbicides which were applied with recommended adjuvants (nonionic surfactant[27] (NIS) at 0.25% v/v, crop oil concentrate[28] (COC) at 1.3% v/v, or methylated seed oil[29] (MSO) at 1.0% v/v) for yellow nutsedge control (Table 1). Herbicide applications were made with a continuous link-belt sprayer traveling at 1.5 km hr^{-1} and equipped with an 8001 even flat-fan nozzle[30] calibrated to deliver 234 L ha^{-1} at 193 kPa of pressure. The soil was moist at the time of herbicide application and soil temperature was 26 $\pm$ 1 C. The experiment was arranged in a randomized complete block design with four replications and was repeated in time. Yellow nutsedge control was evaluated on a scale

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[26]LI-COR. 4421 Superior Street, Lincoln, NE 68504.

[27]Nonionic surfactant was Activator-90, a mixture of alkylpolyoxyethylene ethers and free fatty acids, Loveland Industries Inc., P.O. Box 1289, Greeley, CO 80632.

[28]Crop oil concentrate was Herbimax, paraffinic oil plus emulsifiers plus surfactants, Loveland Industries Inc., P.O. Box 1289, Greeley, CO 80632.

[29]Methylated seed oil was Sun-It II, methylated seed oil plus surfactants, Agsco, Inc., P.O. Box 13458, Grand Forks, ND 58208-3458.

[30]Teejet flat-fan tips. Spraying Systems Co., North Avenue and Schmale Road, Wheaton, IL 60188.

from 0 (no growth suppression) to 100 (complete weed death) 4 and 14 WAT. Height, panicle number, shoot dry weight reduction, tuber number reduction, tuber weight reduction, and total root fresh weight excluding tubers was evaluated 14 WAT after the untreated control had flowered and appeared physiologically mature. Shoot dry weight, root fresh weight, tuber number, and tuber weight reduction were calculated as the percent reduction of the measured parameter compared to the untreated control.

Field Methods.

Research was conducted in 1997 near Alto, MI and 1998 at East Lansing, MI in fields with a previous history of heavy, uniform yellow nutsedge infestation. The soil was a Boyer sandy loam (coarse-loamy, mixed, mesic Typic Hapludalf) with 2.9% organic matter and pH 6.6 in 1997 and a Marlette sandy loam (fine-loamy, mixed mesic Glossoboric Hapludalf) with 1.7% organic matter and pH 6.7 in 1998. The field was cultimulched 8 cm deep May 27, 1997 and nutsedge began to emerge by June 6. In 1998, the field was soil-finished 6 cm deep on May 13 and nutsedge emergence began on May 21.

This experiment was arranged as a randomized complete block design with four replications each year in plots 3 by 6.1 m. Herbicide treatments were the same as previously described. Yellow nutsedge shoot density was 1550 shoots m^{-2} and shoots were 10 to 20 cm tall with 4 to 8 leaves at the time of application. Herbicides were applied on June 26, 1997 and June 23, 1998 with a tractor mounted compressed-air sprayer traveling 6.3 km h^{-1} and delivering 178 L ha^{-1} at 207 kPa equipped with 8003 flat-fan nozzles. In 1997, treatments were applied at 2100 h with an air temperature 25 C and

37% relative humidity. The treatments were applied at 1900 h with an air temperature 33 C and 30% relative humidity in 1998.

Yellow nutsedge height was recorded at frequent intervals from emergence until flowering and the growth rate was fit to a second order polynomial using the scatter plot option in Microsoft Excel-95[31]. Visual weed control was evaluated at 2, 4, and 8 WAT, heights at 2, 4 and 6 WAT, and two 30 by 30 cm quadrats were harvested 2, 4, and 8 WAT from each treatment to evaluate shoot density and vegetative biomass.

Initial tuber densities were measured in the field prior to herbicide application by randomly removing soil cores from each replication. In April (42 WAT) the following year, eight soil cores were removed with a 10 cm diameter putting green cup cutter to a 20 cm depth. Tubers were separated from the soil with a modified Kenmore 80[32] clothes washer powered by a hydraulic orbital motor and sorted by hand into groups of small (<5 mm), medium (5-8 mm), and large (>8 mm) tuber diameter. Hard tubers were counted and weighed for each graded category while decomposing non-viable tubers were soft, hollow, and were discarded. Approximately thirty randomly sampled hard tubers were selected from each plot and sprouted in 150 by 15 mm petri dishes[33] between moist filter paper in an incubator to determine the effect of field applied herbicide treatments on tuber sprout the following year. The tuber was considered sprouted when a bud protruded from the

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[31]Excel-95, Microsoft Corporation, One Microsoft Way, Redmond, Washington 98052-6399.

[32]Sears, Roebuck, and Co., 3333 Beverly Road, Hoffman Estates, IL 60179.

[33]VWR Scientific Products, 800 E. Fabyan Pkwy., Batavia, IL 60510.

scales of the tuber (Holt and Orcutt 1996). Tubers were evaluated every 7 d for 21 d for sprouted tubers which were counted and removed upon evaluation. Percent sprout was calculated as: sprouted tuber number/30*100.

An analysis of variance was conducted and percent data for visual control were transformed to the arc sine prior to the analysis. The transformation did not affect the conclusions so original data was evaluated. Initial tuber counts were reported as a reference and were not part of the data analysis. Means were separated using Fisher's Protected LSD at $p \leq 0.05$. Linear regression analysis was performed, and lines are presented with symbols representing predicted values and significance at $p \leq 0.01$.

RESULTS

Greenhouse Studies.

Yellow nutsedge growth was suppressed by glyphosate at 840 g ha^{-1} 14 WAT (Table 1). There was no difference in yellow nutsedge height, panicle density, shoot dry weight reduction, tuber number, or tuber weight following the glyphosate treatment compared to the untreated control. Chlorimuron, imazethapyr/imazapyr, and halosulfuron controlled yellow nutsedge 77 to 98% and reduced height, panicle density, dry weight, tuber number, tuber weight and root fresh weight more than glyphosate applied alone. Tank mixtures of glyphosate with these herbicides did not increase yellow nutsedge control.

Yellow nutsedge did not form panicles following treatments of chlorimuron, cloransulam, imazethapyr, imazethapyr/imazapyr, or halosulfuron alone or tank mixed with glyphosate. A tank mixture of rimsulfuron plus glyphosate did not increase visual

control of yellow nutsedge compared with rimsulfuron, but panicle number decreased. Reduced panicle formation may reduce seed production although seeds are not considered a primary mechanism of dispersal and establishment (Thullen and Keeley 1979).

Yellow nutsedge control increased 17 to 38% and tuber density and fresh weight decreased when glyphosate was tank mixed with cloransulam or imazethapyr compared with either treatment or glyphosate applied alone. The reduction in tuber density and fresh weight was greatest by chlorimuron = imazethapyr/imazapyr ≥ halosulfuron ≥ pyrithiobac ≥ imazethapyr = rimsulfuron ≥ cloransulam = glyphosate ≥ imazamox. Our results are contrary to greenhouse research by Ackley et al. (1996) where halosulfuron suppressed yellow nutsedge growth more than chlorimuron. We applied chlorimuron at 12 g ha^{-1} while Ackley et al. (1996) evaluated 9 g ha^{-1} of chlorimuron. Imazamox stimulated the number of tubers produced by yellow nutsedge compared to the untreated control; however, most of these tubers were smaller since total tuber weight was similar to the untreated control.

Field Studies.

Glyphosate provided 53% yellow nutsedge control 56 DAT (Table 2). Chlorimuron and halosulfuron controlled yellow nutsedge 86 and 97%, respectively. Halosulfuron provided 23% greater yellow nutsedge control compared to imazethapyr/imazapyr. Tank mixtures of chlorimuron, halosulfuron, or imazethapyr/imazapyr with glyphosate did not increase yellow nutsedge control, or reduce shoot density, dry weight, or height as compared to the ALS-inhibiting herbicides applied alone. Tank mixtures of glyphosate with cloransulam, imazamox, imazethapyr,

rimsulfuron, or pyrithiobac increased visual yellow nutsedge control by 15 to 30% compared to either herbicide applied alone. Similarly, tank mixtures of glyphosate with cloransulam, imazamox, imazethapyr, or rimsulfuron reduced yellow nutsedge shoot density, dry weight, and height more than the ALS-inhibiting herbicide applied alone. Yellow nutsedge control was greater with imazethapyr/imazapyr compared to imazethapyr, while imazethapyr controlled yellow nutsedge and reduced shoot density, dry weight, and height more than imazamox applied alone. Imazamox stimulated shoot production 180% compared to the untreated control. Halosulfuron and pyrithiobac reduced tuber sprouting by 19% compared to the untreated control. In turfgrass research, halosulfuron had no effect on tuber density or weight, but reduced tuber viability with two applications (Molin et al. 1999). As the rate of halosulfuron increased, tuber viability was reduced (Molin et al. 1999). The reduction in tuber sprouting in this research could be due to the effects of the herbicide (Molin et al. 1999, Reddy and Bendixen 1989) or the presence of dormant tubers with reduced germination (Stoller and Wax 1973, Thullen and Keeley 1975).

The initial tuber density and fresh weight in June prior to herbicide application was 2320 tubers m^{-2} and 328 g m^{-2}, respectively (Figure 1a and 1b). There was no difference in tuber density between ALS-inhibiting herbicide treatments compared to tank mixtures of these herbicides with glyphosate except rimsulfuron plus glyphosate which reduced tuber density more than rimsulfuron applied alone; therefore, only data for glyphosate and the ALS-herbicides applied alone were presented. A 220% increase in tuber density was observed in the untreated control after one year. Halosulfuron caused the greatest

reduction in tuber density and fresh weight of the herbicides evaluated, which was similar to chlorimuron, imazethapyr, and imazethapyr/imazapyr. The tuber density and fresh weight with glyphosate was the closest to the initial tuber density (2320 tubers m^{-2}); therefore, comparisons with glyphosate were pertinent. Chlorimuron and halosulfuron reduced tuber density 64 and 79%, and fresh weight 74 and 71%, respectively, compared to glyphosate applied alone. Tuber density was reduced by all herbicide treatments 33 to 90% except imazamox and rimsulfuron applied alone compared to the untreated control, and tuber fresh weight was reduced 45 to 91% by all treatments compared to the untreated control.

All treatments had small, medium, and large tubers in the plots. Small tubers (157 mg) have a shorter life span compared to large (662 mg) tubers which is important for evaluating the longevity of tubers in the soil (Thullen and Keeley 1975); however, no differences in germination between tubers with different sizes have been reported (Thullen and Keeley 1975, Stoller et al. 1972).

A linear relationship between visual control at 8 WAT and measured tuber density the following year (42 WAT) was observed ($P = 0.0001$) (Figure 2). Tuber density was reduced 625 tubers m^{-2} for every 10% increase in visual control. Yellow nutsedge control by the herbicides evaluated was ranked halosulfuron and chlorimuron (> 80% control and reduction in tuber density); imazethapyr/imazapyr, imazethapyr, and glyphosate (50 to 80% control and reduction in tuber density); and cloransulam, rimsulfuron, and imazamox (20 to 50% control and reduction in tuber density). However, pyrithiobac controlled yellow nutsedge 48% and reduced tuber density 60%.

The response of tuber fresh weight to the herbicide treatments was similar to the tuber density data; therefore, tuber fresh weight and density were correlated to provide researchers with a simple method to estimate tuber density from the tuber fresh weight. For every tuber fresh weight g m^{-2} measured, approximately eight tubers m^{-2} were present (Figure 3). Keeley and Thullen (1983) reported that yellow nutsedge tuber number (y) was directly related to the number of shoots (x) at harvest with the equation $y = 15.68x - 57.1$. This equation overestimated the observed tuber density based on the shoot density 8 WAT in our research.

Yellow Nutsedge Growth.

Yellow nutsedge height for 1997 and 1998 was fit to a second order polynomial curve (Figure 4). Yellow nutsedge was 15 to 20 cm tall by the third week in June which would be approximately 4 to 5 weeks after corn and soybean planting in the Midwest. In glyphosate-resistant corn and soybean, glyphosate is applied to weeds that are 10 and 20 cm tall, respectively, to avoid yield loss (Dalley et al. 1998, Gower et al. 1998, Horak et al. 1998, Levkulich, et al. 1998, Loux et al. 1998). This is usually 4 to 5 weeks after planting for corn and soybean, respectively. The critical period for yellow nutsedge control may be prior to 4 to 5 weeks after planting because of the rapid early growth of this weed.

DISCUSSION

Yellow nutsedge is competitive both above and below ground, and is considered to be a more aggressive perennial weed than purple nutsedge or johnsongrass (Holt and Orcutt 1991). Corn yield was reduced 8% for every 100 shoots of yellow nutsedge m^{-2}

(Stoller et al. 1979) while cotton seed yield was reduced 18 kg ha^{-1} for every shoot measured (Patterson et al. 1980). At least a 2 to 6 week yellow nutsedge-free period during crop establishment is needed to have no effect on cotton yield (Keeley and Thullen 1983).

Yellow nutsedge control in the greenhouse with halosulfuron was less than the control observed in the field. In contrast, control with imazethapyr/imazapyr was greater in the greenhouse than in the field. Differences could be due to ample water supply and herbicide uptake in the greenhouse for the imidazolinone herbicides. Imazamox stimulated the number of tubers produced compared to the untreated control in the greenhouse, but had no effect on tuber density in the field. This may be due to the longer period of growth in the greenhouse compared to the field or plant growth regulation symptoms exhibited by some imidazolinone herbicides (Bhalla and Shehata 1991).

Control of yellow nutsedge was observed when glyphosate was tank mixed with several ALS-inhibiting herbicides. Reduced above ground yellow nutsedge growth is important for limiting yellow nutsedge competition for light and moisture with a crop. However, tuber production did not decrease compared with all of the ALS-inhibiting herbicides except rimsulfuron applied alone. Tank mixing glyphosate with imazethapyr, imazamox, cloransulam, or pyrithiobac would be beneficial to reduce aboveground yellow nutsedge competition with the crop. However, for the long-term management of yellow nutsedge, tank mixtures with glyphosate may not reduce the net tuber density in the soil compared to the ALS-inhibiting herbicide applied alone.

Several cultural methods including crop selection and population, planting date, rotations, cultivation, fertility, and weed-free periods may be adopted to manage yellow nutsedge (Ghafar and Watson 1983a and 1983b, Glaze 1987, Jordan-Molero and Stoller 1978, Keeley 1987). Crop selection and population are key factors in suppressing yellow nutsedge (Neeser et al. 1997, Johnson, III and Mullinix, Jr. 1997, Keeley 1987, Keeley and Thullen 1978, Keeley et al. 1979). Yellow nutsedge compensates for low light by increasing shoot growth and reducing root and tuber production (Keeley and Thullen 1978, Santos et al. 1997). A competitive crop would provide additional shading benefits when combined with herbicide treatments that suppress yellow nutsedge and reduce tuber production (Keeley and Thullen 1978, Lotz et al. 1991, Neeser et al. 1997).

In soybean, chlorimuron at 12 g ha^{-1} may reduce total tuber density and fresh weight m^{-2} and would be the treatment of choice followed by imazethapyr, cloransulam, and imazamox. In glyphosate-resistant soybean, glyphosate could be tank mixed for broad spectrum weed control without reducing yellow nutsedge control. Cloransulam and imazamox suppress yellow nutsedge only, and tuber density was not affected over time.

In corn, halosulfuron at 35 g ha^{-1} would control yellow nutsedge and stop tuber production. Imazethapyr or imazethapyr/imazapyr could be applied to IMI corn. These two treatments reduced tuber density and fresh weight 42 WAT similar to halosulfuron, but above ground control indicated halosulfuron provided more control.

Rimsulfuron was applied at the postemergence rate for weed control in potatoes (Renner and Powell 1998) which was one-half the rate applied by Ackley et al. (1996) and approximately 1.3 times the amount applied in premixtures used for weed control in corn.

Rimsulfuron did not control yellow nutsedge, but glyphosate plus rimsulfuron reduced yellow nutsedge tuber density and fresh weight compared to rimsulfuron applied alone.

Pyrithiobac is labeled for postemergence weed control in cotton. The addition of pyrithiobac at 70 g ha^{-1} to glyphosate at 840 g ha^{-1} reduced tuber fresh weight m^{-2} and improved control compared to glyphosate alone. Yellow nutsedge control was greater with 70 g ha^{-1} compared to 35 g ha^{-1} in previous research (Ackley et al. 1996, Wilcut 1999).

The use of preplant incorporated or preemergence herbicides that have yellow nutsedge control or suppression such as sulfentrazone (Dayan et al. 1996, Wehtje et al. 1997), alachlor (Armstrong et al. 1973, Banks 1983), or metolachlor (Dixon and Stoller 1982, Grichar et al. 1996) followed by postemergence herbicide applications evaluated in this research may increase yellow nutsedge control and reduce yellow nutsedge competitiveness prior to a postemergence herbicide treatment. Sequential applications of glyphosate at 840 g ha^{-1} may improve visual yellow nutsedge control when used in glyphosate-resistant crops (Fischer and Harvey 1998). This research allows recommendations for yellow nutsedge control to be based not only on the vegetative symptoms above ground, but also on tuber development and the asexual reproductive potential of this problematic weed. Increasing our understanding of tuber dynamics will improve our recommendations for long-term yellow nutsedge control.

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Table 1. Yellow nutsedge visual control, height, panicle number, root fresh weight, shoot dry weight reduction, tuber number and weight reduction in the greenhouse 14 weeks after treatment with glyphosate plus AMS, ALS-inhibiting herbicides, and tank mixtures of glyphosate and the ALS-inhibiting herbicides.

Herbicide treatment	Rate	Visual control	Plant height	Panicle number	Root fresh weight	Shoot dry weight[a]	Tuber number[b]	Tuber weight[c]
	g ha^{-1} + %v/v	– % –	– cm –	- no. m^{-2} -	– g m^{-2} –	——— % reduction ———		
Untreated	0	0	43	14	4353	0	0	0
Glyphosate	840	29	41	11	3335	-26	12	21
Chlorimuron + NIS	12 + 0.25	98	2	0	70	84	99	99
Chlorimuron + glyphosate + NIS	12 + 840 + 0.25	99	1	0	35	86	99	100
Cloransulam + NIS	17.5 + 0.25	41	36	4	3757	-10	14	19
Cloransulam + glyphosate + NIS	17.5 + 840 + 0.25	58	33	0	2247	13	44	57
Imazamox + MSO	45 + 1.0	28	40	11	3405	-22	-28	3
Imazamox + glyphosate + MSO	45 + 840 + 1.0	41	38	0	3230	-30	-19	5
Imazethapyr + MSO	70 + 1.0	41	40	4	2177	21	11	40
Imazethapyr + glyphosate + MSO	70 + 840 + 1.0	67	28	0	1475	32	52	72
Imazethapyr/imazapyr + MSO	62 + 1.0	90	13	0	316	70	91	95
Imazethapyr/imazapyr + glyphosate + MSO	62 + 840 + 1.0	96	9	0	105	83	99	100
Halosulfuron + NIS	35 + 0.25	77	25	0	737	54	77	86
Halosulfuron + glyphosate + NIS	35 + 840 + 0.25	76	21	0	913	45	72	80
Rimsulfuron + NIS	17.5 + 0.25	42	35	11	3265	-3	14	30
Rimsulfuron + glyphosate + NIS	17.5 + 840 + 0.25	53	34	0	2703	-10	19	36
Pyrithiobac + COC	70 + 1.3	41	41	4	2493	6	46	58
Pyrithiobac + glyphosate + COC	70 + 840 + 1.3	41	43	14	2001	-1	61	65
$LSD_{(p<0.05)}$		—15—	—9—	—11—	—913—	—26—	—25—	—25—

[a]Shoot dry weight for the untreated control was 260 g m^{-2}.
[b]Tuber number for the untreated control was 7860 m^{-2}.
[c]Tuber fresh weight for the untreated control was 907 g m^{-2}.

Table 2. Yellow nutsedge control 8 weeks after treatment (WAT), shoot density 8 WAT, and dry weight reduction 8 WAT, height reduction 6 WAT, and tuber sprout 42 WAT following treatments with glyphosate plus AMS, ALS-inhibiting herbicides, and tank mixtures of glyphosate and the ALS-inhibiting herbicides in the field in 1997 and 1998[a].

		Visual control		Shoot density		Dry weight reduction[b]		Height reduction[c]		Tuber
		Glyphosate		Glyphosate		Glyphosate		Glyphosate		sprout
Herbicide treatment	Rate	-	+	-	+	-	+	-	+	
	g ha^{-1} + %v/v	%		shoots m^{-2}		%				
Untreated	0	0	53	818	452	0	63	0	46	74
Chlorimuron + NIS	12 + 0.25	86	87	108	97	95	96	77	80	60
Cloransulam + NIS	17.5 + 0.25	50	70	721	323	57	77	47	51	69
Imazamox + MSO	45 + 1.0	39	68	1475	431	34	71	45	56	73
Imazethapyr + MSO	70 + 1.0	58	82	732	323	59	84	54	65	62
Imazethapyr/imazapyr + MSO	62 + 1.0	74	83	635	291	64	77	62	67	66
Halosulfuron + NIS	35 + 0.25	97	95	0	43	100	98	83	78	55
Rimsulfuron + NIS	17.5 + 0.25	45	78	1055	312	44	79	39	65	75
Pyrithiobac + COC	70 + 1.3	48	74	538	323	65	82	48	62	55
LSD $_{(p\leq 0.05)}$		11		377		19		9		16

[a]Glyphosate was applied at 840 g ae ha^{-1} and all treatments included AMS at 20 g L^{-1}.
[b]Dry weight for the untreated control was 470 g m^{-2}.
[c]Height measured 6 wk after treatment. The height for the untreated control height was 52 cm.

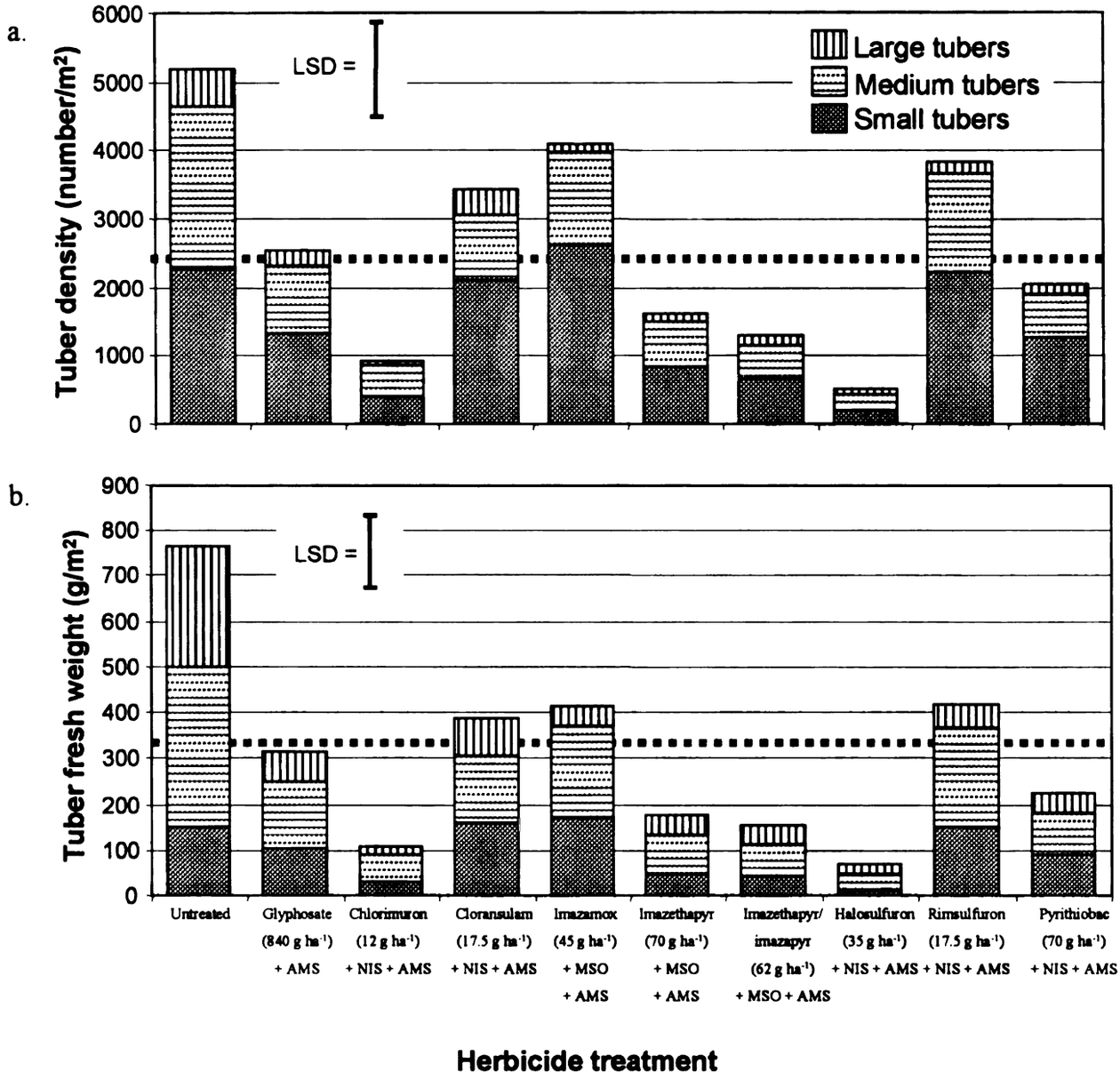

Figure 1. Yellow nutsedge tuber density and fresh weight following glyphosate plus AMS, ALS-inhibiting, and tank mixtures of glyphosate and ALS-inhibiting herbicide treatments in 1997 and 1998. The dotted line indicates the initial tuber density and fresh weight prior to herbicide applications. The solid bars represent small (<5 mm), horizontal lines represent medium (5-8 mm), and vertical lines represent large (>8 mm) tubers in the histogram. The vertical line indicates the LSD at $p \leq 0.05$. Additives included nonionic surfactant (NIS) at 0.25% v/v, crop oil concentrate (COC) at 1.3% v/v, methylated seed oil (MSO) at 1.0% v/v, and ammonium sulfate (AMS) at 20 g L^{-1}.

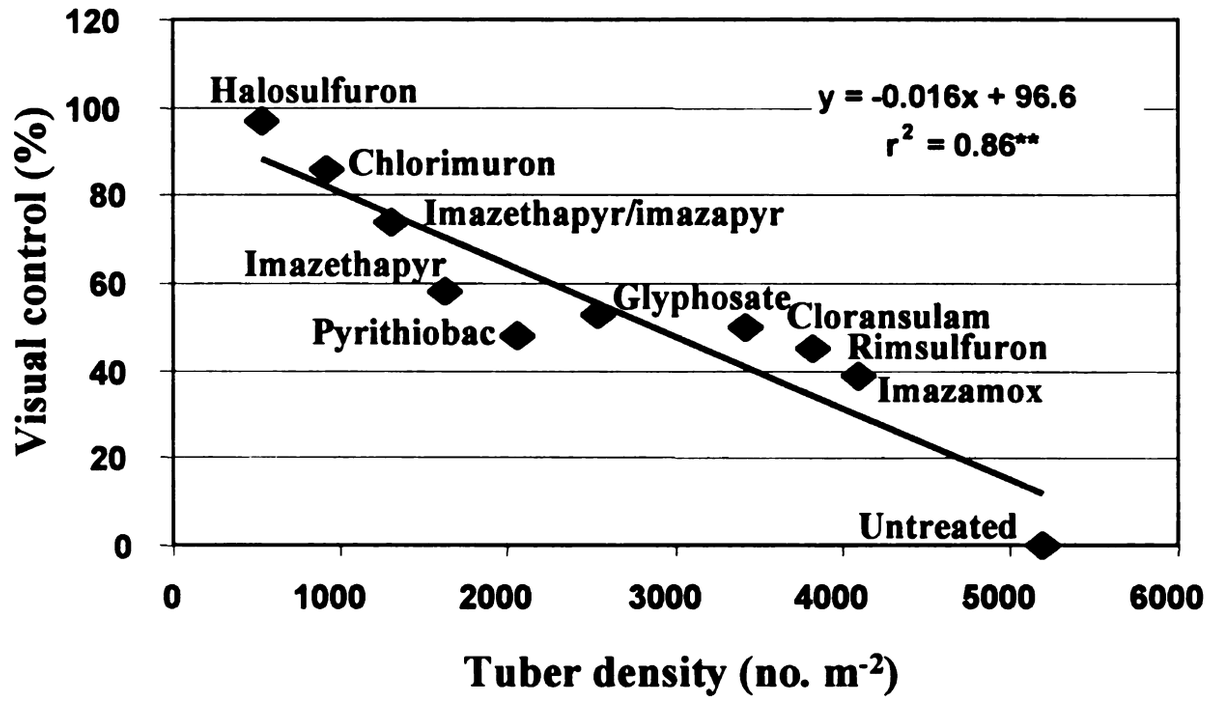

Figure 2. Correlation of yellow nutsedge tuber density (x) in the soil 42 weeks after herbicide treatment at recommended rates with observed visual control (y) of above ground plants averaged over 1997 and 1998.

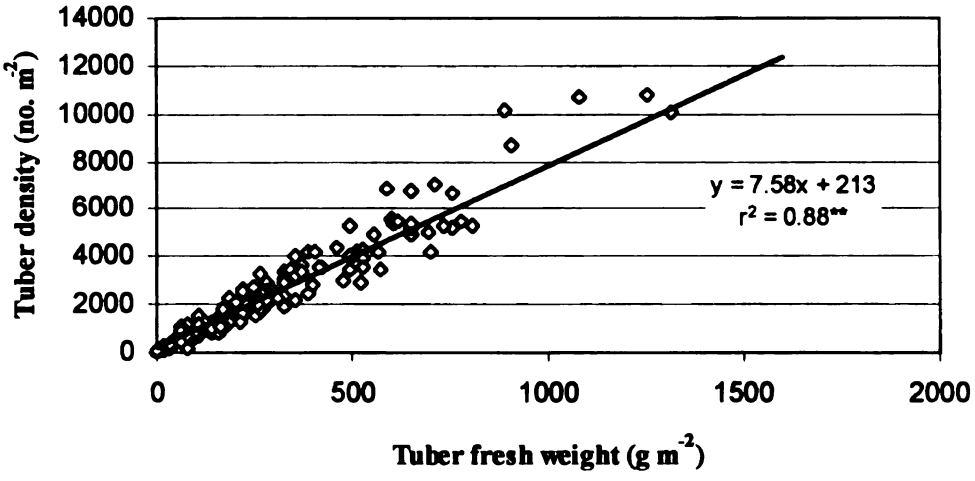

Figure 3. Correlation of yellow nutsedge tuber fresh weight (x) with tuber density (y) averaged over 1997 and 1998.

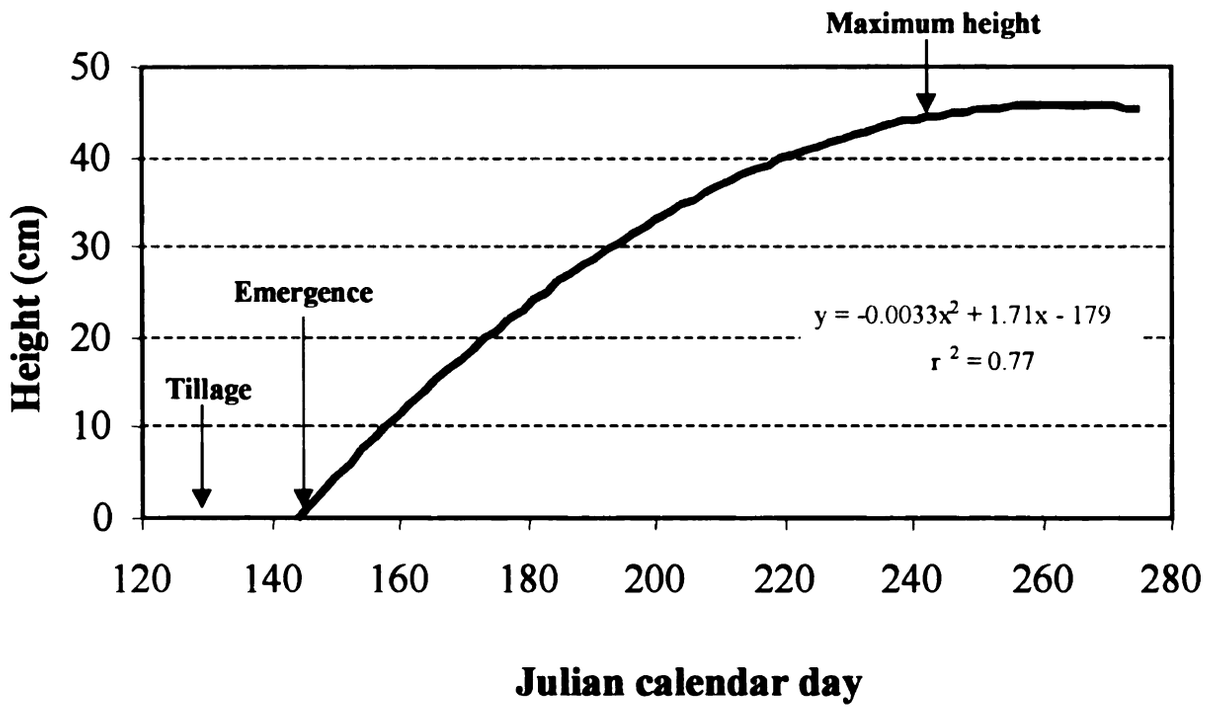

Figure 4. Yellow nutsedge shoot height analysis in the field in 1997 and 1998.

CHAPTER 7

YELLOW NUTSEDGE (*Cyperus esculentus*) CONTROL AND TUBER YIELD WITH GLYPHOSATE AND GLUFOSINATE

Abstract: Greenhouse and field research was conducted to evaluate yellow nutsedge control and the effects on tuber production with glufosinate, glyphosate, and glyphosate plus additional adjuvant. Yellow nutsedge dry weight reduction with glufosinate or glyphosate was not affected by spray volumes ranging from 140 to 1038 L ha^{-1}. The addition of ammonium sulfate (AMS) to glyphosate and glufosinate reduced yellow nutsedge dry weight 19% more than the herbicides alone. Glyphosate at 0.51 kg ae ha^{-1} and glufosinate at 0.59 kg ae ha^{-1} reduced yellow nutsedge growth by 50% in the greenhouse. Glyphosate at 0.84 kg ha^{-1} controlled yellow nutsedge 29 to 53% and glufosinate at 0.4 kg ha^{-1} controlled yellow nutsedge 16 to 19% in the field and greenhouse. Glufosinate at 0.4 kg ha^{-1} reduced tuber density and fresh weight in the greenhouse compared to untreated plants. Glyphosate at 0.84 kg ha^{-1} reduced yellow nutsedge tuber density and sprouting in the field and tuber fresh weight in the field and greenhouse compared to untreated plants. The addition of a nonionic surfactant, methylated seed oil, or crop oil concentrate to glyphosate plus AMS did not increase the effectiveness of glyphosate in the greenhouse or field.

Nomenclature: glufosinate, 2-amino-4-(hydroxymethylphosphinyl)butanoic acid; glyphosate, *N*-(phosphonomethyl)glycine; yellow nutsedge, *Cyperus esculentus* L. #[34] CYPES.

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[34]Letters following this symbol are a WSSA-approved computer code from Composite List of Weeds, Revised 1989. Available from WSSA, 1508 West University Ave., Champaign, IL 61821-3133.

Additional index words: ammonium sulfate, crop oil concentrate, dose-response, methylated seed oil, nonionic surfactant, perennial weed, spray volume, and tuber production.

Abbreviations: AMS, ammonium sulfate ($(NH_4)_2SO_4$); COC, crop oil concentrate; DAT, d after treatment; GR_{50}, rate causing 50% growth reduction; and NIS, nonionic surfactant.

INTRODUCTION

Yellow nutsedge is a problematic perennial weed found throughout the world in crop production fields (Bendixen and Nandihalli 1987, Lapham 1985, Schippers et al. 1995). Yellow nutsedge produces tubers at the terminal end of the rhizome which are primarily responsible for the propagation and spread of this weed (Bendixen 1973, Wills et al. 1980). The introduction of glyphosate- and glufosinate-resistant crop varieties has provided producers with opportunities for non-selective weed control and additional options for perennial weed management. Glyphosate has been evaluated as a non-selective herbicide for control of numerous perennial weeds including foxtail barley (*Hordeum jubatum* L.) (Donald 1988), bermudagrass [*Cynodon dactylon* (L.) Pers.] (Jordan 1977), johnsongrass [*Sorghum halepense* (L.) Pers.] (McKinley et al. 1999, Salisbury et al. 1991), hemp dogbane (*Apocynum cannabinum* L.) (Schultz and Burnside 1980, Wyrill, III and Burnside 1977), Canada thistle [*Cirsium arvense* (L.) Scop.] (Darwent et al. 1994), quackgrass [*Elytrigia repens* (L.) Nevski] (Devine et al. 1983, Claus and Behrens 1976), purple nutsedge (*Cyperus rotundus* L.) (Bariuan et al. 1999, Villaneuva et al. 1985, Zandstra et al. 1974, Zandstra and Nishimoto 1977), and yellow nutsedge (*Cyperus esculentus* L.) (Appleby and Paller 1978, Periera and Crabtree 1986, Stoller et al. 1975).

Yellow nutsedge, however, is difficult to control with glyphosate. The glyphosate application rate (Appleby and Paller 1978, Pereira and Crabtree 1986, Stoller et al. 1975, Villaneuva et al. 1985) and plant age at the time of application (Appleby and Paller 1978, Keeley et al. 1985, Stoller et al. 1975) has influenced yellow nutsedge control. Regrowth of yellow nutsedge following an application of glyphosate at 1.1 to 2.2 kg ha^{-1} (Pereiera and Crabtree 1986), and conversely, control from glyphosate at 1.1 kg ha^{-1} or more (Derr and Wilcult 1993, Stoller et al. 1975) have been reported. Glyphosate reduced tuber production (Pereira and Crabtree 1986), germination (Stoller et al. 1975), and fresh weight (Pereira and Crabtree 1986), and increased occurrence of nonviable tubers (Appleby and Paller 1978). In a fallow cropping system, glyphosate at 2.24 kg ha^{-1} did not reduce shoot densities during the fallow period, but shoot number decreased by 95% the following year (Keeley et al. 1979).

Application variables such as spray volume (Buhler and Burnside 1983, Buhler and Burnside 1987, Jordan 1977, Krausz et al. 1996, Stahlman and Phillips 1979), the addition of ammonium sulfate (Bruce and Kells 1990, Donald 1988, Jordan et al. 1997, Salisbury et al. 1991), and the use of additional adjuvant (Buhler and Burnside 1983, Wyrill, III and Burnside 1977) increased weed control with glyphosate. However, none of these factors have been investigated to determine their influence on yellow nutsedge control with glyphosate. Additional adjuvant is known to increase herbicide absorption (Thompson et al. 1996, Young and Hart 1998) and weed control with other herbicides under water-stressed conditions (Levene and Owen 1995, Wanamarta and Penner 1989). The cuticle is the major limiting barrier for herbicide absorption under such conditions (Hess 1985), and

yellow nutsedge has a thick waxy cuticle on the adaxial leaf surface (Wills et al. 1980). Since adjuvants increase efficacy under hot, dry conditions then additional adjuvant may help increase yellow nutsedge control with glyphosate. In addition, several premixtures of glyphosate and other herbicides are currently marketed. Additional adjuvant for a second herbicide may be necessary when glyphosate is tank mixed with other herbicides to increase control of weeds like yellow nutsedge. However, additional surfactants may also reduce the uptake of glyphosate in grass species (Gaskin and Stevens 1993). Therefore, the effect of additional adjuvant on yellow nutsedge control with glyphosate needs to be investigated.

Research with glufosinate has focused on annual weed control (Higgins et al. 1991, Krausz et al. 1999, Lanie et al. 1994, Steckel et al. 1997a, Steckel et al 1997b, Tharp et al. 1999, Wilson et al. 1985). Several studies have compared annual weed control between glufosinate and glyphosate to determine efficacy differences (Blackshaw 1989, Bruce and Kells 1990, Lanie et al. 1994, Higgins et al. 1991, Tharp et al. 1999, Wilson et al. 1985). The relative sensitivity of barnyardgrass [*Echinochloa crus-galli* (L.) Beauv.], common ragweed (*Ambrosia artemisiifolia* L.), fall panicum (*Panicum dichotomiflorum* Michx.), giant foxtail (*Setaria faberi* Herrm.), and large crabgrass [*Digitaria sanguinalis* (L.) Scop.] (Tharp et al. 1999) to glyphosate and glufosinate was similar. However, research on perennial weed control with glufosinate is limited (Donn 1982), and no research has compared perennial weed control with glyphosate and glufosinate or the effect of glufosinate on yellow nutsedge tuber production.

The objectives of this research were to determine: 1) the effect of spray volume and AMS on yellow nutsedge control with glyphosate and glufosinate, 2) the effect of glyphosate and glufosinate on yellow nutsedge control and tuber yield, 3) if additional adjuvant with glyphosate plus AMS affected yellow nutsedge control, and 4) the sensitivity of yellow nutsedge to glyphosate and glufosinate.

MATERIALS AND METHODS

Greenhouse Methods. Locally collected tubers were planted at in 910 ml plastic pots in BACCTO potting soil[35]. A single tuber was planted in each pot and plants were fertilized with 0.1 g of water soluble fertilizer (20% N, 20% P_2O_2, 20% K_2O) in 50 ml of water every two weeks and surface irrigated as needed. Plants were grown in a greenhouse with a 16-h photoperiod of natural and supplemental sodium vapor lighting which provided a photosynthetic photon flux density[36] of 120 $\mu E\ m^{-2} \cdot s^{-1}$. These cultural methods were similar for all research unless described otherwise.

Effect of Spray Volume on Yellow Nutsedge Control with Glyphosate and Glufosinate with and without AMS. Glyphosate (formulated as Roundup Ultra®) at 0.84 kg ha^{-1} and glufosinate at 0.4 kg ha^{-1} were applied at four spray volumes in the absence and presence of AMS at 20 g L^{-1} to yellow nutsedge 15 cm in height with six leaves. Herbicides were applied at 1230 h with a traveling-belt sprayer traveling at 1.5 km h^{-1} with 152 kPa. Spray volume was changed using 8001, 8002, 8004, and 8008 flat-fan nozzle spray nozzles at the same pressure to attain an output of 140, 271, 542, and 1038 L ha^{-1}, respectively.

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[35]BACCTO is a product of Michigan Peat Co. Houston, TX 77098.

[36]LI-COR. 4421 Superior Street, Lincoln, NE 68504.

Aboveground yellow nutsedge shoot tissue was harvested 28 d after treatment (DAT), oven-dried, and the percent dry weight reduction compared to the non-treated control was calculated. The experiment was analyzed as a three factor randomized complete block design with four replications repeated twice. The factors were herbicide treatment (glyphosate and glufosinate), the presence or absence of AMS, and spray volume. Data were subjected to an analysis of variance, means separated using Fisher's Protected LSD at $p \leq 0.05$, and main effects for each factor were presented since no interactions were observed.

Herbicide Injection Experiment. Yellow nutsedge plants were grown in potting soil as previously described. Herbicides were injected into plants that were 15 cm tall with six leaves with a syringe[37]. A volume of 10 µl of solution injected above the basal bulb was equivalent to the postemergence application rate previously described. These treatments were injected above the basal bulb to overcome any effects of the cuticle on absorption. Untreated plants injected with water were included as controls. This study was arranged as a randomized complete block design with four replications and was repeated twice. Height and visual control 28 DAT was recorded and analyzed using an analysis of variance and means were separated using Fisher's Protected LSD ($p \leq 0.05$).

Effect of Glufosinate, Glyphosate, and Additional Adjuvant with Glyphosate on Yellow Nutsedge Control, Tuber Production, and Tuber Sprout. Yellow nutsedge tubers were sprouted in a Freas 815 Incubator[38] between two moist filter papers for three d at 31 C

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[37]Hamilton Company, P.O. Box 10030, Reno, Nevada, 89520-0012.

[38]Precision Scientific Co., 3737 West Cortland St., Chicago, IL 60647.

prior to planting. Two moderately sized (5 to 8 mm) sprouted tubers were transferred to four-L pots of Spinks loamy sand (sand, mixed, mesic Psammentic Hapludalfs). Postemergence herbicide treatments included glufosinate at 0.4 kg ha^{-1} plus AMS at 20 g L^{-1} and glyphosate (formulated as Roundup Ultra) at 0.84 kg ha^{-1} plus AMS at 20 g L^{-1} applied alone and with nonionic surfactant[39] (NIS) at 0.25% v/v, crop oil concentrate[40] (COC) at 1.3% v/v, methylated seed oil[41] (MSO) at 1.0% v/v. Yellow nutsedge was 15 cm tall with six leaves at the time of application. Herbicides were applied at 1800 h with a traveling-belt sprayer traveling at 1.5 km h^{-1}, and delivering 234 L ha^{-1} at 193 kPa, and equipped with an 8001 even flat-fan nozzle. Plants were visually rated on a scale of 0 (no visual injury or plant suppression) to 100% (complete plant death) control four and 14 WAT. Plants were harvested after the untreated control had flowered. Plant height, aboveground dry weight, root fresh weight, tuber density and tuber fresh weight were measured 14 WAT. Percent dry weight reduction was calculated as 100*[1-(treated plant dry weight/untreated plant dry weight)]. This research was arranged as a randomized complete block design with four replications and was repeated twice. Data were subjected to an analysis of variance and means were separated using Fisher's Protected LSD ($p \leq 0.05$).

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[39]Nonionic surfactant was Activator-90, a mixture of alkyl polyoxyethylene ether and free fatty acids, Loveland Industries Inc., P.O. Box 1289, Greeley, CO 80632.

[40]Crop oil concentrate was Herbimax, 83% petroleum oil and 17% surfactant, Loveland Industries Inc., P.O. Box 1289, Greeley, CO 80632.

[41]Methylated seed oil was SunIt-II, methylated seed oil and emulsifiers, Agsco, Inc., P.O. Box 13458, Grand Forks, ND 58208-3458.

Dose-Response of Glyphosate and Glufosinate. A dose-response comparison of glyphosate and glufosinate applied at 0, 0.02, 0.08, 0.35, 1.4, 5.6, and 22.4 kg ae ha^{-1} was arranged in a completely randomized design with five replications and was repeated three times. Glyphosate was formulated as Roundup Ultra®[42]. All treatments included 20 g L^{-1} spray-grade ammonium sulfate. Herbicides were applied at 1200 h with a traveling-belt sprayer traveling at 1.5 km h^{-1}, delivering 234 L ha^{-1} at 193 kPa, and equipped with an 8001[43] even flat-fan nozzle. Yellow nutsedge was 15 cm tall with six leaves at the time of application. Aboveground plant shoot tissue was harvested 28 DAT, oven-dried, and the dry weight reduction as a percent of the untreated control was calculated. Data were subjected to a nonlinear regression analysis using a log-logistic dose-response model as previously described by Schabenberger et al. (1999) and Tharp et al. (1999).

Field Methods. Research was conducted in 1997 near Alto, MI and 1998 at East Lansing, MI in fields with a previous history of heavy yellow nutsedge infestation. The soil was a Boyer sandy loam (coarse-loamy, mixed, mesic Typic Hapludalf) with 2.9% organic matter and pH 6.6 in 1997 and a Marlette sandy loam (fine-loamy, mixed mesic Glossoboric Hapludalf) with 1.7% organic matter and pH 6.7 in 1998. The fields were cultimulched eight cm deep on May 27, 1997 and soil-finished six cm deep on May 13, 1998.

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[42]Roundup Ultra®, isopropylamine salt of glyphosate with surfactant. Monsanto Agricultural Company, St. Louis, MO 63167.

[43]Teejet flat-fan tips. Spraying Systems Co., North Avenue and Schmale Road, Wheaton, IL 60188.

Herbicide treatments included glyphosate (formulated as Roundup Ultra) at 0.84 kg ha^{-1} plus AMS at 20 g L^{-1} applied alone and with NIS at 0.25% v/v, COC at 1.3% v/v, MSO at 1.0% v/v. Glufosinate was also applied at 0.4 kg ha^{-1} with AMS at 20 g L^{-1}. Yellow nutsedge plants were 10 to 20 cm tall with four to eight leaves at the time of application in 1997 and 1998. Herbicides were applied on June 26, 1997 and June 23, 1998 with a tractor-mounted compressed-air sprayer traveling at 6.3 km h^{-1}, delivering 178 L ha^{-1} at 207 kPa, and equipped with 8003 flat-fan nozzles. In 1997, treatments were applied at 2100 h, with an air temperature of 25 C and 37% relative humidity. The treatments were applied at 1930 h, with an air temperature of 33 C and 30% relative humidity in 1998.

Visual weed control was evaluated at 2, 4, and 8 WAT, heights were measured at 2, 4 and 6 WAT, and two 30 by 30 cm quadrats were harvested 8 WAT from each plot and oven-dried. Initial tuber densities were measured from each replication in the field prior to herbicide application. Initial tuber density averaged over both years was 2320 tubers m^{-2} with a fresh weight of 328 g m^{-2}. Eight soil cores were randomly sampled from each plot in April (42 WAT) of the following year with a 10 cm diameter golf cup cutter to a 20 cm depth. Tubers were separated from the soil with a custom-modified Kenmore 80[44] clothes washer basin. Tubers were counted and weighed for each treatment. Thirty hard tubers were randomly sampled from each treatment. The tubers were arranged between two pieces of moist filter paper and were placed in 150 by 15 mm petri dishes[45]

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[44]Sears, Roebuck, and Co., 3333 Beverly Road, Hoffman Estates, IL 60179.

[45]VWR Scientific Products, 800 E. Fabyan Pkwy., Batavia, IL 60510.

which were transferred to an incubator set at 30 C to determine the effect of herbicide treatments on tuber sprouting the following year. Tubers were evaluated for sprouting weekly up to 21 d after placement in the incubator. Sprouted tubers were counted and removed upon evaluation. A tuber was considered sprouted when a bud protruded from the scales of the tuber (Holt and Orcutt 1996).

The experiment was arranged as a randomized complete block design with four replications each year in plots 3 by 6.1 m. Data were subjected to an analysis of variance and percent visual control data were transformed to the arcsine. Field and greenhouse visual percent data were transformed to the arcsine square root; however, transformation did not affect data interpretation and untransformed data are presented. Visual control data were subjected to an *F Max* test for homogeneity (Kuehl 1994) and data were combined over location since variances for both locations were homogenous. Means were separated using Fisher's Protected LSD at $p \leq 0.05$.

RESULTS AND DISCUSSION

Effect of Spray Volume and AMS on Yellow Nutsedge Control with Glyphosate and Glufosinate. Glyphosate at 0.84 kg ha^{-1} reduced yellow nutsedge dry weight 64% whereas glufosinate at 0.4 kg ha^{-1} reduced dry weight only 22% when averaged over AMS and spray volume treatments (Table 1). The addition of AMS to the herbicide containing spray solution reduced yellow nutsedge dry weight an additional 19% compared with no AMS. Increased control of other weed species including prickly sida (*Sida spinosa* L.) (Jordan et al. 1997), entireleaf morningglory (*Ipomoea hederacea* var. *intergriuscula*) (Jordan et al. 1997), foxtail barley (Donald 1988), johnsongrass (Salisbury et al. 1991),

and purple nutsedge (Suwunnamek and Parker 1975) was reported when AMS was added to glyphosate. Additionally, there was three times less regrowth of three and six week old purple nutsedge treated with glyphosate plus AMS compared with no AMS (Suwunnamek and Parker 1975). In contrast, yellow nutsedge control did not increase when AMS was added to glyphosate in glyphosate-resistant corn research (Fischer and Harvey 1998). However, AMS increased annual weed control with glufosinate applied at 0.125 to 0.25 kg ha^{-1} (Blackshaw 1989). The increased control of yellow nutsedge with AMS compared to no AMS in our research illustrates the importance of this spray additive to increase yellow nutsedge suppression with either glyphosate or glufosinate.

Spray volume did not affect yellow nutsedge dry weight reduction (Table 1). Bruce and Kells (1990) similarly reported that spray volume did not influence horseweed [*Conyza canadensis* (L.) Cronq.] control. In contrast, others have shown that spray volume affects control of annual weeds (Buhler and Burnside 1987, Krausz et al. 1996). An interaction between herbicide and spray volume was expected in our research since lower spray volumes increased control of annual grasses with glyphosate in other research (Buhler and Burnside 1987) and higher spray volumes may increase coverage and control with glufosinate since it is a contact herbicide. However, the leaf morphology of yellow nutsedge may allow the applied herbicide spray solution to accumulate in the whorl of the plants when applied as spray volume increases so that there is little affect of spray volume on dry weight reduction.

Herbicide Injection Experiment. A thick waxy cuticle (1 to 1.3 μm) covers a layer of epidermal cells on the adaxial leaf surface of yellow nutsedge (Wills et al. 1980).

Glyphosate and glufosinate were injected into yellow nutsedge plants above the basal bulb to reduce the potential effects of the cuticle on absorption. Glufosinate and glyphosate decreased yellow nutsedge height similarly 28 DAT (Table 2). Glyphosate at 0.84 kg ha^{-1} controlled yellow nutsedge 88% while glufosinate at 0.4 kg ha^{-1} provided 68% control. The increased control with this technique supports our hypothesis that reduced absorption across the cuticle may be a major limitation for yellow nutsedge control with glufosinate. Common lambsquarters was the most tolerant weed species to glufosinate in the field (Steckel et al. 1997b) and greenhouse (Steckel et al. 1997a), and had the lowest absorption compared to giant foxtail, barnyardgrass, and velvetleaf (Steckel et al. 1997a).

Effect of Glyphosate and Glufosinate on Yellow Nutsedge Control, Tuber Production, and Sprouting. Glyphosate at 0.84 kg ha^{-1} provided greater suppression of yellow nutsedge compared to glufosinate at 0.4 kg ha^{-1}, but neither herbicide controlled yellow nutsedge greater than 53% in the greenhouse or field (Table 3). Glyphosate control of yellow nutsedge was 13 to 34% greater than glufosinate in the greenhouse and field. Glufosinate reduced tuber density 19%, and glufosinate and glyphosate reduced tuber fresh weight 22 and 24%, respectively, compared to untreated plants in the greenhouse. Glyphosate controlled yellow nutsedge 53% 8 WAT which resulted in a 51% reduction in tuber density, 59% reduction in tuber fresh weight, and a 17% reduction in tuber sprouting 42 WAT compared to untreated plants (Tables 3). Tubers that did not sprout remained hard and turgid (data not presented). Other researchers reported a 43 to 45% reduction in shoot fresh weight 10 DAT and a 27 to 59% reduction in parent tuber sprouting from a postemergence application of glyphosate at 0.6 kg ha^{-1} to 2 to 4 week

old plants (Keeley et al. 1985). Similarly, the tubers that did not sprout remained firm (Keeley et al. 1985). A reduction in purple nutsedge viability has also been reported with glyphosate (Zandstra et al. 1974). However, glyphosate at 1.1 and 2.2 kg ha^{-1} did not affect parent tuber viability in other research (Stoller et al. 1975). The translocation of glyphosate to yellow nutsedge tubers (Keeley et al. 1985) may explain a reduction in tuber sprout reported in this research. Glufosinate controlled yellow nutsedge 19% in the field and had no effect on tuber density, fresh weight, or tuber sprouting (Table 3) 42 WAT compared to untreated plants.

Effect of Additional Adjuvant with Glyphosate on Yellow Nutsedge Control, Tuber Production, and Tuber Sprout. There was no effect of additional adjuvant on yellow nutsedge control, height, dry weight, tuber density or fresh weight in the greenhouse or field (Table 4). Yellow nutsedge leaves appeared bleached and chlorotic at the base of the leaves one week after glyphosate was applied, and new sprouts emerging from the basal bulb and rhizomes allowed the propagation of this weed in the greenhouse and field. Buhler and Burnside (1987) increased annual weed control at 190 L ha^{-1} with additional adjuvant compared to lower spray volumes; therefore, an increase in control may be expected at higher application volumes as used in our research. Similarly, Bariuan et al. (1999) found that organosilicone surfactants did not affect purple nutsedge control with glyphosate formulated as Roundup Ultra®. Adding an organosilicone surfactant to glyphosate did not affect absorption or translocation of ^{14}C-glyphosate by purple nutsedge (Bariuan et al. 1999).

All glyphosate treatments had similar tuber sprouting (Table 4) and rotten tubers present (data not presented), but glyphosate plus AMS had more hard, turgid tubers than the treatments that included additional adjuvant (data not presented). Glyphosate may act as a sprout inhibitor since 0.3 to 0.6 kg ha^{-1} increased the number of dormant tubers produced in greenhouse research (Appleby and Paller 1978) and glyphosate was translocated to parent tubers in other research (Keeley et al. 1985). The adjuvants evaluated (Table 4) did not increase or decrease herbicide activity of glyphosate. These adjuvants may not have been the appropriate adjuvants for use with glyphosate, but if used with glyphosate in various tank mixtures these adjuvants will not have a negative effect on yellow nutsedge control.

Dose-Response of Glyphosate and Glufosinate. Yellow nutsedge dry weight decreased as the glyphosate and glufosinate rate increased (Figure 1). GR_{50} values of 0.51 and 0.59 kg ha^{-1} for glyphosate and glufosinate, respectively, were statistically similar between these non-selective herbicides. Appleby and Paller (1978) reported 52, 97, and 100% yellow nutsedge control from glyphosate at 0.6, 1.1, and 2.2 kg ha^{-1}, respectively. Research with annual weeds has indicated GR_{50} values from 0.063 to 0.235 kg ha^{-1} for glufosinate (Steckel et al. 1997, Tharp et al. 1999) and 0.064 to 0.16 kg ha^{-1} for glyphosate (Tharp et al. 1999). The dose-response curve indicated glufosinate had a narrow rate range (0.27 to 1.05 kg ha^{-1}) between 90% and 10% of the control while glyphosate had a broad rate range (0.09 to 2.10 kg ha^{-1}) (Figure 1). Our GR_{50} values for yellow nutsedge were equal to 61% of the glyphosate (0.84 kg ha^{-1}) (Jordan et al. 1997) and 148% of the glufosinate (0.4 kg ha^{-1}) (Steckel et al 1997b) rate applied for annual weed control in glyphosate- or

glufosinate-resistant crops. Therefore, yellow nutsedge control in the field would be greater with glyphosate than glufosinate at rates applied for annual weed control which confirmed previously conducted field and greenhouse research.

Herbicide rate and growth stage affect annual weed control with glufosinate (Steckel et al. 1997a and 1997b, Tharp et al. 1999) and yellow nutsedge control with glyphosate (Jordan et al. 1997, Pereira and Crabtree 1986, Stoller et al. 1975). Our research only evaluated control of yellow nutsedge at one growth stage. Yellow nutsedge grows rapidly early in the season (Holt and Orcutt 1996) and control by glufosinate and glyphosate may differ at other growth stages.

In conclusion, neither glufosinate at 0.4 kg ha^{-1} nor glyphosate at 0.84 kg ha^{-1} controlled yellow nutsedge greater than 65%. Similarly, a single application of glyphosate at 0.43, 0.63 or 0.84 kg ha^{-1} in glyphosate-resistant corn did not control yellow nutsedge (Fischer and Harvey 1998). Glyphosate provided greater aboveground yellow nutsedge control compared to glufosinate, but treatments had similar tuber production. Dose-response data (Tharp et al. 1999) and annual weed control comparisons between glyphosate and glufosinate (Higgins et al. 1991, Lanie et al. 1994) indicate higher rates of glufosinate may provide more consistent control of annual weeds. An increase in glufosinate rate may increase yellow nutsedge control. The addition of AMS increased yellow nutsedge control with glyphosate and glufosinate, but spray volume with either herbicide or additional adjuvant tested with glyphosate did not affect yellow nutsedge control. Split applications of these herbicides (Fischer and Harvey 1998, Krausz et al. 1999), tank mixture combinations with other postemergence herbicides that control yellow

nutsedge (Ackley et al. 1996, Derr et al. 1996, Derr and Wilcut 1993, Reddy and Bendixen 1988), or the use of preplant incorporated or preemergence herbicides (Armstrong et al. 1973, Dixon and Stoller 1982, Grichar et al. 1996, Reddy and Bendixen 1989, Wehtje et al. 1997) for yellow nutsedge suppression or control are options that will need to be utilized to increase yellow nutsedge control and reduce tuber production in glyphosate or glufosinate-resistant crops. In addition, crop canopy closure may increase yellow nutsedge suppression by competing for light and other resources and altering yellow nutsedge growth. Yellow nutsedge compensates for low light levels by increasing shoot growth and reducing root and tuber production (Jordan-Molero and Stoller 1978, Keeley and Thullen 1978, Lotz et al. 1991, Santos et al. 1997) which reduces the propagative and reproductive potential of this perennial weed. Yellow nutsedge must be managed in glyphosate- and glufosinate-resistant crops by practices other than a single application of these herbicides or this weed species could become highly problematic in these weed management systems.

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Table 1. The effect of ammonium sulfate and spray volume on yellow nutsedge dry weight reduction with glyphosate and glufosinate 28 d after treatment.

	Dry weight reduction
Herbicide treatment	— % —
Glufosinate 0.4 kg ae ha^{-1}	22
Glyphosate 0.84 kg ae ha^{-1}	64
LSD $_{(p\leq0.05)}$	— 10 —
Ammonium sulfate	
0 g L^{-1}	34
20 g L^{-1}	53
LSD $_{(p\leq0.05)}$	— 10 —
Spray volume	
140 L ha^{-1}	41
271 L ha^{-1}	48
542 L ha^{-1}	40
1038 L ha^{-1}	46
LSD $_{(p\leq0.05)}$	— NS —

Table 2. Height and control of yellow nutsedge by glyphosate and glufosinate injected into plants grown in the greenhouse 28 d after treatment.

Treatment	Rate	Height	Control
	kg ae ha^{-1}	cm	%
Glyphosate	0.84	10	88
Glufosinate	0.4	10	68
Untreated	0	25	0
$LSD_{(p\leq 0.05)}$		3	8

Table 3. Yellow nutsedge control, tuber number, fresh weight, and sprouting following glyphosate and glufosinate in the greenhouse and field in 1997 and 1998.

Herbicide treatment			Tuber		
Greenhouse Research[a]	Rate	Control	Shoot density	Fresh weight	Sprout
	kg ae ha^{-1}	– % –	– no. m^{-2} –	— g m^{-2} —	– % –
Glyphosate + AMS[b]	0.84 + 20	29	6990	700	—
Glufosinate + AMS	0.4 + 20	16	6320	680	—
Untreated	0	0	7830	900	—
LSD $_{(p\leq 0.05)}$		— 8 —	— 980 —	— 150 —	—
Field Research[c]	Rate	Control	Shoot density	Fresh weight	Sprout[d]
	kg ae ha^{-1}	– % –	– no. m^{-2} –	— g m^{-2} —	– % –
Glyphosate + AMS	0.84 + 20	53	2540	320	65
Glufosinate + AMS	0.4 + 20	19	3450	430	83
Untreated	0	0	5190	770	82
LSD $_{(p\leq 0.05)}$		— 3 —	— 2180 —	— 370 —	— 14 —

[a]Greenhouse research evaluated control, tuber density, and tuber fresh weight 14 WAT and were repeated in time. AMS was applied at 20 g L^{-1}.
[b]Abbreviations: AMS, ammonium sulfate; WAT, weeks after treatment.
[c]Field research evaluated control 8 WAT, tuber density, and tuber fresh weight 42 WAT.
[d]Data were the percentage of thirty tubers per plot that were hard, rotten, or sprouted upon visually examination for three weeks after placement in an incubator at 30 C. Tubers were randomly selected from soil cores sampled to a 20 cm depth 42 WAT.

Table 4. Yellow nutsedge control, height, dry weight, tuber density, tuber fresh weight, and tuber sprouting with glyphosate plus AMS alone and with NIS, COC, and MSO in the greenhouse and field in 1997 and 1998.

					Tuber		
Herbicide treatment[a]	Adjuvant rate	Control	Height	Dry weight	Shoot density	Fresh weight	Sprout
Greenhouse Research[b]	% v/v	— % —	– cm –	–g m^{-2} –	no. m^{-2}	–g m^{-2} –	– % –
Glyphosate + AMS[c]	0	29	41	312	6985	695	—
Glyphosate + AMS + NIS	0.25	42	35	242	5827	530	—
Glyphosate + AMS + COC	1.3	32	39	316	7863	772	—
Glyphosate + AMS + MSO	1.0	35	39	295	7582	786	—
LSD $_{(p\leq 0.05)}$		– NS –	– NS –	– NS –	– NS –	– NS –	—
Field Research[d]	% v/v	— % —	– cm –	–g m^{-2} –	no. m^{-2}	–g m^{-2} –	– % –
Glyphosate + AMS	0	53	28	387	2545	314	65
Glyphosate + AMS + NIS	0.25	58	26	323	3047	331	81
Glyphosate + AMS + COC	1.3	56	27	258	2474	306	76
Glyphosate + AMS + MSO	1.0	57	28	301	2192	264	74
LSD $_{(p\leq 0.05)}$		– NS –	– NS –	– NS –	– NS –	– NS –	– NS –

[a]All glyphosate treatments were applied at 0.84 kg ae ha^{-1} with AMS at 20 g L^{-1}.
[b]Greenhouse research evaluated control, height, dry weight, tuber density, and tuber fresh weight 14 WAT and were repeated in time.
[c]Abbreviations: AMS, ammonium sulfate; NIS, nonionic surfactant; COC, crop oil concentrate; MSO, methylated seed oil; and WAT, weeks after treatment.
[d]Field research evaluated control 8 WAT, height 6 WAT, dry weight 8 WAT, tuber density, and tuber fresh weight 42 WAT.

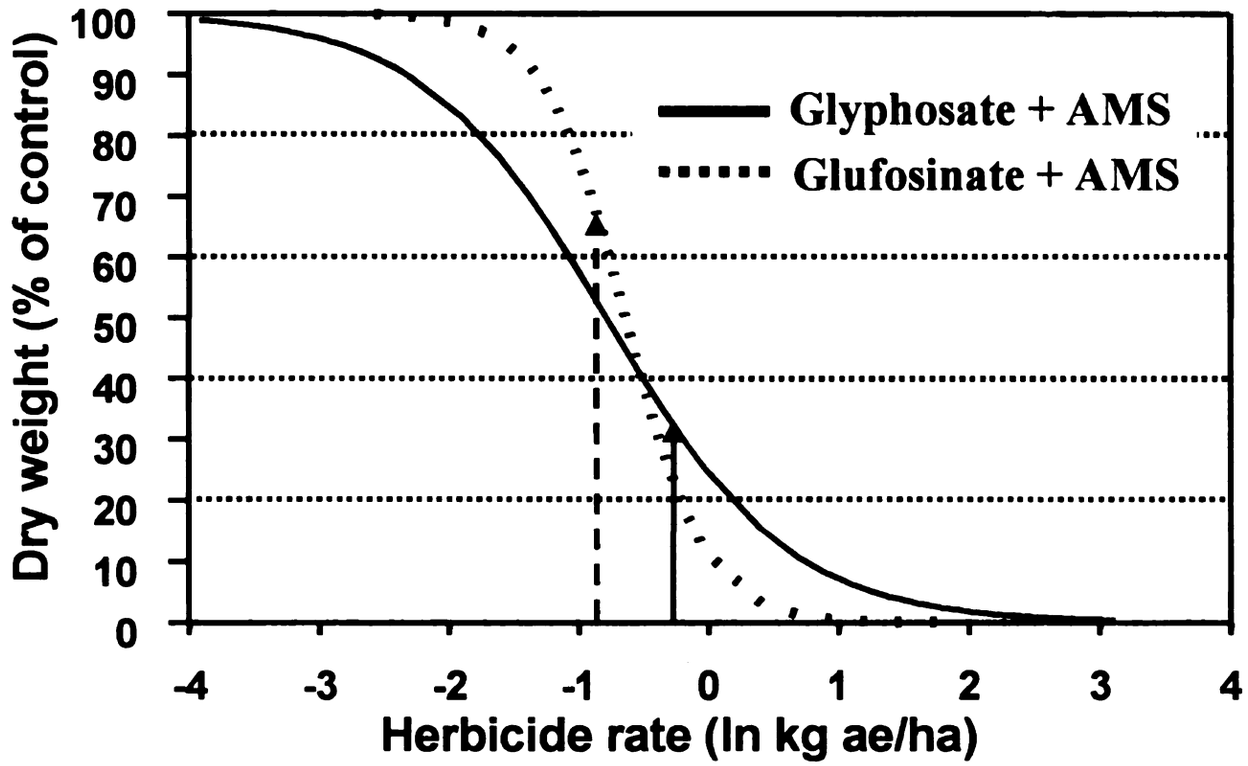

Figure 1. Response of yellow nutsedge shoot dry weight to glyphosate (solid log-logistic curve) and glufosinate (dotted log-logistic curve) plus ammonium sulfate (AMS) at 20 g L^{-1} 4 weeks after treatment. GR_{50} values for glyphosate and glufosinate were 0.51 and 0.59 kg ha^{-1}, respectively. Vertical lines with arrows indicate the predicted control with glyphosate at 0.84 kg ha^{-1} and glufosinate at 0.4 kg ha^{-1}.

SUMMARY

A postemergence application of bentazon/acifluorfen plus thifensulfuron plus sethoxydim and lactofen plus bentazon plus clethodim applied at the V5 stage of soybean development reduced canopy development, delayed reproductive development, and reduced soybean yield 130 to 270 kg ha^{-1} compared to untreated soybean in the absence of white mold. Yield of soybean cultivars varied by year with a ranking of 'A1900' = 'A2704' = 'AG2701' > 'AG1901' in 1997, and 'A2704' > 'AG2701' > 'A1900' > 'AG1901' in 1998. In the presence of white mold, near isogenic glyphosate-resistant ('S14-M7', 'S20-B9', 'GL2600', 'P93B01') and non-resistant ('S 12-49', 'S 19-90', 'GL2415', 'P9281') soybean cultivars were equally susceptible to white mold. However, cultivar selection was important for white mold management since 'S 12-49', 'S14-M7', 'S 19-90', and 'S20-B9' had a lower incidence of white mold than 'GL2415', 'GL2600','P9281', 'P93B01'. Glyphosate did not affect the incidence of white mold in glyphosate-resistant soybean. A reduction in white mold incidence following an application of lactofen may be attributed to increased phytoalexin production, reduced canopy development, and delayed reproductive development. Lactofen may be used as a tool to help manage white mold, but increased soybean yield may not always occur. Other protoporphyrinogen oxidase-inhibiting herbicides such as oxyfluorfen, sulfentrazone, and oxadiazon suppressed *Sclerotinia sclerotiorum* lesion growth similar to lactofen. Sulfentrazone and lactofen increased phytoalexin production similarly.

Yellow nutsedge was suppressed by glyphosate at 840 g ae ha^{-1} 13 to 34% greater than glufosinate at 400 g ae ha^{-1}. Ammonium sulfate increased yellow nutsedge control

with glyphosate and glufosinate, but the addition of a nonionic surfactant, methylated seed oil, or crop oil concentrate applied with glyphosate at 840 g ha^{-1} plus ammonium sulfate at 20 g L^{-1} did not increase yellow nutsedge control. Yellow nutsedge must be managed in glyphosate- and glufosinate-resistant crops by increased use rates or more than a single application of these herbicides or this weed species could become highly problematic in these weed management systems. Tank mixtures of glyphosate with the ALS-inhibiting herbicides increased visual control of yellow nutsedge, but did not reduce tuber production compared to the ALS-inhibiting herbicides applied alone. Yellow nutsedge control by the herbicides evaluated and applied at recommended use rates was ranked halosulfuron and chlorimuron (> 80% control and reduction in tuber density); imazethapyr/imazapyr, imazethapyr, and glyphosate (50 to 80% control and reduction in tuber density); and cloransulam, rimsulfuron, and imazamox (20 to 50% control and reduction in tuber density). However, pyrithiobac controlled yellow nutsedge 48% and reduced tuber density 60%. Long-term yellow nutsedge management may be achieved with treatments that reduce tuber production. This information will help producers in the Midwest and Michigan make informed decisions on how weed management programs impact soybean development, incidence of white mold, soybean yield, and yellow nutsedge tuber yield.