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Résistance de la sétaire verte (*Setaria viridis*) aux inhibiteurs de l'acétolactate synthase

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See table of contents

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Article abstract

Green foxtail (*Setaria viridis*) plants putatively resistant to acetolactate synthase (ALS) inhibitors were identified in a Wisconsin USA no-tillage soybean (*Glycine max*) field in 1999. Resistance to imidazolinone and sulfonylurea herbicides was characterized at the whole-plant level and enzyme level. Three- to four-leaf stage green foxtail plants were 1020, 53, and 6.5-fold resistant to imazethapyr, imazamox, and nicosulfuron, respectively, compared to susceptible plants. In vivo ALS was 1300 and 1.7-fold resistant to imazethapyr and nicosulfuron, respectively. These results suggested that this green foxtail accession was highly resistant to imazethapyr and imazamox, and that resistance was associated with an insensitive ALS enzyme.

Green foxtail (*Setaria viridis*) resistance to acetolactate synthase inhibitors

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[Résistance de la sétaire verte (*Setaria viridis*) aux inhibiteurs de l'acétolactate synthase]

Des sétaires vertes (*Setaria viridis*) présumées résistantes aux inhibiteurs de l'acétolactate synthase (ALS) ont été identifiées en 1999 au Wisconsin, É.-U., dans un champ de soja (*Glycine max*) issu d'un semis direct. La résistance aux herbicides imidazolinone et sulfonylurée a été caractérisée au niveau de la plante entière et de celui des enzymes. Ces sétaires vertes au stade trois à quatre feuilles étaient respectivement 1020, 53 et 6,5 fois plus résistantes à l'imazethapyr, à l'imazamox et au nicosulfuron que les sétaires sensibles. L'ALS *in vivo* était respectivement 1300 et 1,7 fois plus résistante à l'imazethapyr et au nicosulfuron. Ces résultats laissent supposer que ce groupe de sétaires vertes était très résistant à l'imazethapyr et à l'imazamox, et que la résistance est associée à un enzyme ALS insensible.

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INTRODUCTION

Green foxtail (*Setaria viridis* var. *viridis* (L.) Beauv.) is native to Eurasia (Li *et al.* 1942; Li *et al.* 1945) and is found within each state of the continental United States (Lorenzi and Jeffery 1987) and in all provinces of Canada (Douglas *et al.* 1985; Hunter *et al.* 1990). Green foxtail is a major weed problem in temperate regions and is part of the *S. viridis* complex comprised of var. *weinmanni* (R. & S.) Brand. (Hubbard 1915), giant green foxtail var. *major* (Gaud.) Posp. (Slife 1954), robust white foxtail var. *robusta-alba* Schreiber (Schreiber and Oliver 1971), and robust purple foxtail var. *robusta-purpurea* Schreiber (Schreiber and Oliver 1971), representing some of the world's worst weeds (Holm *et al.* 1977). Although phenology and growth characteristics may differ among varieties, no genetic differentiation appears to occur among them (Wang *et al.* 1995). Green foxtail can substantially reduce crop yields in small grains (Blackshaw *et al.* 1981) and row crops such as maize (*Zea mays* L.) (Sibuga and Bandeen 1980). Green foxtail also serves as an alternative host for several pathogenic organisms that are detrimental to small grain production (Haber and Harder 1992; Krupinsky 1992).

Although green foxtail has been typically managed effectively with herbicides, the persistent use of herbicides has created a high level of selection intensity for resistant plants. Green foxtail resistance has been confirmed to three chemical classes of herbicides. In Canada, green foxtail has evolved resistance to dinitroaniline herbicides (Morrison *et al.* 1989) and acetyl-coenzyme A carboxylase (ACCase; EC 6.4.1.2) inhibitors (Heap and Morrison 1996; Marles *et al.* 1993). Furthermore, several populations have developed multiple resistance to these herbicide classes (Morrison and Devine 1994). In the USA, green foxtail has evolved resistance to dinitroanilines only (Heap 2001). In France (De Prado and Menendez 1996), Spain (De Prado *et al.* 2000), and Yugoslavia (Heap 2001), populations of green foxtail have evolved resistance to triazine herbicides.

Mechanisms of green foxtail resistance to herbicides have been associated with changes at the herbicide site of action in most cases. Dinitroanilines inhibit cell division in susceptible species by binding to β-tubulin, and therefore interfere with tubulin polymerization (Devine *et al.* 1993). The mechanism of green foxtail resistance to dinitroanilines has been linked to an alteration of a microtubule-associated protein (Smeda *et al.* 1992). In contrast, goosegrass (*Eleusine indica* (L.) Gaertn.) resistance to dinitroanilines is conferred by a modified β-tubulin (Vaughn and Vaughan 1990), but this mechanism was not associated with gene modifications of α, β or γ tubulin (Mysore and Baird 1995). However, subsequent research (Anthony *et al.* 1998) has shown that a base change in the α tubulin gene of goosegrass results in an amino acid change from threonine to isoleucine that confers resistance to dinitroanilines. The mechanism of green foxtail resistance to ACCase inhibitors is due to an insensitive ACCase (Marles *et al.* 1993). The green foxtail accession from Spain has two mechanisms of resistance within the plant to triazines, an alteration in the target site of photosystem II and enhanced metabolism (De Prado *et al.* 2000).

Weed resistance to herbicides that inhibit acetolactate synthase (ALS) (EC 4.1.3.18) activity has increased dramatically since their introduction in 1982. In 2001, 69 weed species were reported to be resistant to at least one of the five commercialized classes of ALS-inhibiting herbicides (Heap 2001). The resistance mechanism is typically associated with an insensitive ALS enzyme, although non-target site resistance has been reported (Christopher *et al.* 1994; Mallory-Smith *et al.* 1999; Veldhuis *et al.* 2000). Imazethapyr [2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1 *H*-imidazol-2-yl]-5-ethyl-3-pyridinecarboxylic acid] and imazamox [2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1*H*-imidazol-2-yl]-5-(methoxymethyl)-3-pyridinecarboxylic acid] are herbicides commonly used throughout the Canadian prairies and the Midwestern USA grain belt for management of monocotyledonous and dicotyledonous weed

species (Anonymous 2001; Boerboom *et al.* 2000). The herbicide nicosulfuron [2-[[[[4,6-dimethoxy-2-pyrimidinyl)amino]carbonyl]amino]sulfonyl]-*N*,*N*-dimethyl-3-pyridinecarboxamide] is also commonly used to manage monocotyledonous and dicotyledonous weed species in the Midwestern USA grain belt, but has limited use in the Canadian prairies (Anonymous 2001; Boerboom *et al.* 2000). Imazethapyr and imazamox are classified as imidazolinone herbicides, whereas nicosulfuron is classified as a sulfonylurea herbicide; each of these herbicides inhibits ALS. In Canada, imazethapyr is used in various dicotyledonous crops including field pea (*Pisum sativum* L.), dry bean (*Phaseolus vulgaris* L.), herbicide-resistant canola (*Brassica napus* L.) varieties, and seedling alfalfa (*Medicago sativa* L.), whereas in the Midwestern USA, imazethapyr is used primarily in soybean (*Glycine max* (L.) Merr.). Imazamox is used in herbicide- resistant canola varieties and field pea in Canada and primarily in soybean within the Midwestern USA. Nicosulfuron is used in Canada and the Midwestern USA in maize.

In August 1999, a farmer in northwestern Wisconsin reported lack of green foxtail control in a no-tillage soybean field in which imazethapyr and thifensulfuron [3-[[[[(4-methoxy-6-methyl-1,3-5-triazin-2-yl)amino] carbonyl]amino]sulfonyl]-2- thiophenecarboxylic acid] had been applied postemergence at 53 g a.i. ha^{-1} and 2.2 g a.i. ha^{-1}, respectively. The field had been in a maize-soybean rotation since 1994. Sulfonylurea and imidazolinone herbicides were applied for the first time in this field in 1995, but were applied for 3 consecutive yr, starting in 1997 (Table 1). The objectives of our experiments were to confirm and quantify resistance of green foxtail based on whole-plant response to imazethapyr, imazamox, and nicosulfuron, and to determine ALS sensitivity to imazethapyr and nicosulfuron.

Table 1. Crop and herbicide use in a Wisconsin USA field from which a putative acetolactate synthase (ALS) inhibitor-resistant accession of green foxtail was identified

Year	Crop[a]	Herbicide[b]
1991	Alfalfa	none
1992	Alfalfa	none
1993	Alfalfa	glyphosate, 2,4-D
1994	Maize	2,4-D, pendimethalin, dicamba
1995	Soybean	glyphosate, 2,4-D, imazethapyr, thifensulfuron
1996	Maize	glyphosate, 2,4-D, pendimethalin, dicamba
1997	Soybean	glyphosate, 2,4-D, imazethapyr, thifensulfuron
1998	Maize	glyphosate, 2,4-D, metolachlor, nicosulfuron
1999	Soybean	glyphosate, 2,4-D, imazethapyr, thifensulfuron, sethoxydim

[a] Alfalfa (*Medicago sativa* L.); maize (*Zea mays* L.); soybean (*Glycine max* (L.) Merr.).
[b] Glyphosate [*N*-(phosphonomethyl)glycine]; 2,4-D [(2,4-dichlorophenoxy)acetic acid]; pendimethalin [*N*-(1-ethylpropyl)-3,4-dimethyl-2,6-dinitrobenzenamine]; dicamba [3,6-dichloro-2-methoxybenzoic acid]; imazethapyr [2-[4,5-dihydro-4-methyl-4-(1- methylethyl)-5-oxo-1 *H*-imidazol-2-yl]-5-ethyl-3-pyridinecarboxylic acid]; thifensulfuron [3-[[[[(4-methoxy-6-methyl-1,3-5-triazin-2-yl)amino]carbonyl]amino]sulfonyl]-2- thiophenecarboxylic acid]; metolachlor [2-chloro-*N*-(2-ethyl-6-methylphenyl)-*N*-(2-methoxy-1-methylethyl)acetamide]; nicosulfuron [2-[[[[(4,6-dimethoxy-2-pyrimidinyl)amino]carbonyl]amino]sulfonyl]-*N*,*N*-dimethyl-3-pyridinecarboxamide]; sethoxydim [2-[1-(ethoxyimino)butyl]-5-[2-(ethylthio)propyl]-3-hydroxy-2-cyclohexen-1- one].

MATERIALS AND METHODS

Seed sources

In August 1999, three putative herbicide-resistant green foxtail plants were transplanted from a soybean field in Chippewa County, Wisconsin, to a greenhouse at the University of Wisconsin-Madison. Plants were grown to maturity and seeds were collected for experiments. Seeds from putative herbicide-susceptible green foxtail plants were collected in September 1999 in a maize field in Dane County, Wisconsin, in which no ALS-inhibiting herbicides had been applied. All plants were identified as green foxtail *Setaria viridis* var. *viridis* based on morphological characteristics described by Douglas *et al.* (1985).

Whole-plant dose-response

Experiments were conducted in a greenhouse at the University of Wisconsin-Madison using three- to four-leaf stage green foxtail plants. Approximately 10 seeds of resistant or susceptible green foxtail accessions were placed on the surface of an autoclaved soil mixture of silt loam and sand (3:2 v:v) in a 325 mL container and covered with 1 cm of the soil mixture. Four d after emergence, plants were thinned to one plant per container. Plants were grown under natural light supplemented with artificial light from metal halide lamps to extend the photoperiod to 16 h at 25/20°C (day/night) temperatures.

Commercially formulated herbicides were used in all dose-response experiments. Each experiment consisted of a single herbicide. Herbicide doses were determined from preliminary experiments. Imazethapyr was applied at 0, 0.70, 1.4, 2.8, 5.6, and 11 g a.i. ha^{-1} to susceptible plants and at 0, 70, 140, 280, 560, and 1120 g a.i. ha^{-1} to resistant plants. Imazamox was applied at 0, 0.3, 0.6, 1.1, 2.2, and 4.4 g a.i. ha^{-1} to susceptible plants and at 0, 44, 88, 176, 352, and 704 g a.i. ha^{-1} to resistant plants. Nicosulfuron was applied at 0, 0.11, 0.22, 0.44, 0.88, and 1.75 g a.i. ha^{-1} to susceptible plants and at 0, 0.88, 1.75, 3.5, 7.0, and 14 g a.i. ha^{-1} to resistant plants. Each herbicide treatment included nonionic surfactant (NIS) at 0.25% (v:v) and 28% nitrogen (N) as urea-ammonium nitrate [$(CO(NH_2)_2)$-NH_4NO_3] at 1.25% (v:v). A stationary pot sprayer with one 8002E nozzle calibrated to deliver 187 L ha^{-1} at 275 kPa was used for each herbicide application.

Shoot biomass was harvested 14 d after treatment (DAT), dried at 70°C for 72 h, and weighed. The experimental design was completely randomized for each experiment with four replicates per treatment. The experimental unit was one plant. Experiments were repeated at least once. Each herbicide was evaluated as a separate experiment. Analysis of variance was performed on all data. For each herbicide, the experiment by treatment interaction was not significant and the data from repeated experiments were pooled. A non-linear logistic four-parameter model was simultaneously fit to the dose-response data for the resistant and susceptible accessions of each replication (Stoltenberg and Wiederholt 1995; Volenberg *et al.* 2000a; Volenberg *et al.* 2001). To obtain dose-response curves, data for each replication were regressed using the following model: $y = C + (D-C)/(1 + exp((logI_{50} - logX)\ b))$. The model estimated the dosage of ALS inhibitor that reduced shoot dry biomass by 50% (ED_{50}) relative to non-treated plants and included the following parameters: y, shoot dry biomass; C, the lower limit of the ALS inhibitor dose-response curve at the highest herbicide concentration; D, the upper limit of the ALS inhibitor dose-response curve at the lowest herbicide concentration; b, the slope of the ALS inhibitor dose-response curve around the ED_{50}; and X, the ALS inhibitor concentration. Nonlinear regression equations were calculated using GraphPad Prism version 3.02 (GraphPad Software, San Diego, CA) curve fitting software. A Runs Test determined if sigmoidal equations adequately described the data ($P = 0.05$) (Bradley 1968). The ratio of resistant to susceptible ED_{50} values was calculated to determine the level of resistance to each herbicide.

In vivo ALS assay

Experiments were conducted in a greenhouse and laboratory at the University of Wisconsin-Madison following proce-

dures described by Lovell *et al.* (1996). Green foxtail plants were grown as described above. Resistant or susceptible three- to four- leaf stage plants were treated with 0, 0.007, 0.07, 0.70, 7.0, 70, 700, and 1120 g a.i. ha^{-1} imazethapyr or nicosulfuron. Herbicides were applied with a stationary pot sprayer as described above. All herbicide treatments included NIS at 0.25% (v:v) and 28% N at 1.25% (v:v). Plants were treated with 1,1-cyclopropanedicarboxylic acid (CPCA) at 766 g ha^{-1} containing NIS at 0.25% (v:v) 21 h after herbicide application (Gerwick *et al.* 1993; Simpson *et al.* 1995). Resistant or susceptible negative control plants were not treated with herbicide or CPCA.

Acetolactate synthase activity was measured by the accumulation of acetolactate in leaf tissue 24 h after herbicide treatment. The third and fourth leaf were removed from each plant and cut into 1 to 2 mm wide sections. A total of 0.2 g of leaf tissue was harvested per plant and stored at -20°C for 24 h to rupture cell walls. Three mL of purified water was added to each sample, which was incubated at 60°C for 30 min and at 20°C for 45 min. Each sample was mixed every 15 min: twice at 60°C and three times at 20°C. A 2 mL aliquot was removed from each sample and 50 µL 6 N H_2SO_4 were added. Aliquots were incubated at 60°C for 30 min to decarboxylate acetolactate to acetoin. One mL of a creatine (9 g L^{-1}) and napthol (90 g L^{-1}) solution in 2.5 N NaOH was added to each sample, which was mixed and incubated at 60°C for 30 min. Samples were cooled to ambient room temperature and centrifuged at 10 000 *g* for 10 min. Absorbance of acetoin was measured at 530 nm. Extracts from plant leaves not treated with herbicide or CPCA were used as background for spectrophotometric measurements. Absorbance measurements were converted to µg acetoin using a standard curve. Enzyme activity was expressed as µg acetoin g^{-1} (fresh biomass) h^{-1}.

The experimental design was completely randomized with four replications per treatment. The experimental unit was one plant. Experiments were repeated once. Analysis of variance was performed on all data. For each herbicide, the experiment by treatment interaction was not significant and the data from repeated experiments were pooled. Non-linear dose-response analysis was conducted as described above, and the dose that inhibited ALS activity by 50% (I_{50}) relative to non-treated plants was calculated. The ratio of resistant to susceptible I_{50} values was calculated to determine the level of resistance to each herbicide.

Table 2. Parameter values of whole-plant dose-response curves (shown in Figure 1) for resistant and susceptible accessions of green foxtail as influenced by imazethapyr, imazamox, and nicosulfuron

	Parameter value[a]							
	Accession							
	Resistant				Susceptible			
Herbicide	C mg $plant^{-1}$	D mg $plant^{-1}$	b	R^2	C mg $plant^{-1}$	D mg $plant^{-1}$	b	R^2
Imazethapyr	450 ± 24[b]	980 ± 21	-2.42 ± 0.51	0.98	68 ± 18	1590 ± 59	-1.08 ± 0.27	0.97
Imazamox	150 ± 46	885 ± 59	-2.32 ± 0.75	0.98	16 ± 14	830 ± 63	-1.88 ± 0.70	0.98
Nicosulfuron	207 ± 37	690 ± 15	-1.99 ± 0.37	0.97	158 ± 19	1128 ± 35	-0.70 ± 0.31	0.96

[a] $y = C + (D-C)/(1 + \exp((\log I_{50} - \log X)\, b))$; y, shoot dry biomass; C, the lower limit of the dose-response curve at the highest herbicide concentration; D, the upper limit of the dose-response curve at the lowest herbicide concentration; b, the slope of the dose-response curve around the ED_{50}; and X, the herbicide concentration.

[b] Fitted value ± standard error.

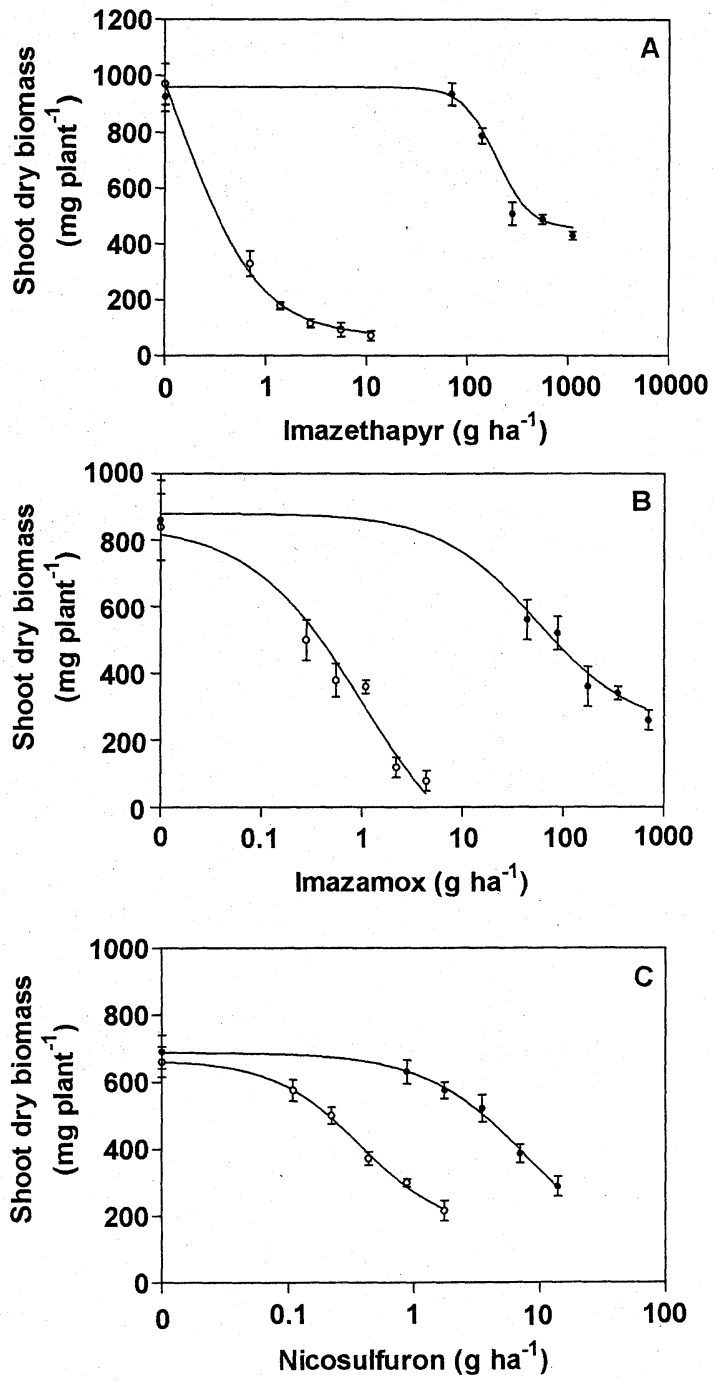

Figure 1. Whole-plant dose response of resistant (•) and susceptible (○) accessions of green foxtail to imazethapyr (A), imazamox (B), and nicosulfuron (C). Parameter values for dose-response curves are shown in Table 2. Each point represents the mean of eight replicates ± SE.

Table 3. Whole-plant dose-response (ED_{50}) of resistant (R) and susceptible (S) green foxtail to imazethapyr, imazamox, and nicosulfuron

	ED_{50} value[a] (g ha^{-1})		
	Accession		
Herbicide	R	S	R/S
Imazethapyr	175 ± 11	0.2 ± <0.1	1020 ± 99
Imazamox	59 ± 8	1.2 ± 0.2	53 ± 10
Nicosulfuron	4.6 ± 1.4	1.0 ± 0.6	6.5 ± 1.7

[a] ED_{50} value indicates effective herbicide dose that reduced shoot dry biomass by 50% relative to non-treated plants. Each value represents the mean of eight replicates ± 95% confidence interval.

RESULTS

The non-linear model fit all shoot dry biomass data (Table 2; Fig. 1). Resistance to imazethapyr and imazamox was confirmed by the high ratios of R to S whole-plant ED_{50} values (Table 3). The imazethapyr ED_{50} value was 175 g ha^{-1} for the resistant accession and 0.2 g ha^{-1} for the susceptible accession. The imazamox ED_{50} value was 59 g ha^{-1} for the resistant accession and 1.2 g ha^{-1} for the susceptible accession. Based on ED_{50} values, the resistant accession was 1020 and 53-fold less sensitive to imazethapyr and imazamox, respectively, compared to the susceptible accession. Furthermore, the imidazolinone-resistant green foxtail accession was also resistant to the sulfonylurea herbicide nicosulfuron at the whole-plant level. The nicosulfuron ED_{50} value was 4.6 g ha^{-1} for the resistant accession and 1.0 g ha^{-1} for the susceptible accession. Based on these values, the resistant accession was 6.5-fold resistant to nicosulfuron. The resistant accession was susceptible to the ACCase inhibitors sethoxydim [2-[1-(ethoxyimino)butyl]-5-[2-(ethylthio)propyl]-3-hydroxy-2-cyclohex-1- one] at 315 g a.i. ha^{-1} in the field and fluazifop-P [(*R*)-2-[4-[[5-(trifluoromethyl)-2- pyridinyl]oxy]phenoxy]propionic acid] at 210 g a.i. ha^{-1} in the greenhouse

Table 4. Parameter values of acetolactate synthase (ALS) dose-response curves (shown in Figure 2) for resistant and susceptible accessions of green foxtail as influenced by imazethapyr and nicosulfuron

	Parameter value[a]							
	Accession							
	Resistant				Susceptible			
Herbicide	C (µg acetoin g $FW^{-1}h^{-1}$)	D	b	R^2	C (µg acetoin g $FW^{-1}h^{-1}$)	D	b	R^2
Imazethapyr	0.95 ± 0.02[b]	1.86 ± 0.02	-0.89 ± 0.09	0.95	0.33 ± 0.13	2.37 ± 0.40	-0.38 ± 0.12	0.92
Nicosulfuron	0.84 ± 0.03	1.63 ± 0.03	-1.50 ± 0.65	0.96	0.30 ± 0.05	1.56 ± 0.07	-0.95 ± 0.29	0.94

[a] $y = C + (D-C)/(1 + \exp((\log I_{50} - \log X)\, b))$; y, acetoin concentration; C, the lower limit of the dose-response curve at the highest herbicide concentration; D, the upper limit of the dose-response curve at the lowest herbicide concentration; b, the slope of the dose-response curve around the I_{50}; and X, the herbicide concentration.

[b] Fitted value ± standard error.

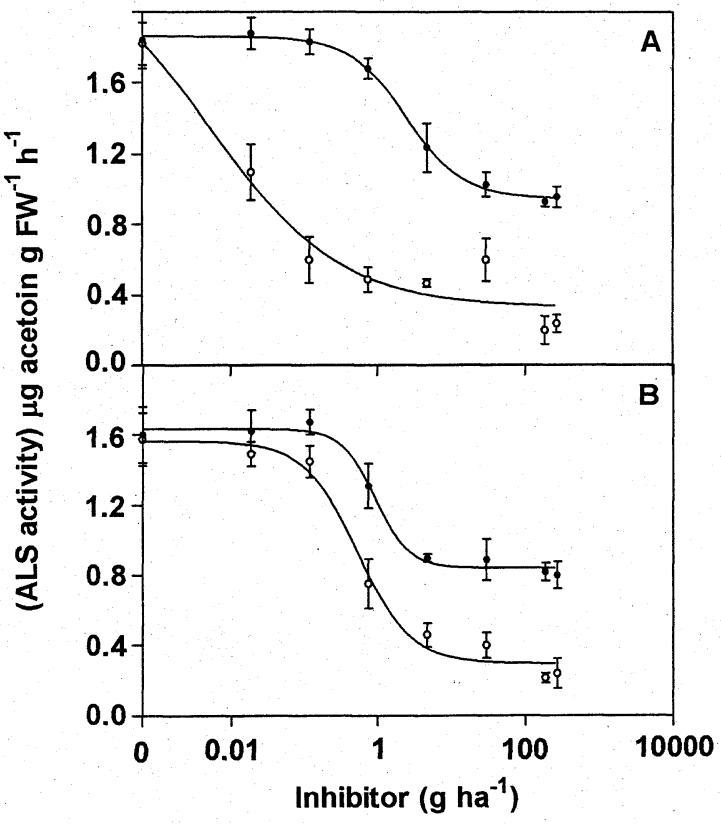

Figure 2. ***In vivo*** **inhibition of acetolactate synthase (ALS) from resistant (•) and susceptible (°) accessions of green foxtail by imazethapyr (A) and nicosulfuron (B).** Parameter values for dose-response curves are shown in Table 4. Each point represents the mean of eight replicates ± SE.

Table 5. Acetolactate synthase (ALS) enzyme response (I_{50}) of resistant (R) and susceptible (S) green foxtail to imazethapyr and nicosulfuron

	I_{50} value[a] (g ha^{-1})		
	Accession		
Herbicide	R	S	R/S
Imazethapyr	3.2 ± 0.3	2.4 x 10^{-3} ± 1.6 x 10^{-4}	1300 ± 100
Nicosulfuron	0.74 ± 0.07	0.42 ± 0.04	1.7 ± 0.7

[a] I_{50} value indicates effective herbicide dose that inhibited ALS activity by 50% relative to non-treated plants. Each value represents the mean of eight replicates ± 95% confidence interval.

(data not shown), suggesting no cross-resistance to ACCase-inhibiting herbicides.

The non-linear model fit all data from the ALS *in vivo* assay (Table 4; Fig. 2). Imazethapyr and nicosulfuron I_{50} values were greater for the ALS-inhibitor resistant accession than for the susceptible accession (Table 5). The imazethapyr I_{50} value was 3.2 g ha^{-1} for the resistant accession and 0.0024 g ha^{-1} for the susceptible accession. The nicosulfuron I_{50} value was 0.7 g ha^{-1} for the resistant accession and 0.4 g ha^{-1} for the susceptible accession. Based on I_{50} values, the ALS-inhibitor resistant green foxtail accession was 1300- and 1.7-fold resistant to imazthapyr and nicosulfuron, respectively, compared to the susceptible accession.

DISCUSSION

These results indicated that green foxtail from Wisconsin is highly resistant to imazethapyr and imazamox, but much less resistant to nicosulfuron. The mechanism of resistance is due to an insensitive ALS enzyme. Relatively few grass species have evolved resistance to imidazolinone and sulfonylurea herbicides. Rigid ryegrass (*Lolium rigidum* Gaud.) in Australia (Heap and Knight 1986) and blackgrass (*Alopecurus myosuroides* Huds.) in England (Moss and Cussans 1991) have evolved cross-resistance to sulfonylureas, but were selected with non-ALS inhibiting herbicides. Goosegrass and Honduras grass (*Ixophorus unisetus* (Presl) Schlecht.) in Costa Rica selected with imazapyr were cross-resistant to both imidazolinone and sulfonylurea herbicides (Valverde *et al.* 1993). Within the USA, few grass species have developed resistance to ALS-inhibiting herbicides, including shattercane (*Sorghum bicolor* (L.) Moench) (Anderson *et al.* 1998), Italian ryegrass (*Lolium multiflorum* L.) (Taylor and Coats 1996), wild oat (*Avena fatua* L.) (Nandula and Messersmith 2000) and giant foxtail (*Setaria faberi* Herrm.) (Volenberg *et al.* 2001). ALS inhibitor-resistant giant foxtail accessions from Wisconsin, Illinois, and Minnesota are cross-resistant to imidazolinone and sulfonylurea herbicides (Volenberg *et al.* 2001).

The selection of resistant green foxtail occurred in a Wisconsin field where imidazolinone or sulfonylurea herbicides were applied annually for three consecutive yr. Other ALS-inhibitor resistant weed species have evolved under similar herbicide selection intensities. Eastern black nightshade (*Solanum ptycanthum* L.) evolved ALS-inhibitor resistance after only four applications of imazethapyr over a 5-yr period (Volenberg *et al.* 2000a). However, selection for resistance to ALS inhibitors can occur over a longer period. A giant foxtail accession resistant to ALS inhibitors was selected in a Wisconsin maize field where nicosulfuron was applied annually for 9 yr (Volenberg *et al.* 2000b).

Multiple herbicide resistance has evolved in green foxtail populations to dinitroaniline and ACCase-inhibiting herbicides in Canada (Morrison and Devine 1994). However, it is not known whether these populations were selected for by herbicide use or by interbreeding between dinitroaniline- and ACCase-resistant populations. Although green foxtail is considered self-pollinating, outcrossing has been quantified in the field at levels as high as 2.2% (Bottraud *et al.* 1992). However, it is unlikely that multiple herbicide resistant green foxtail evolved through interbreeding since resistance to the dinitroaniline trifluralin (2,6-dinitro-*N,N*-dipropyl-4-(trifluoromethyl)benzenamine) is inherited as a recessive trait (Jasieniuk *et al.* 1994). Therefore, management of resistant green foxtail should address the spread of resistance traits via seed immigration.

Populations resistant to herbicides can become widespread in a short period of time as evidenced by dinitroaniline-resistant green foxtail in Canada. Five yr after the confirmation of resistance (Morrison *et al.* 1989), the number of fields infested with dinitroaniline-resistant green foxtail across the prairies was so large that it was difficult to estimate the number of fields infested (Morrison and Devine 1994). Furthermore, increased availability and adoption of transgenic herbicide-resistant

crop species may be associated with even greater reliance on single herbicide chemistries (e.g. ALS inhibitors) for weed management (Warwick *et al.* 1999). As such, management practices that integrate cultural, mechanical, and alternative chemical approaches should be implemented for management of ALS inhibitor-resistant green foxtail populations, as well as other herbicide-resistant weed species, and to help delay development of additional resistance problems.

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