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Triallate-resistant wild oat (*Avena fatua* L.): unexpected resistance to pyroxasulfone and sulfentrazone

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Abstract: Wild oat is the most economically detrimental weed species in the Canadian Prairies and effective herbicidal control options are limited due to widespread resistance to ACCase inhibitors, ALS inhibitors, and lipid biosynthesis inhibitors; therefore, evaluation of new herbicidal modes of action such as pyroxasulfone and sulfentrazone for control is critical. Two wild oat populations (HR08-210 and HR11-151) were first subjected to a discriminating dose screen to characterize resistance to ACCase and ALS inhibiting herbicides in comparison with a susceptible population (S1988). Dose-response experiments with triallate, pyroxasulfone, and sulfentrazone were then conducted to evaluate potential cross-resistance. Screening indicated both herbicide-resistant (HR) populations were resistant to ACCase- and ALS inhibiting herbicides, most likely due to enhanced metabolism and an ACCase mutation. HR08-210 and HR11-151 were resistant to triallate (resistance ratios of 2.53 and 3.39, respectively), but cross-resistance to pyroxasulfone (2.78) and sulfentrazone (2.0) was only observed in HR11-151. Results indicate previously selected resistance to ACCase and ALS inhibitors (enhanced metabolism and ACCase mutation) or triallate (enhanced endogenous gibberellins) could limit the utility of new herbicide modes of action for control of wild oat.

Key words: cross-resistance, multiple resistance.

Résumé : La folle avoine est la mauvaise herbe économiquement la plus nuisible dans les Prairies canadiennes et les herbicides permettant de lutter contre elle sont peu nombreux en raison de la résistance très répandue aux inhibiteurs de l'ACCase, de l'ALS et de la biosynthèse des lipides. Par conséquent, il est capital d'évaluer le mode d'action des nouveaux herbicides comme le pyroxasulfone et le sulfentrazone si l'on veut combattre l'adventice. Deux peuplements de folle avoine (HR08-210 et HR11-151) ont été soumis à une dose de discrimination d'herbicides pour déterminer s'ils étaient résistants aux herbicides inhibant l'ACCase et l'ALS, comparativement à un peuplement sensible (S1988). Les auteurs ont ensuite effectué des expériences dose-réponse avec du triallate, du pyroxasulfone et du sulfentrazone pour vérifier s'il y avait résistance croisée. L'analyse initiale indique que les deux populations testées résistaient aux herbicides inhibant l'ACCase et l'ALS, sans doute en raison d'un meilleur métabolisme et d'une mutation de l'ACCase. HR08-210 et HR11-151 résistent au triallate (rapport de 2,53 et de 3,39, respectivement), mais la résistance croisée au pyroxasulfone (2,78) et au sulfentrazone (2,0) n'a été observée que chez HR11-151. Ces résultats indiquent qu'une résistance initiale aux inhibiteurs de l'ACCase et de l'ALS (meilleur métabolisme et mutation de l'ACCase) ou au triallate (meilleures gibbérellines endogènes) pourrait restreindre l'utilité des herbicides à nouveau mode d'action pour lutter contre la folle avoine. [Traduit par la Rédaction]

Mots-clés : résistance croisée, résistance multiple.

Introduction

Wild oat (*Avena fatua L.*) is one of the 15 most important herbicide-resistant (HR) weed species worldwide (Heap 2015), and the most abundant HR weed species in the Canadian Prairies (Beckie et al. 2013). Triallate (Group 8/N), a soil-applied, lipid synthesis inhibitor, was first introduced in 1961 for pre-emergent selective wild oat control and was used extensively until more

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Abbreviations: HR, herbicide resistant; **ACCase**, acetyl-CoA carboxylase; **ALS**, acetoacetate synthase; **ai**, active ingredient; **ha**, hectare; **LD50**, 50% of the lethal dose; **MOA**, mechanism of action.

efficacious ACCase inhibitors (Group 1/A) and ALS inhibitors (Group 2/B) were introduced beginning in the late 1970s (Beckie et al. 1999). Wild oat resistant to triallate and cross-resistant to difenzoquat was reported by O'Donovan et al. (1994), and reports of resistance to ACCase inhibitors and ALS inhibitors followed (Joseph et al. 1990; Beckie et al. 1999). Wild oat resistance is now common across the prairies; Beckie et al. (2013) reported 28%, 12%, and 8% of fields surveyed between 2007 and 2009 contained wild oat populations resistant to ACCase inhibitors, ALS inhibitors, and triallate, respectively. Multiple resistant HR populations reported were ACCase + ALS (8%), ACCase + triallate (5%), ALS + triallate (2%), and ACCase + ALS + triallate (3%).

Cross-resistance between herbicides with different sites of action may be conferred by non-target site resistance such as herbicide metabolism, or multiple-resistance can be conferred by several target sites and (or) non-target site resistance mechanisms (Preston et al. 2001; Beckie and Tardif 2012). The mechanisms of resistance in wild oat have not all been clarified. Rashid et al. (1998) proposed that increased levels of endogenous gibberellin in triallate-resistant wild oat increased germination and accelerated shoot growth, allowing for seedlings to avoid toxic herbicidal effects. Alternatively, Kern et al. (1996) reported a correlation between reduced triallate metabolic activation (sulfoxidation) and triallate resistance in wild oat. However, reduced metabolism does not explain resistance to other selective herbicides or to difenzoquat, as difenzoquat does not require sulfoxidation for activation (Sharma et al. 1976; Kern et al. 1996). Kern and Dyer (1998) proposed a separate resistance mechanism of cell wall sequestration was conferring resistance to difenzoquat in these populations. Upon further investigation into the triallate mechanism of resistance, Kern et al. (2002) suggested that two independently segregating, recessive genes (*TRR1, TRR2*) controlled resistance. Target-site mutations and increased metabolism are the primary mechanisms responsible for HR ACCase and ALS populations (Beckie et al. 2012).

Due to the widespread resistance to current wild oat herbicides, Canadian producers are looking for options to control these HR populations. Pyroxasulfone (Group 15/K3), a very-long chain fatty acid (VLCFA) biosynthesis inhibitor, has recently been introduced in Canada. It is a pre-emergent herbicide registered for control of grasses and small-seeded broadleaves in corn, soybean, sunflower, and field pea (Tanetani et al. 2009). Pyroxasulfone has shown success in managing other problematic HR weeds in the United States and Australia due to its ability to inhibit multiple steps in the elongation pathways of VLCFA compared with other lipid synthesis inhibitors (Tanetani et al. 2009; Busi et al. 2012, 2014). Pyroxasulfone control of wild oat was reported by Tidemann et al. (2014) in combination with the soil-applied PPO inhibitor sulfentrazone (Group 14/E).

Resistance to sulfentrazone has been reported in populations of smooth pigweed (*Amaranthus hybridus*), common ragweed (*Ambrosia artmisiifolia*), and common waterhemp species (*Amaranthus tuberculatus*; *Amaranthus rudis*) that were also resistant to multiple other PPO inhibitors. Sulfentrazone resistance was attributed to target site mutations to protoporphyrinogen oxidase, a single codon deletion (ΔG210 PPX2L) in waterhemp and a point mutation in common ragweed (R98L PPXL) (Patzoldt et al. 2006; Rousonelos et al. 2012).

Worldwide, four grass species, including annual ryegrass (*Lolium rigidum* L. Gaud.), have been reported resistant to VLCFA inhibitors (other than pyroxasulfone) (Heap 2015). Screening of susceptible annual ryegrass with high doses of pyroxasulfone did not select for resistant individuals, indicating that any major genes potentially responsible for resistance either have very low penetrance or are extremely rare (Busi et al. 2012). However, low-dose selection of a multi-resistant (metabolism based) population of annual ryegrass by pyroxasulfone showed a shift towards pyroxasulfone resistance with LD_{50} increasing from 46 g ai ha^{-1} in the parent to 208 g ai ha^{-1} by the third generation (Busi et al. 2012). This resistance was later characterized as non-target site-based and conferred by one semi-dominant allele (Busi et al. 2014). This shift towards pyroxasulfone resistance showed a corresponding increase in resistance to prosulfocarb and triallate (Busi and Powles 2013), suggesting cross-resistance from a single mechanism. The annual ryegrass population with the highest resistance to pyroxasulfone showed 81% and 39% survival for prosulfocarb and triallate, respectively, at label rates (Busi and Powles 2013). Enhanced metabolic herbicide detoxification is thought to be the main mechanism of resistance in annual ryegrass and is also the main mechanism of selectivity in wheat (Shimabukuro et al. 1979; Shimabukuro and Hoffer 1991; Busi and Powles 2013; Tanetani et al. 2013).

The objective of this study was to determine if two Canadian wild oat populations that have been selected for resistance to triallate exhibited cross-resistance to pyroxasulfone similar to annual ryegrass in Australia. Additionally, cross-resistance to the pre-emergent PPO inhibitor sulfentrazone was quantified because it may be an additional tool for managing HR weed populations.

Materials and Methods

Plant material

Seed of the susceptible wild oat population was collected from the University of Alberta farm in the 1988 growing season (S1988). Two resistant wild oat populations were submitted to Saskatoon Research Centre, Agriculture and Agri-Food Canada by growers for testing with suspected resistance to triallate: an HR08-210 population from Olds, AB and an HR11-151 population from Rivers, MB. In 2014, resistant seeds were grown in the

Table 1. Herbicides used in wild oat resistance screening.

Herbicide	Formulation	Mode of action	Selective in wheat	Rate (g ai ha^{-1})
Fenoxaprop	120 g L^{-1} EC	ACCase Inhibitor (Aryloxyphenoxypropionate)	Yes	150
Quizalofop	96 g L^{-1} EC	ACCase Inhibitor (Aryloxyphenoxypropionate	No	35
Imazamethabenz	300 g L^{-1} SC	ALS inhibitor (Imidazolinone)	Yes	500
Imazapyr	240 g L^{-1} SN	ALS inhibitor (Imidazolinone)	No	717

absence of herbicide in separate locations in the field at the University of Alberta and progeny were collected. Germination tests were conducted to ensure each population had a minimum of 60% germination prior to initiation of dose-response experiments (data not shown).

Herbicide screening tests

Wild oat biotypes were screened with selective and non-selective ACCase and ALS inhibitors (Table 1) to distinguish between the resistance mechanisms that could be present. Wild oats were seeded in Sunshine Professional Growing Mix® and populations were screened with the ACCase-inhibitor herbicides fenoxaprop and quizalofop, and ALS-inhibitor herbicides imazamethabenz and imazapyr (Table 1). Fenoxaprop and imazamethabenz are selective herbicides and metabolized in wheat, while quizalofop and imazapyr are not metabolized. If a biotype is resistant only to the herbicide that is metabolized, it is an indication that metabolism may be responsible for resistance rather than a target-site mutation. Herbicides were applied with a moving track cabinet chamber sprayer calibrated for 200 L ha^{-1} at 207 kPa using an Air Bubble Jet 110015 nozzle at the three-leaf stage of wild oat. After treatment, plants were returned to a greenhouse and natural light was supplemented with 16 h of artificial light and at a temperature of 21 °C.

Screening methods were adapted from Beckie et al. (2013); individual plants were visually assessed as HR (2, some injury but new growth, or 3, no injury) or herbicide susceptible (HS) (0 = dead or 1 = nearly dead) at 21 d after treatment (DAT). Approximately 50 plants were screened in each resistance test and treatments were replicated four times. We assumed wild oat populations submitted for testing were heterogeneous, consisting of both HS and HR individuals having one or multiple herbicide resistance mechanisms.

Dose response studies

Dose-response experiments were completed for pyroxasulfone, sulfentrazone, and triallate. Because activity of these herbicides may be affected by soil organic matter, field soil collected from Kinsella Research Station in the fall of 2014 was homogenized in a soil mixer and used to plant 15 intact seeds of each population 1 cm deep in 8.25 cm diameter pots. The soil was a sandy loam with 58% sand, 30% silt, and 12% clay with an organic matter content of 6%. Immediately after seeding, the soil surface was sprayed with a proportion of the label rate of pyroxasulfone (150 g ai ha^{-1}), sulfentrazone (140 g ai ha^{-1}) or triallate (1180 g ai ha^{-1}). Application rates were either pyroxasulfone (85% WG) at 0, 37.5, 75, 150, 300, and 600 g ai ha^{-1}, sulfentrazone (470 g L^{-1} SC) at 0, 35, 70, 140, 280, and 560 g ai ha^{-1}, or triallate (480 g L^{-1} EC) at 0, 295, 590, 1180, 2360, and 4720 g ai ha^{-1}. Herbicides were applied using a moving track cabinet chamber sprayer calibrated for 200 L ha^{-1} at 207 kPa using an Air Bubble Jet 110015 nozzle. Immediately after treatment, pots were placed in a greenhouse. Natural light was supplemented with 16 h of artificial light and at a temperature of 21 °C. Pots were watered and rotated daily to reduce positional effects. Emergence counts were conducted 2 wk after seeding. At 4 wk, plants were uprooted and washed. Plants were classified as either dead if they ceased growth due to injuries, or healthy if growth continued. Survival was determined by dividing the number of wild oats healthy and alive at 4 wk by the total number of germinated seeds per pot. Shoot length, root length (data not shown), and fresh weight were quantified for each plant. Each pot was considered an experimental unit.

The experimental design was a randomized complete block with four replicates for each dose response experiment and replicates were seeded one per week for 4 wk. Survival data was analyzed using a binomial two-parameter log-logistic model using the *drm()* function in the *drc* package in the software program R (v. 0.98.1091). Population response to herbicide treatments was measured as the *R*/*S* (resistant/susceptible) ratio of estimated LD_{50} values.

Results and Discussion

Herbicide screening tests

Results from the screen with ACCase and ALS inhibitors indicated that HR08-210 and HR11-151 were resistant to ACCase and ALS inhibitors (Table 2). HR08-210 and HR11-151 wild oat populations acted similar in response to all herbicides. All populations exhibited visual symptoms after fenoxaprop application, but resistant populations showed regrowth, suggesting metabolism-based resistance. A high level of resistance to the non-selective herbicide quizalofop indicates an ACCase target-site mutation is also conferring resistance to ACCase inhibitors. HR08-210 and HR11-151 were highly resistant to the ALS inhibitor imazamethabenz, yet susceptible to

Table 2. Resistant screening of wild oat populations S1988, HR08-210, and HR11-151. The number in brackets indicated the mean % of *R* (resistant) individuals present in each screen ($N = 4$).

		Population		
Herbicide	Site of action	S1988	HR08-210	HR11-151
Fenoxaprop	ACCase	*S* (0)	*R* (35%)	*R* (36%)
Quizalofop	ACCase	*S* (0)	*R* (85%)	*R* (86%)
Imazamethabenz	ALS	*S* (0)	*R* (86%)	*R* (87%)
Imazapyr	ALS	*S* (0)	*S* (0)	*S* (0)

Note: R, resistant; S, susceptible.

Table 3. Estimated LD50 values and *R*/*S* ratios for wild oat populations S1988, HR08-210 and HR11-151 in a dose response study with triallate, pyroxasulfone, and sulfentrazone.

Biotype	LD_{50} (±SE)[a] (g ai ha^{-1})	*R*/*S*[b]	*P*-value[c]
Triallate			
S1988	270 (50)	—	—
HR08-210	681 (95)	2.53	0.0088**
HR11-151	915 (81)	3.39	0.0006**
Pyroxasulfone			
S1988	41 (8)	—	—
HR08-210	46 (13)	1.13	0.7429
HR11-151	114 (13)	2.78	0.0063**
Sulfentrazone			
S1988	183 (13)	—	—
HR08-210	236 (34)	1.28	0.1740
HR11-151	375 (83)	2.0	0.0290*

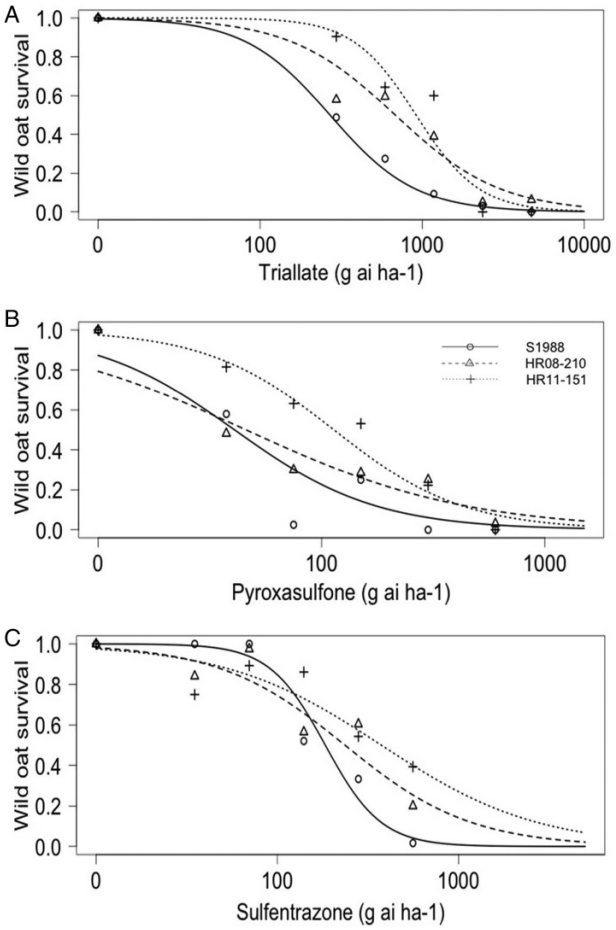
[a]LD_{50}, lethal dose required for 50% survivorship of wild oat biotypes. The values in parentheses are standard errors.
[b]R/S, ratio (resistant/susceptible) referred to the standard susceptible population S1988.
[c]*P*-value, indicated significance between LD_{50} values when treated with a particular herbicide (*$P < 0.05$, **$P < 0.001$).

imazapyr. This suggests that resistance to ALS inhibitors was due to enhanced metabolism and not an ALS target-site mutation. Resistance to ALS inhibitors in wild oat is more commonly due to enhanced metabolism rather than target-site mutation; in contrast, ACCase inhibitor resistance in the species is often due to target-site mutation, followed by enhanced metabolism by P450 monooxygenases (Beckie et al. 2012).

Dose response studies

Both wild oat biotypes HR08-210 and HR11-151 were resistant to triallate, with *R*/*S* ratios of 3.39 ($P < 0.01$) for HR11-151 and 2.53 ($P < 0.01$) for HR08-210 (Table 3). Wild oat population HR11-151 showed cross-resistance to both pyroxasulfone and sulfentrazone with *R*/*S* ratios of 2.78 ($P < 0.01$) and 2.0 ($P < 0.05$), respectively. The HR08-210 population was not classified as resistant, with *R*/*S* ratios

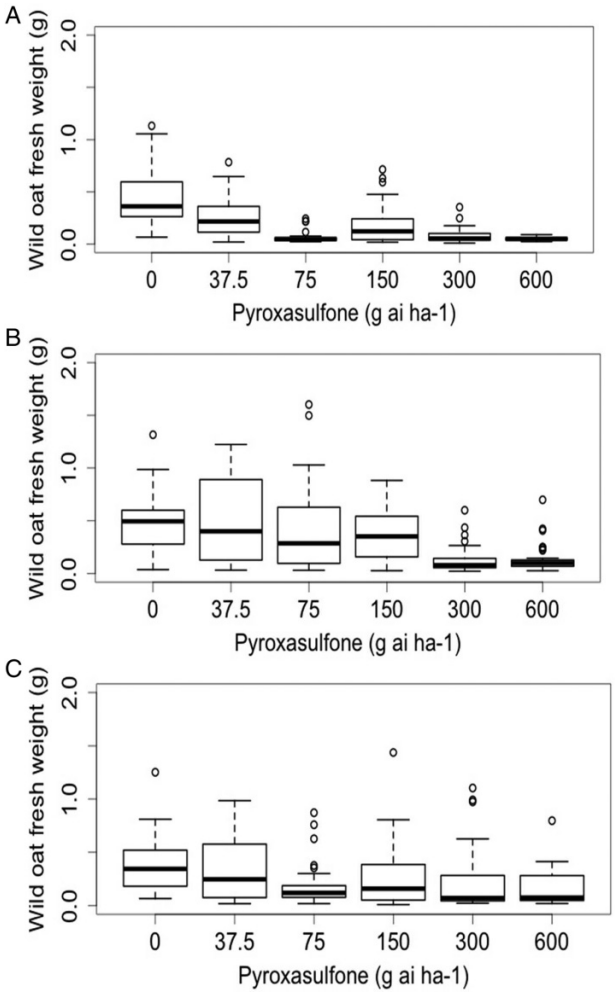
Fig. 1. Wild oat biotypes S1988, HR11-151, and HR08-210 survival in a dose response study to (A) triallate, (B) pyroxasulfone, and (C) sulfentrazone. Survival was calculated by live plants divided by the total number of germinated seeds in each pot.



of 1.13 ($P > 0.05$) for pyroxasulfone and 1.28 ($P > 0.05$) for sulfentrazone (Fig. 1).

Pyroxasulfone and sulfentrazone are both soil-applied pre-emergent herbicides, but have different target sites. The wild oat populations tested have never been exposed to pyroxasulfone or sulfentrazone, so the possibility of target-site mutations conferring multiple-resistance mechanisms for these three herbicides is unlikely. Cross-resistance between triallate and pyroxasulfone in annual ryegrass is believed to be due to increased metabolism because cross-resistance was not observed for herbicides not metabolized by wheat (Busi and Powles 2013). The resistance mechanism for triallate-resistant wild oat populations is believed to be increased levels of endogenous gibberellin (Rashid et al. 1998). Increased endogenous gibberellin would allow for rapid germination and meristematic growth, allowing plants to avoid toxic levels of herbicide from reaching its target site (O'Donovan et al. 1999). It is probable that an increased level of endogenous gibberellin in triallate

Fig. 2. Fresh weight per plant 28 d after treatment of wild oat populations (A) S1988, (B) HR11-151, and (C) HR08-210, 28 d after treatment with increasing rates of pyroxasulfone (N = 60).



resistant wild oat population are allowing seedlings to avoid toxic effects of both soil-active herbicides pyroxasulfone and sulfentrazone. Alternatively, resistance to pyroxasulfone and (or) sulfentrazone could be conferred by enhanced metabolism by P450 monoxygenases selected for by previous use of ACCase and ALS inhibitors.

When the resistant populations were treated with higher rates of triallate, pyroxasulfone, and sulfentrazone, a few individuals appeared unaffected (Fig. 2). Although LD_{50} values suggest that the overall resistance level was low in HR11-151 (R/S = 2.78) and not significantly different in HR08-210 relative to the S1988 population (Table 3), the presence of some individuals unaffected by herbicide within the two HR populations suggest that further selection for resistance may be rapid. These populations will be further selected by pyroxasulfone to determine if variance was due to random error or to genetic differences in the population. Rapid selection of resistance to pyroxasulfone in annual ryegrass has been reported previously by Busi et al. (2012).

This is the first report of a grass species resistant to pyroxasulfone or sulfentrazone in North America. However, resistance to triallate, ACCase inhibitors, and ALS inhibitors is widespread in wild oat and some other grasses (Heap 2015). Research is required to determine if triallate-resistant wild oat commonly exhibits resistance to these herbicides having different sites of action. If it is a common occurrence, cross-resistance to pyroxasulfone and sulfentrazone will further limit wild oat control options in the Canadian Prairies. In response to widespread herbicide resistance in wild oat to ACCase and ALS inhibitors, producers are looking to newer herbicides of differing sites of action, such as pyroxasulfone and sulfentrazone. Alternatively, they may return to older soil-applied products such as triallate or trifluralin. Nevertheless, this research suggests that alternative herbicides will likely have a short utility in controlling this important grass weed due to the prevalence of non-target-site resistance mechanisms conferring resistance across multiple herbicide sites of action. Knowledge of cross-resistance patterns is important to facilitate management of herbicide-resistant wild oat populations. Repeated selection of the experimental populations is underway to quantify the rate of selection for pyroxasulfone resistance in these wild oat populations.

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