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Protoporphyrinogen oxidase (PPO)-inhibitor resistance in kochia (*Bassia scoparia*)

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Abstract

Kochia [*Bassia scoparia* (L.) A.J. Scott] is an invasive tumbleweed in the North American Great Plains that is difficult to manage in croplands and ruderal areas due to widespread resistance to up to four herbicide sites of action, including auxin mimics (Herbicide Resistance Action Committee [HRAC] Group 4) and inhibitors of acetolactate synthase (HRAC Group 2), photosystem II (HRAC Group 5), and 5-enolpyruvylshikimate-3-phosphate synthase (HRAC Group 9). Poor *B. scoparia* control with protoporphyrinogen oxidase (PPO)-inhibiting (HRAC Group 14) herbicides was noted in a brown mustard [*Brassica juncea* (L.) Czern.] field near Kindersley, SK, Canada, in 2021. Similar observations were made in a sunflower (*Helianthus annuus* L.) field near Mandan, ND, USA, and in research plots near Minot, ND, USA, in 2022. Whole-plant dose–response experiments were conducted to determine whether these *B. scoparia* accessions were resistant to the PPO-inhibiting herbicides saflufenacil and carfentrazone and the level of resistance observed. All three *B. scoparia* accessions were highly resistant to foliar-applied saflufenacil and carfentrazone compared with two locally relevant susceptible accessions. The Kindersley accession exhibited 57- to 87-fold resistance to saflufenacil and 97- to 121-fold resistance to carfentrazone based on biomass dry weight at 21 d after treatment (DAT). Similarly, the Mandan accession exhibited 204- to 321-fold resistance to saflufenacil and 111- to 330-fold resistance to carfentrazone, while the Minot accession exhibited 45- to 71-fold resistance to saflufenacil and 88- to 264-fold resistance to carfentrazone. Substantial differences in visible control at 7 and 21/28 DAT were also observed between the putative-resistant and susceptible accessions. This study represents the first confirmations of PPO inhibitor–resistant *B. scoparia* globally and the fifth herbicide site of action to which *B. scoparia* has evolved resistance. It also documents this issue present at three locations in the Northern Great Plains region that occur up to 790 km apart and on both sides of the Canada/U.S. border.

Introduction

Kochia [*Bassia scoparia* (L.) A.J. Scott] is a tumbleweed that is native to Eurasia and was introduced to North America as an ornamental plant in the mid- to late-1800s (Friesen et al. 2009). *Bassia scoparia* can be an invasive and troublesome weed in cropping systems, pastureland, and ruderal areas due to its summer annual life cycle, early seedling emergence (Kumar et al. 2018; Schwinghamer and Van Acker 2008), abiotic stress tolerance (Friesen et al. 2009), competitiveness (Geddes and Sharpe 2022), prolific seed production (Beckie et al. 2016), and short-lived seed persistence in both aerial (Geddes and Pittman 2023) and soil seedbanks (Beckie et al. 2018; Dille et al. 2017; Schwinghamer and Van Acker 2008). High genetic diversity (Martin et al. 2020) combined with efficient pollen- and seed-mediated gene flow (Beckie et al. 2016) cause rapid evolution of *B. scoparia* in response to management practices. Herbicides remain the primary method used to manage this weed, and in response, *B. scoparia* populations have evolved resistance to up to four herbicide sites of action (Beckie et al. 2019; Varanasi et al. 2015).

Multiple herbicide–resistant *B. scoparia* is a widespread issue throughout the Great Plains of North America (Kumar et al. 2019), where it can cause substantial crop yield losses if left unmanaged (Geddes and Sharpe 2022). *Bassia scoparia* was the most-abundant herbicide-resistant broadleaf weed in a 2019/2020 survey of Saskatchewan, where uncontrolled populations occupied an estimated 39% of annual-cropped fields (Geddes et al. 2024). Herbicide resistance in *B. scoparia*

Table 1. Recent herbicide use history in the Kindersley, SK, Canada, field where protoporphyrinogen oxidase (PPO) inhibitor–resistant *Bassia scoparia* was confirmed in 2021.

Application timing					Herbicide information	
Year	Month	Day	Window[a]	Crop[b]	Common name[c]	Rate
						g ai/ae ha^{-1}
2019			PRE	Flax	Glyphosate + sulfentrazone	890 + 113
2019			POST	Flax	Bromoxynil + MCPA	278 + 278
2020			PRE	Chickpea	Glyphosate + sulfentrazone	890 + 113
2021	May	06	PRE	Brown mustard	Glyphosate + sulfentrazone + carfentrazone	890 + 75 + 18
2021	May	16	PRE	Brown mustard	Glyphosate + carfentrazone + bromoxynil	890 + 9 + 70

[a] POST, postemergence; PRE, preemergence.

[b] Brown mustard, *Brassica juncea* (L.) Czern.; chickpea, *Cicer arietinum* L.; flax, *Linum usitatissimum* L.

[c] PPO-inhibiting active ingredients are underlined.

dates back to 1976, when photosystem II (PSII) inhibitor (Herbicide Resistance Action Committee [HRAC] Group 5) resistance was reported in Kansas (Heap 2024). *Bassia scoparia* resistant to acetolactate synthase (ALS)-inhibiting (HRAC Group 2) herbicides was reported first in Kansas and North Dakota in 1987. ALS inhibitor–resistant *B. scoparia* is widespread and was present in all survey samples tested in Canada in recent decades (Beckie et al. 2015; Hall et al. 2014). *Bassia scoparia* with resistance to auxin mimics (HRAC Group 4) was reported first in Montana in 1993/1994 and only recently in Canada since 2015 (Beckie et al. 2019; Cranston et al. 2001; Geddes et al. 2022a, 2022b, 2022c, 2023; Heap 2024). *Bassia scoparia* resistant to glyphosate (HRAC Group 9) was first documented in Kansas in 2007 and later in multiple states and provinces (Beckie et al. 2013; Hall et al. 2014; Heap 2024). After only a single decade since the first report of glyphosate-resistant *B. scoparia* in Canada (2011), this biotype was present in about three-quarters of *B. scoparia* samples tested ($n = 889$) between 2018 and 2021 (Geddes et al. 2022c, 2023; Sharpe et al. 2023).

Interest in protoporphyrinogen oxidase (PPO)-inhibiting herbicides has grown recently, due in part to the continued evolution and spread of glyphosate-resistant weeds (Barker et al. 2023; Dayan et al. 2018). Herbicides targeting this site of action have been commercialized for more than a half century, despite their mechanism of action only being elucidated in recent decades (Matringe et al. 1989a, 1989b). In susceptible plants, PPO inhibitors cause chlorosis, wilting, and necrosis; they have been referred to colloquially as bleaching or peroxidizing herbicides. Following plant uptake, the PPO-inhibiting active ingredient enters photosynthetically active parenchyma cells, where it inhibits PPO isoforms, PPO1 and PPO2, located in the chloroplast. Protogen is then leaked into the cytoplasm, where it is converted to photodynamic protoporphyrin IX (proto). Proto generates a flush of reactive oxygen species under light, which ultimately causes membrane lipid peroxidation (Barker et al. 2023). There are currently 21 unique herbicide active ingredients commercialized, spanning four different chemical families, that inhibit PPO (HRAC 2024b).

Rapid evolution and spread of multiple herbicide resistance traits in *B. scoparia*, and widespread glyphosate resistance in particular, resulted in greater reliance on PPO-inhibiting herbicides for *B. scoparia* control. Several studies document excellent foliar- and soil-applied activity of PPO inhibitors on *B. scoparia* (Kumar and Jha 2015; Torbiak et al. 2021a, 2021b, 2022, 2024; Yadav et al. 2020). For example, preemergence sulfentrazone (105 g ai ha^{-1}) controlled glyphosate-resistant *B. scoparia* in spring wheat (*Triticum aestivum* L.) by 95% to 99% 3 wk after postemergence herbicides were applied (Torbiak et al. 2021b). Carfentrazone + sulfentrazone (9 + 105 g ai ha^{-1}) applied preemergence controlled glyphosate- and ALS inhibitor–resistant *B. scoparia* in field pea (*Pisum sativum* L.) by 94% on average 3 wk after the postemergence herbicide treatment timing (Torbiak et al. 2022). In chemical fallow, glyphosate (450 g ae ha^{-1}) mixed with saflufenacil (18 or 50 g ai ha^{-1}), carfentrazone (18 g ai ha^{-1}), or carfentrazone + sulfentrazone (9 + 53 or 9 + 105 g ai ha^{-1}) resulted in ≥90% control of glyphosate-resistant *B. scoparia* in Alberta (Torbiak et al. 2021a). In Montana, saflufenacil (25 g ai ha^{-1}) applied postemergence controlled *B. scoparia* by 90% 1 wk after treatment (WAT), which decreased to 67% by 5 WAT absent crop interference (Kumar and Jha 2015). Glyphosate + sulfentrazone (1,261 + 210 g ae/ai ha^{-1}) resulted in near-complete control and 97% to 100% biomass reduction of *B. scoparia* in glyphosate/dicamba-resistant soybean [*Glycine max* (L.) Merr.] grown in Montana and Kansas (Yadav et al. 2020). Excellent *B. scoparia* control with PPO-inhibiting herbicides resulted in extensive adoption of glyphosate and PPO-inhibiting herbicide mixtures to control glyphosate-resistant *B. scoparia* before crop planting in the conservation tillage systems that dominate the Great Plains region. However, due to widespread glyphosate resistance in this species, this resulted in only a single herbicide site of action with sufficient activity on *B. scoparia*. When this is combined with widespread ALS-inhibitor resistance in *B. scoparia* resulting in no effective postemergence herbicides in many pulse crops grown in the region, and auxinic herbicide resistance limiting postemergence weed control in small grain cereals, heavy reliance on PPO inhibitors for *B. scoparia* control could increase risk of selection for PPO-inhibitor resistance (Sharpe and Novek 2024).

Poor control of *B. scoparia* with PPO-inhibiting herbicides was identified in a brown mustard [*Brassica juncea* (L.) Czern.] field located near Kindersley, SK, Canada, in 2021. Glyphosate and sulfentrazone either alone or mixed with carfentrazone were applied preemergence during the previous three growing seasons (Table 1). Similarly, poor *B. scoparia* control with carfentrazone + sulfentrazone was noted in a sunflower (*Helianthus annuus* L.) field near Mandan, ND, USA, and in research plots near Minot, ND, USA, in 2022 (Table 2). The objectives of this research were to determine (1) if the *B. scoparia* accessions collected from Saskatchewan and North Dakota were resistant to the foliar-applied PPO-inhibiting herbicides saflufenacil and carfentrazone, and (2) if so, the level of resistance observed.

Materials and Methods

Plant Material

Mature seeds from at least 20 uncontrolled *B. scoparia* plants were collected at random from the fields of interest (Figure 1). The

Table 2. Recent protoporphyrinogen oxidase (PPO)-inhibiting herbicide use in the Mandan and Minot, ND, USA, fields where PPO inhibitor–resistant *Bassia scoparia* was confirmed in 2022.

	Mandan		Minot
Year	Crop[a]	Herbicide common name	Herbicide common name[b]
2016	Wheat		
2017	Corn		Carfentrazone; flumioxazin; saflufenacil; sulfentrazone
2018	Sunflower	Carfentrazone + sulfentrazone	
2019	Wheat		Saflufenacil; sulfentrazone
2020	Field pea	Carfentrazone + sulfentrazone	Sulfentrazone
2021	Wheat		Carfentrazone; flumioxazin; sulfentrazone
2022	Sunflower	Carfentrazone + sulfentrazone	Saflufenacil; sulfentrazone

[a] Corn, *Zea mays* L.; field pea, *Pisum sativum* L.; sunflower *Helianthus annuus* L.; wheat, *Triticum aestivum* L

[b] These herbicides were applied in research plots in various parts of the field, and research plots were moved around the field each year. The rest of the field was typically seeded to wheat where no PPO-inhibiting herbicides were used.

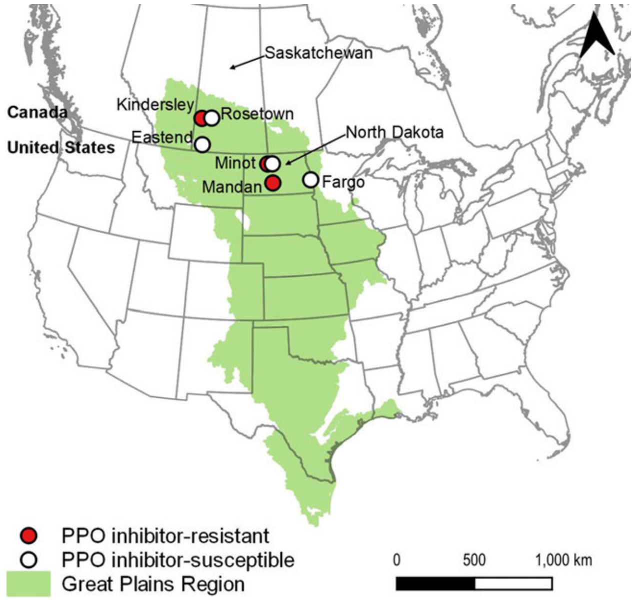

Figure 1. Map of Canada and the United States showing the collection locations of the protoporphyrinogen oxidase (PPO) inhibitor–resistant *Bassia scoparia* accessions and the susceptible control accessions used for the first confirmations of PPO inhibitor–resistant *Bassia scoparia* in 2021 and 2022. Collection locations are adjusted to the nearest city or town.

Saskatchewan fields were sampled in October 2021 and the North Dakota fields were sampled in October 2022. The putative-resistant sample from Saskatchewan was collected from a field planted to brown mustard near Kindersley, SK, and designated "KindersleyR" (coordinates not provided to protect farmer identity). Two susceptible control accessions were also collected, one from a field near Eastend, SK (hereafter "EastendS") and another being a lab-maintained ALS inhibitor–resistant, but glyphosate- and auxin mimic–susceptible control collected near Rosetown, SK (hereafter "RosetownS"). Two previously collected accessions were used as susceptible controls in the North Dakota experiments. A field near Fargo, ND (hereafter "FargoS") was sampled in 2012, and a field in Minot, ND (hereafter "MinotS") was sampled in approximately 2010. The putative-resistant accessions collected near Mandan and Minot, ND, in 2022 were designated "MandanR" and "MinotR", respectively.

The field-collected samples from Saskatchewan were subjected initially to single-dose screening with saflufenacil (Heat® LQ, BASF Canada, Mississauga, ON, Canada) at 50 g ai ha^{-1}. The single-dose screening was unreplicated and consisted of three *B. scoparia* accessions (KindersleyR, EastendS, and RosetownS) and two herbicide regimes (treated and untreated). The *B. scoparia* accessions were planted in 24 by 24 by 5 cm greenhouse flats filled with Cornell soilless potting medium (Sheldrake and Boodley 1966) targeting 40 plants flat^{-1}. The flats were placed in the greenhouse at the Agriculture and Agri-Food Canada Lethbridge Research and Development Centre where they were watered daily. The greenhouse followed a 20/18 C day/night temperature regime with 16-h photophase and 8-h scotophase. Fluence RAZR 3 light-emitting diode bulbs (Fluence, Austin, TX, USA) provided 230 µmol m^{-2} s^{-1} supplemental light. The herbicide was applied at 5- to 8-cm plant height using a moving-nozzle cabinet sprayer with a TeeJet® (Wheaton, IL, USA) flat-fan 8002VS nozzle calibrated to deliver 200 L ha^{-1} spray solution at 275 kPa when traveling at 2.4 km h^{-1}. To limit the potential impact of parental environment on the phenotypic expression of resistance and to demonstrate transfer of the resistance trait to subsequent generations (HRAC 2024a), survivors from the treated KindersleyR accession and untreated EastendS and RosetownS accessions were transplanted separately into larger containers 21 d after treatment (DAT) and grown for seed under pollination bags created from 10-µm nylon mesh (Miami Aqua-culture, Boynton Beach, FL, USA). Four surviving plants from each accession were placed under a pollination bag where they were allowed to cross-pollinate within each accession to avoid inbreeding depression. The second-generation seeds were hand harvested and threshed, and the seed was stored at 4 C until used for the dose–response experiments. The North Dakota accessions (MandanR, MinotR, FargoS, and MinotS) were field-collected samples that were not regenerated under controlled-environment conditions.

Dose Response

Separate dose–response bioassays were conducted for the Saskatchewan (KindersleyR, EastendS, and RosetownS) and North Dakota (MandanR, MinotR, FargoS, and MinotS) accessions in the greenhouses at the Lethbridge Research and Development Centre and North Dakota State University, respectively. Each experiment included either one (Saskatchewan) or two (North Dakota) putative PPO inhibitor–resistant *B. scoparia* accessions and two locally relevant susceptible control accessions. At each location, separate dose–response experiments were conducted for saflufenacil and carfentrazone (Aim® EC, FMC of Canada, Mississauga, ON, Canada) and were repeated once. Each experiment followed a factorial randomized complete block design in which the first factor consisted of *B. scoparia* accession and the second factor was herbicide rate. The rate structure followed 0 (untreated), 0.01, 0.1, 1, 3.16, 10, 31.6, and 100× the U.S. field rate for each active ingredient (25 g ai ha^{-1} for saflufenacil and 17.5 g ai ha^{-1} for carfentrazone; Ikley et al. 2024). The Saskatchewan accession experiments (Figure 2) included nine blocks consisting of one *B. scoparia* plant in each 10 by 10 cm plastic greenhouse pot. The Saskatchewan experiments used the same potting medium, growth environment,

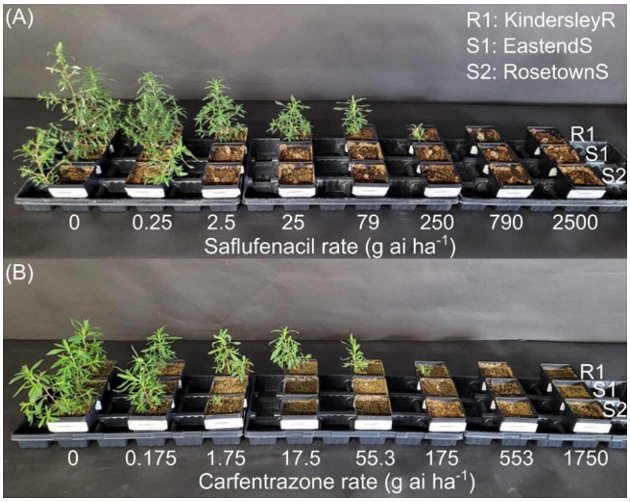

Figure 2. One replicate of the (A) saflufenacil and (B) carfentrazone dose–response experiments at 21 d after treatment (DAT) for one putative-resistant (KindersleyR) and two susceptible (RosetownS and EastendS) *Bassia scoparia* accessions from Saskatchewan, Canada.

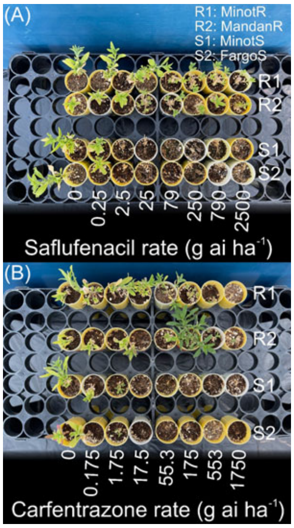

Figure 3. One replicate of the (A) saflufenacil at 28 d after treatment (DAT) and (B) carfentrazone at 21 DAT dose–response experiments for two putative-resistant (MandanR and MinotR) and two susceptible (MinotS and FargoS) *Bassia scoparia* accessions from North Dakota, USA.

and herbicide treatment methodology described previously. The North Dakota accession experiments (Figure 3) included 10 blocks consisting of one *B. scoparia* plant in each 4-cm diameter by 21-cm deep Cone-tainer™ (Stuewe & Sons, Tangent, OR, USA) filled with four parts potting soil (Promix BX, Premier Horticulture, Quakertown, PA, USA) and one part sandy loam soil. In the North Dakota experiments, plants were treated when they reached 2 to 3 cm in height using a moving-nozzle cabinet sprayer equipped with a TeeJet® XR 8002E nozzle calibrated to deliver 140 L ha^{-1} spray solution at 207 kPa when traveling 5.4 km h^{-1}. Ammonium sulfate and methylated seed oil were added to the spray solution at 10 g L^{-1} and 1% v/v, respectively. Greenhouse temperatures were maintained between 24 and 27 C with a 16-h photophase and 8-h scotophase supplemented with light from 1,000-W high-pressure sodium lamps (P.L. Light Systems, Beamsville, ON, Canada). All experiments were watered from above daily and did not receive fertilization other than that provided by the potting medium.

The *B. scoparia* measurements included visible control at 7 and 21 DAT and shoot biomass fresh weight (FW) and dry weight (DW) at 21 DAT; save for the North Dakota saflufenacil experiments that included visible control, FW, and DW measurements at 28 DAT. *Bassia scoparia* visible control was estimated as a percentage from 0% to 100% control relative to the untreated control within each accession and block following the rating scale reported by the Canadian Weed Science Society (2018). *Bassia scoparia* shoot biomass was determined by harvesting all living and dead tissue above the soil surface and weighing (FW), followed by drying in an oven at 60 C until constant weight and weighing again (DW). Both biomass FW and DW were included as response variables to account for the impacts of dead *B. scoparia* tissue at high herbicide rates on the dose–response relationship due to differential moisture retention between living and dead plant tissue.

Statistical Analyses

The experiments using the Saskatchewan and North Dakota *B. scoparia* accessions were analyzed separately following the same two-stage procedure including ANOVA followed by nonlinear regression. Visible control (at 7 and 21/28 DAT) and biomass (FW and DW at 21/28 DAT) data were subjected to ANOVA using PROC MIXED in SAS software v. 9.4 (SAS Institute, Cary, NC, USA). *Bassia scoparia* accession, herbicide rate, experimental run, and their interactions were considered fixed factors, while block nested within run was considered a random factor. The model assumptions were assessed using PROC UNIVARIATE based on the Shapiro-Wilk statistic and by plotting the residuals and fitted values (Littell et al. 2006). Variance component analyses were used to determine the percentage of total model sums of squares allocated to each factor. All main and interaction effects including experimental run accounted for <5% of the total sums of squares, and this factor was therefore removed from the final analysis after confirming homogeneous variance across runs.

The *B. scoparia* visible control (at 7 and 21/28 DAT) and biomass (FW and DW at 21/28 DAT) data were analyzed using nonlinear regression in the DRC package of R v. 4.3.1 (R Core Team 2023). The analysis used the three-parameter log-logistic function (Equation 1)

$$y = \frac{d}{1 + \exp\{b[\log(x) - \log(e)]\}} \qquad [1]$$

where y is the response variable, d is the upper asymptote, b is the slope of the regression line at dose e, e is the regression line inflection point, and x is the herbicide rate (in g ai ha^{-1}) (Ritz et al. 2015). A four-parameter log-logistic function was fit initially, but the lower asymptote did not differ from zero ($\alpha = 0.05$), and so a common lower asymptote was fit based on model parsimony. A similar approach was taken to fit a common upper asymptote when modeling visible control data only when the upper asymptote for each regression curve did not differ from 100% control, in which case the d parameter was fit individually for each *B. scoparia* accession. The *ED* and *EDcomp* functions were used to determine herbicide effective doses resulting in 50% and 80% visible control (ED_{50} and ED_{80}, respectively) or biomass reduction (GR_{50} and

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Table 3. Saflufenacil and carfentrazone resistance indices for one putative protoporphyrinogen oxidase (PPO) inhibitor–resistant *Bassia scoparia* accession collected from Saskatchewan in 2021 and two putative PPO inhibitor–resistant *Bassia scoparia* accessions collected from North Dakota in 2022 compared with two locally relevant susceptible control accessions based on visible control at 7 and 21/28 d after treatment (DAT) and biomass fresh weight (FW) and dry weight (DW) at 21/28 DAT.

Province/state	Herbicide	Accession	Response variable	R/S1[a,b]	R/S2[a,c]
Saskatchewan	Saflufenacil	KindersleyR	Visible control 7 DAT	47.4***	46.9***
		KindersleyR	Visible control 21 DAT	56.5	101.1***
		KindersleyR	Biomass FW 21 DAT	63.4***	82.2***
		KindersleyR	Biomass DW 21 DAT	57.0***	87.2***
	Carfentrazone	KindersleyR	Visible control 7 DAT	45.5***	48.1***
		KindersleyR	Visible control 21 DAT	38.0***	24.3***
		KindersleyR	Biomass FW 21 DAT	70.6**	72.8*
		KindersleyR	Biomass DW 21 DAT	120.9**	97.0*
North Dakota	Saflufenacil	MandanR	Visible control 7 DAT	292.8	294.8
		MandanR	Visible control 28 DAT	460.6	419.8
		MandanR	Biomass FW 28 DAT	158.9**	164.2*
		MandanR	Biomass DW 28 DAT	204.0*	320.5*
		MinotR	Visible control 7 DAT	355.6	358.1
		MinotR	Visible control 28 DAT	504.1	459.5
		MinotR	Biomass FW 28 DAT	38.3*	39.6*
		MinotR	Biomass DW 28 DAT	45.4*	71.3*
	Carfentrazone	MandanR	Visible control 7 DAT	292.1	180.4
		MandanR	Visible control 21 DAT	1008.1*	515.7*
		MandanR	Biomass FW 21 DAT	506.8	149.8
		MandanR	Biomass DW 21 DAT	330.1	110.5
		MinotR	Visible control 7 DAT	166.4***	102.8***
		MinotR	Visible control 21 DAT	210.9***	107.9**
		MinotR	Biomass FW 21 DAT	336.4	99.5
		MinotR	Biomass DW 21 DAT	264.1	88.4

[a] Significant difference of the resistance index from unity at $^*P < 0.05$; $^{**}P < 0.01$; $^{***}P < 0.001$.

[b] R/S1 indicates the resistance index relative to the first susceptible control accession; EastendS for Saskatchewan and FargoS for North Dakota.

[c] R/S2 indicates the resistance index relative to the second susceptible control accession; RosetownS for Saskatchewan and MinotS for North Dakota.

GR$_{80}$, respectively) and compare among them ($\alpha = 0.05$). The effective doses for visible control were determined relative to the limits of 0% and 100% control, while the effective doses for biomass were determined relative to the predicted biomass for the untreated control within each *B. scoparia* accession. The resistance index was calculated by dividing the ED$_{50}$ or GR$_{50}$ value for each putative-resistant accession by that for each corresponding susceptible control accession. The putative PPO inhibitor–resistant *B. scoparia* accessions were considered to be highly resistant if the resistance index was ≥10 (HRAC 2024a).

Results and Discussion

The putative PPO inhibitor–resistant *B. scoparia* accessions from Saskatchewan (KindersleyR) and North Dakota (MandanR and MinotR) were highly resistant to foliar-applied saflufenacil and carfentrazone. These represent the first reports of PPO inhibitor–resistant *B. scoparia* globally and that the issue was present in the Northern Great Plains region at sites located up to 790 km apart and on either side of the Canada/U.S. border (Figure 1).

Saflufenacil Resistance

Saskatchewan

The *B. scoparia* accession collected near Kindersley, SK, Canada, in 2021 was highly resistant to foliar-applied saflufenacil. KindersleyR exhibited 57.0- to 87.2-fold resistance to saflufenacil based on biomass DW (Table 3; Figures 2 and 4). The differential response of KindersleyR to saflufenacil compared with the susceptible control accessions was evident visually by 7 DAT and extended to at least 21 DAT (Table 3; Figure 4). Visible control resistance indices ranged from 46.9- to 47.4-fold resistance at 7 DAT and increased to 56.5- to 101.1-fold resistance by 21 DAT (Table 3; Figure 4). The estimated rate of saflufenacil causing 80% reduction in biomass DW was 126.9 g ai ha^{-1} (Table 4). This was well above the high field rate registered in western Canada (50 g ai ha^{-1}) (Anonymous 2024a, 2024b). It was also >125-fold greater than the saflufenacil rate causing 80% decline in biomass DW for the susceptible control accessions, EastendS and RosetownS (0.8 and 1.0 g ai ha^{-1}, respectively).

North Dakota

The *B. scoparia* accessions collected near Mandan and Minot, ND, USA, in 2022 were highly resistant to saflufenacil, similar to the KindersleyR accession. The saflufenacil resistance indices for MinotR were about one-quarter that of MandanR. For example, MandanR exhibited 204.0- to 320.5-fold resistance, while MinotR exhibited 45.4- to 71.3-fold resistance to foliar-applied saflufenacil based on biomass DW (Table 3; Figures 3 and 5). While both accessions were highly resistant based on guidelines recommended by the Global HRAC (HRAC 2024a), the difference in resistance indices between these two accessions was due to very low GR$_{50}$ values for the susceptible control accessions that were ≤0.7 g ai ha^{-1} of saflufenacil (Table 5). Like the Saskatchewan accessions, differential response of the resistant from the susceptible North Dakota accessions was evident by 7 DAT and extended to at least 28 DAT (Table 3; Figure 5). However, resistance indices based on visible control ratings were not statistically different from 1 despite R/S ratios that were ≥292.8 (Table 3). This was due, in part, to variability around the dose–response model inflection point (Table 5), which could reflect the variable nature of the North Dakota field-collected samples that were absentregeneration under controlled environment or incomplete (70% to 72%) visible control

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Table 4. Regression parameter estimates for the three-parameter log-logistic model fit to describe the response of three Saskatchewan *Bassia scoparia* accessions to a rate titration of saflufenacil or carfentrazone based on visible control at 7 and 21 d after treatment (DAT) and shoot biomass fresh weight (FW) and dry weight (DW) at 21 DAT[a].

Herbicide	Response variable	Accession	*b* (±SE)	*d* (±SE)	*e* (±SE)	ED_{80} (±SE)
					g ai ha^{-1}	
Saflufenacil	Visible control 7 DAT	EastendS	−1.95 (0.21)		0.8 (0.1)	1.5 (0.2)
	RSE = 12.2	RosetownS	−1.98 (0.21)		0.7 (0.1)	1.5 (0.2)
		KindersleyR	−0.73 (0.05)		35.2 (3.6)	234.3 (33.9)
	Visible control 21 DAT	EastendS	−2.19 (0.47)		1.0 (0.2)	2.0 (0.3)
	RSE = 17.6	RosetownS	−6.66 (79.14)		1.9 (6.6)	2.3 (2.4)
		KindersleyR	−0.60 (0.05)		104.9 (16.3)	1,049.0 (239.7)
	Biomass FW 21 DAT	EastendS	1.78 (0.58)	8.0 (0.3)	0.3 (0.4)	0.7 (0.3)
	RSE = 1.2	RosetownS	2.03 (0.72)	6.4 (0.3)	0.4 (0.1)	0.9 (0.4)
		KindersleyR	0.83 (0.09)	7.0 (0.2)	27.5 (4.5)	147.6 (29.1)
	Biomass DW 21 DAT	EastendS	1.39 (0.33)	0.7 (0.1)	0.3 (0.1)	0.8 (0.2)
	RSE = 0.2	RosetownS	1.74 (0.53)	1.0 (0.1)	0.5 (0.1)	1.0 (0.4)
		KindersleyR	0.88 (0.10)	1.2 (0.1)	26.2 (4.2)	126.9 (25.0)
Carfentrazone						
	Visible control 7 DAT	EastendS	−1.12 (0.17)		1.1 (0.1)	3.9 (0.9)
	RSE = 15.4	RosetownS	−1.95 (0.73)		1.2 (0.2)	2.4 (0.4)
		KindersleyR	−0.93 (0.09)		55.0 (5.9)	241.0 (37.3)
	Visible control 21 DAT	EastendS	−1.05 (0.11)		4.3 (0.6)	16.3 (3.1)
	RSE = 16.3	RosetownS	−1.11 (0.13)		2.8 (0.4)	9.7 (2.1)
		KindersleyR	−1.11 (0.12)		105.3 (10.2)	365.8 (55.5)
	Biomass FW 21 DAT	EastendS	0.93 (0.24)	6.7 (0.5)	0.7 (0.3)	3.3 (1.5)
	RSE = 2.2	RosetownS	1.28 (0.32)	8.3 (0.5)	0.8 (0.2)	2.2 (0.6)
		KindersleyR	1.33 (0.42)	6.0 (0.3)	53.6 (12.8)	151.7 (48.8)
	Biomass DW 21 DAT	EastendS	1.01 (0.31)	0.6 (0.1)	0.75 (0.3)	3.0 (1.4)
	RSE = 0.2	RosetownS	1.16 (0.27)	0.8 (0.1)	0.6 (0.2)	2.0 (0.7)
		KindersleyR	1.80 (0.56)	0.6 (0.1)	73.2 (14.5)	157.9 (48.1)

[a] Abbreviations: *b*, slope of the response curve at inflection point; *d* upper asymptote; *e*, response curve inflection point considered ED_{50} for visible control or GR_{50} for biomass; ED_{80}, effective dose of herbicide resulting 80% visible control (ED_{80}) or biomass reduction (GR_{80}); RSE, residual standard error.

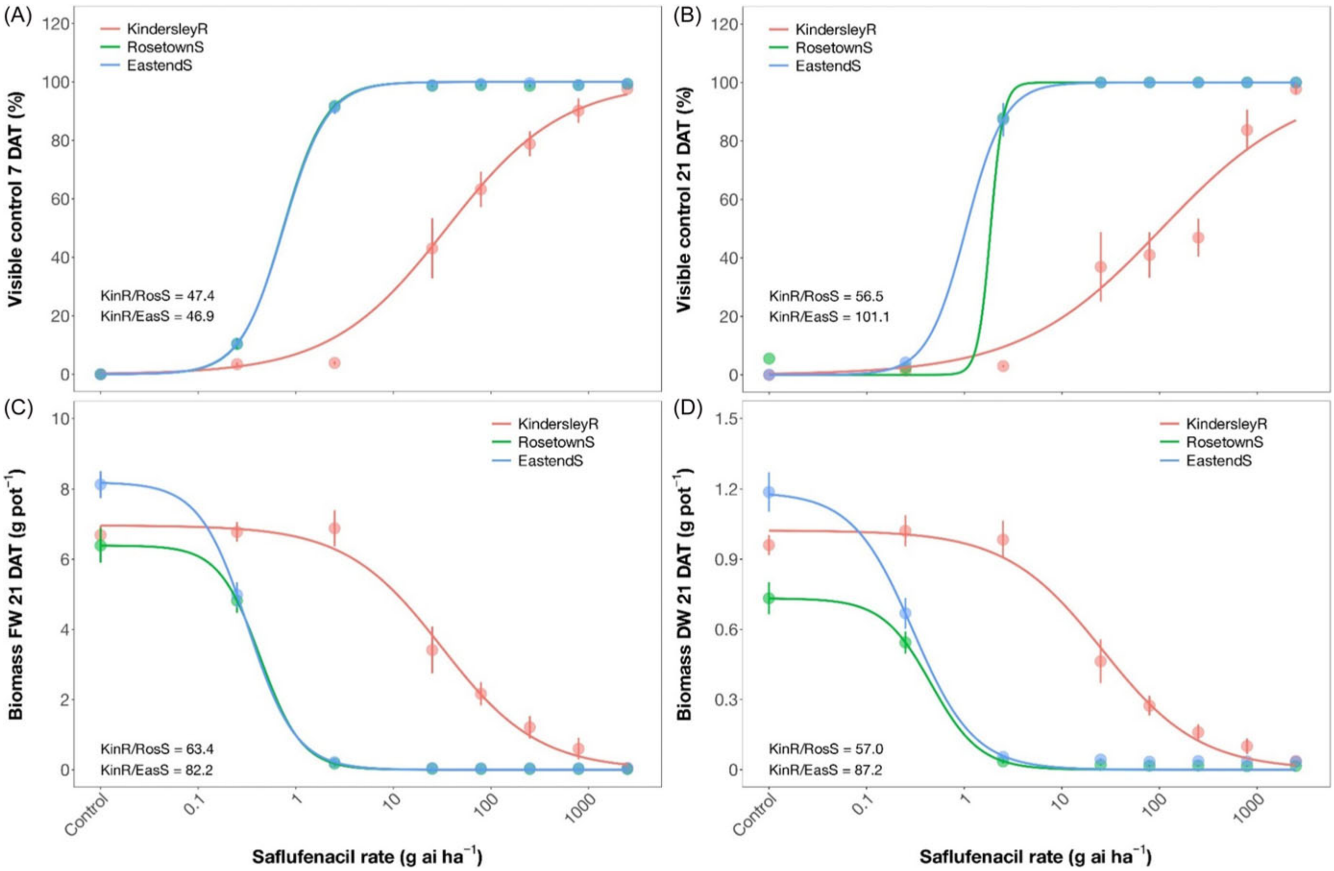

Figure 4. The response of one putative-resistant (KindersleyR) and two susceptible (RosetownS and EastendS) *Bassia scoparia* accessions from Saskatchewan, Canada, to a range of foliar-applied saflufenacil rates based on visible control at (A) 7 and (B) 21 d after treatment (DAT) and shoot biomass (C) fresh weight (FW) and (D) dry weight (DW) at 21 DAT. Dots indicate treatment means; bars represent standard errors. Embedded text indicates the resistance index (R/S ratio) for the putative-resistant accession relative to each susceptible accession.

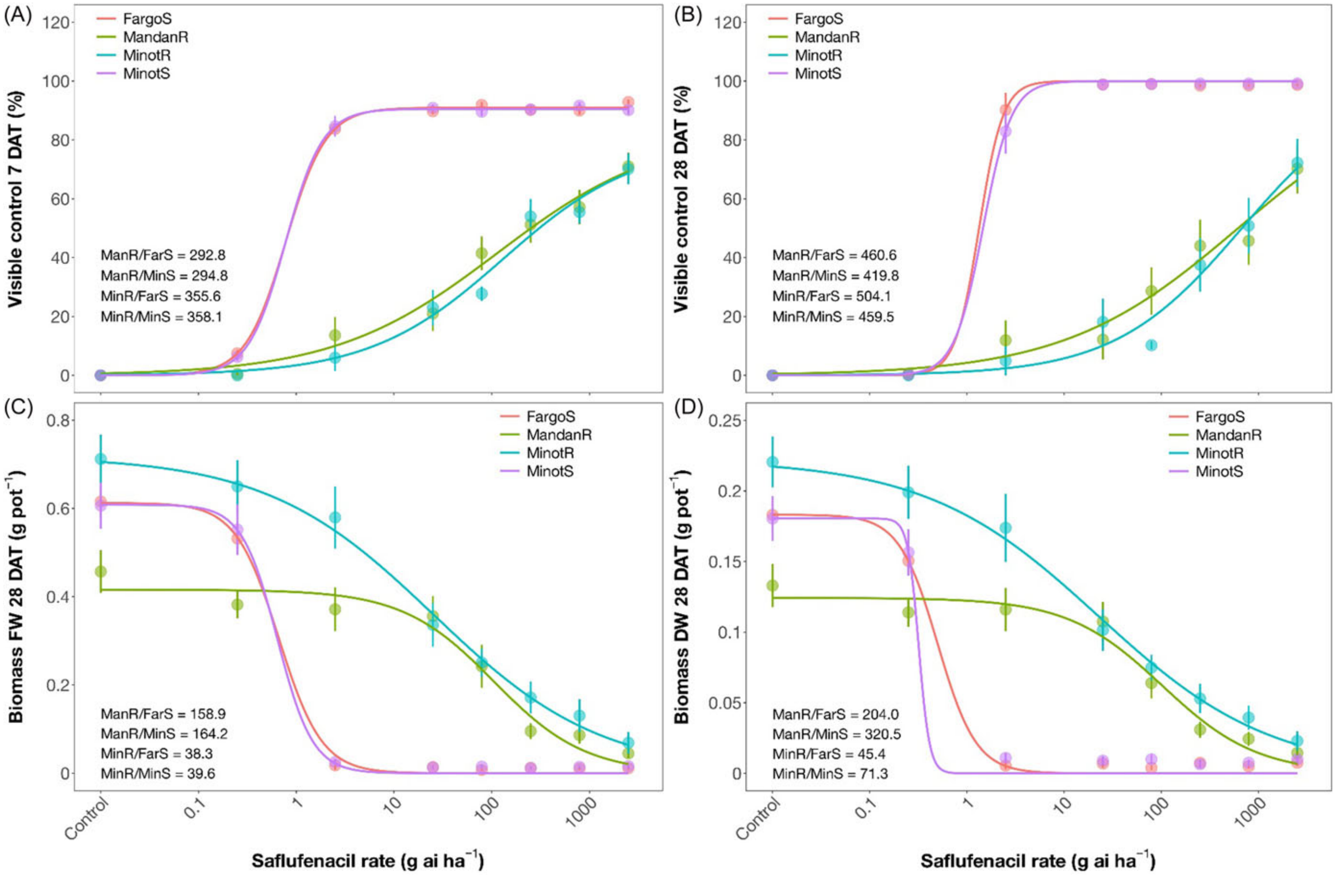

Figure 5. The response of two putative-resistant (MandanR and MinotR) and two susceptible (FargoS and MinotS) *Bassia scoparia* accessions from North Dakota, USA, to a range of foliar-applied saflufenacil rates based on visible control at (A) 7 and (B) 28 d after treatment (DAT) and shoot biomass (C) fresh weight (FW) and (D) dry weight (DW) at 28 DAT. Dots indicate treatment means; bars represent standard errors. Embedded text indicates the resistance index (R/S ratio) for the putative-resistant accession relative to each susceptible accession.

of the resistant accessions at the highest saflufenacil rate (2,500 g ai ha^{-1}) (Figure 5). Mean visible control at 28 DAT for the susceptible accessions increased from 3% at 0.25 g ha^{-1} of saflufenacil to 88% at 2.5 g ai ha^{-1}, also contributing to inaccurate estimation of the ED_{50} values and no statistical difference when determining the visible control resistance indices. The estimated saflufenacil rate causing 80% reduction in biomass DW was 485.6 and 387.8 g ai ha^{-1} for MandanR and MinotR compared with 0.9 and 0.4 g ai ha^{-1} for FargoS and MinotS (Table 5), about 16 to 18 times the typical U.S. burndown rate of 25 g ai ha^{-1} (Ikley et al. 2024).

Carfentrazone Resistance

Saskatchewan

The KindersleyR accession was highly resistant to foliar-applied carfentrazone. KindersleyR exhibited 97.0- to 120.9-fold resistance to carfentrazone based on biomass DW, compared with the two susceptible control accessions (Table 3; Figures 2 and 6). Like the response to saflufenacil, differential response to carfentrazone was obvious by 7 DAT and extended to 21 DAT (Table 3; Figure 6). The estimated carfentrazone rate causing 80% reduction in biomass DW of KindersleyR was 157.9 g ai ha^{-1}, which was well above that for the susceptible accessions (2.0 to 3.0 g ai ha^{-1}) (Table 4) and 6 to 18 times the registered burndown field rates (9 to 28 g ai ha^{-1}) for carfentrazone in western Canada (Anonymous 2024a, 2024b).

North Dakota

The MandanR and MinotR accessions were also highly resistant to carfentrazone. For example, resistance indices based on biomass DW ranged from 110.5- to 330.1-fold for MandanR and from 88.4- to 264.1-fold for MinotR (Table 3; Figures 3 and 7). However, resistance indices based on GR_{50} values were not significantly different from 1 ($\alpha = 0.05$) for these North Dakota accessions (Table 3). Despite this, resistance indices based on visible control at 21 DAT were significantly different from 1 and ranged from 515.7- to 1,008.1-fold resistance for MandanR and from 107.0- to 210.9-fold resistance for MinotR. Like the response of these accessions to saflufenacil, high resistance indices but lack of significant differences from unity for some response variables and not others were caused by a combination of incomplete (50% to 76%) control of the resistant accessions at the highest carfentrazone rate (1,750 g ai ha^{-1}), high variability around the model inflection point, and natural variability in the first-generation field-collected samples (Table 5; Figure 7). Nevertheless, taken together, observations across response variables, accessions, and locations suggest that these putative PPO inhibitor–resistant *B. scoparia* accessions were highly resistant to carfentrazone (HRAC 2024a).

Untreated *B. scoparia* plants in the Saskatchewan experiments grew approximately 5 to 10 times larger than those in the North Dakota experiments (Figures 2–7), which may have contributed to the larger resistance indices observed in North Dakota than

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Table 5. Regression parameter estimates for the three-parameter log-logistic model fit to describe the response of four North Dakota *Bassia scoparia* accessions to a rate titration of saflufenacil or carfentrazone based on visible control at 7 and 21/28 d after treatment (DAT) and shoot biomass fresh weight (FW) and dry weight (DW) at 21/28 DAT[a].

Herbicide	Response variable	Accession	*b* (±SE)	*d* (±SE)	*e* (±SE)	ED_{80} (±SE)
					g ai ha^{-1}	
Saflufenacil	Visible control 7 DAT RSE = 15.3	FargoS	−2.11 (0.32)	91.0 (1.5)	0.8 (0.1)	2.0 (0.5)
		MinotS	−2.29 (0.39)	90.5 (1.5)	0.8 (0.2)	1.9 (0.5)
		MandanR	−0.52 (0.11)	83.4 (11.8)	115.8 (79.3)	50,969.0 (94,450.0)
		MinotR	−0.64 (0.13)	79.3 (9.6)	131.9 (66.5)	N/A
	Visible control 28 DAT RSE = 22.1	FargoS	−3.53 (7.81)		1.3 (1.9)	2.0 (1.1)
		MinotS	−2.94 (3.93)		1.5 (1.1)	2.3 (0.3)
		MandanR	−0.48 (0.06)		613.9 (137.4)	10,719.0 (5,407.1)
		MinotR	−0.66 (0.09)		671.9 (117.9)	5,423.6 (2,018.1)
	Biomass FW 28 DAT RSE = 0.2	FargoS	1.97 (0.42)	0.61 (0.03)	0.7 (0.2)	1.3 (0.4)
		MinotS	2.40 (0.81)	0.61 (0.03)	0.6 (0.2)	1.1 (0.5)
		MandanR	0.93 (0.28)	0.42 (0.02)	105.2 (27.8)	468.9 (211.1)
		MinotR	0.51 (0.06)	0.72 (0.03)	25.4 (7.6)	391.5 (124.5)
	Biomass DW 28 DAT RSE = 0.1	FargoS	2.19 (0.94)	0.18 (0.01)	0.5 (0.2)	0.9 (0.5)
		MinotS	7.74 (9.75)	0.18 (0.01)	0.3 (0.1)	0.4 (0.2)
		MandanR	0.89 (0.24)	0.12 (0.01)	102.1 (28.7)	485.6 (221.9)
		MinotR	0.49 (0.05)	0.22 (0.01)	22.7 (7.1)	387.8 (126.2)
Carfentrazone	Visible control 7 DAT RSE = 17.6	FargoS	−1.19 (0.19)	99.55 (1.92)	1.12 (0.2)	3.7 (0.8)
		MinotS	−1.46 (0.41)	99.12 (1.98)	1.81 (0.2)	4.8 (1.5)
		MandanR	−0.63 (0.20)	84.10 (17.47)	180.0 (144.5)	20,094.0 (44,566.0)
		MinotR	−1.09 (0.20)	92.59 (7.15)	162.4 (36.1)	889.0 (431.5)
	Visible control 21 DAT RSE = 23.8	FargoS	−1.50 (0.52)		1.9 (0.3)	4.9 (1.9)
		MinotS	−2.27 (0.80)		3.8 (1.1)	7.0 (3.4)
		MandanR	−0.46 (0.09)		1955.6 (788.1)	40,404.6 (37,105.2)
		MinotR	−1.05 (0.15)		409.2 (57.5)	1,532.0 (393.7)
	Biomass FW 21 DAT RSE = 0.3	FargoS	0.75 (0.29)	0.66 (0.06)	0.2 (0.1)	1.3 (0.9)
		MinotS	1.12 (0.45)	0.56 (0.06)	0.7 (0.3)	2.4 (1.4)
		MandanR	0.51 (0.12)	0.89 (0.05)	106.1 (45.1)	1,582.8 (795.7)
		MinotR	0.80 (0.15)	0.86 (0.04)	70.4 (20.0)	401.2 (130.2)
	Biomass DW 21 DAT *RSE* = 0.1	FargoS	0.73 (0.29)	0.18 (0.02)	0.2 (0.1)	1.6 (1.2)
		MinotS	1.11 (0.46)	0.16 (0.02)	0.7 (0.3)	2.5 (1.4)
		MandanR	0.54 (0.11)	0.25 (0.01)	78.6 (31.6)	1,040.3 (458.7)
		MinotR	0.76 (0.13)	0.24 (0.01)	62.9 (17.0)	390.0 (124.8)

[a]Abbreviations: *b*, slope of the response curve at inflection point; *d* upper asymptote; *e*, response curve inflection point considered ED_{50} for visible control or GR_{50} for biomass; ED_{80}, effective dose of herbicide resulting 80% visible control (ED_{80}) or biomass reduction (GR_{80}); RSE, residual standard error.

Saskatchewan (Table 3). These differences in biomass were caused by earlier herbicide treatment (2- to 3-cm height) and more restricted *B. scoparia* growth in the 4-cm-diameter Cone-tainers used in North Dakota compared with slightly later treatment (5- to 8-cm height) and larger (10 by 10 cm) pots used in Saskatchewan. The smaller size of the Cone-tainers may have restricted growth of the North Dakota plants and potentially also led to nutrient deficiency by 21/28 DAT. Indeed, the untreated plants grown in Saskatchewan appeared visually healthy at 21 DAT (Figure 2), while those at 21/28DAT in North Dakota appeared less so (Figure 3). Despite this, apparent stress to the *B. scoparia* plants in the North Dakota experiments did not seem to influence herbicide efficacy, as the effective doses for control of the susceptible accessions remained similar between experiments conducted at both locations (Tables 4 and 5; Figures 4–7). Interestingly, untreated plants from both susceptible accessions accumulated less biomass than the resistant accessions in the carfentrazone but not the saflufenacil experiments in North Dakota (Figures 5 and 7). This difference could be explained, in part, by the difference in timing of the biomass measurements in North Dakota, which took place at 28 DAT for saflufenacil and 21 DAT for carfentrazone, while greater heterogeneity of these field-collected samples likely also played a role.

To date, 17 different weed species have evolved resistance to PPO-inhibiting herbicides globally, and the majority of cases reporting PPO-inhibitor resistance in the international database also report resistance to other herbicide sites of action (i.e., cross- or multiple resistance) (Barker et al. 2023; Heap 2024). PPO inhibitor–resistant waterhemp [*Amaranthus tuberculatus* (Moq.) Sauer] was documented in Kansas in 2001, representing the first case of resistance to this site of action among weed species (Shoup et al. 2003). Since then, PPO inhibitor–resistant weeds have been documented in 10 countries worldwide and in a range of crop species (Heap 2024). *Bassia scoparia* represents the fourth and seventh weed species to evolve PPO-inhibitor resistance in Canada and the United States, respectively. It remains unknown whether the three PPO inhibitor–resistant *B. scoparia* accessions identified in the current study also exhibit resistance to other herbicide sites of action. This knowledge gap is one focal point of several new questions regarding PPO inhibitor–resistant *B. scoparia* that warrants further investigation.

Similar to cases of PPO-inhibitor resistance in some other weed species (Dayan et al. 2018), the resistant *B. scoparia* plants exhibited initial necrosis after foliar treatment with saflufenacil or carfentrazone followed by healthy new regrowth shortly thereafter (CMG and QDL, personal observations). The initial symptomology typical of foliar treatment with PPO-inhibiting herbicides could make field diagnostics difficult if scouting is conducted shortly after application. However, differential control of the resistant and susceptible accessions was evident visually by 7 DAT under controlled-environment conditions (Figures 4–7), which may also translate to a field scenario. Results from the current

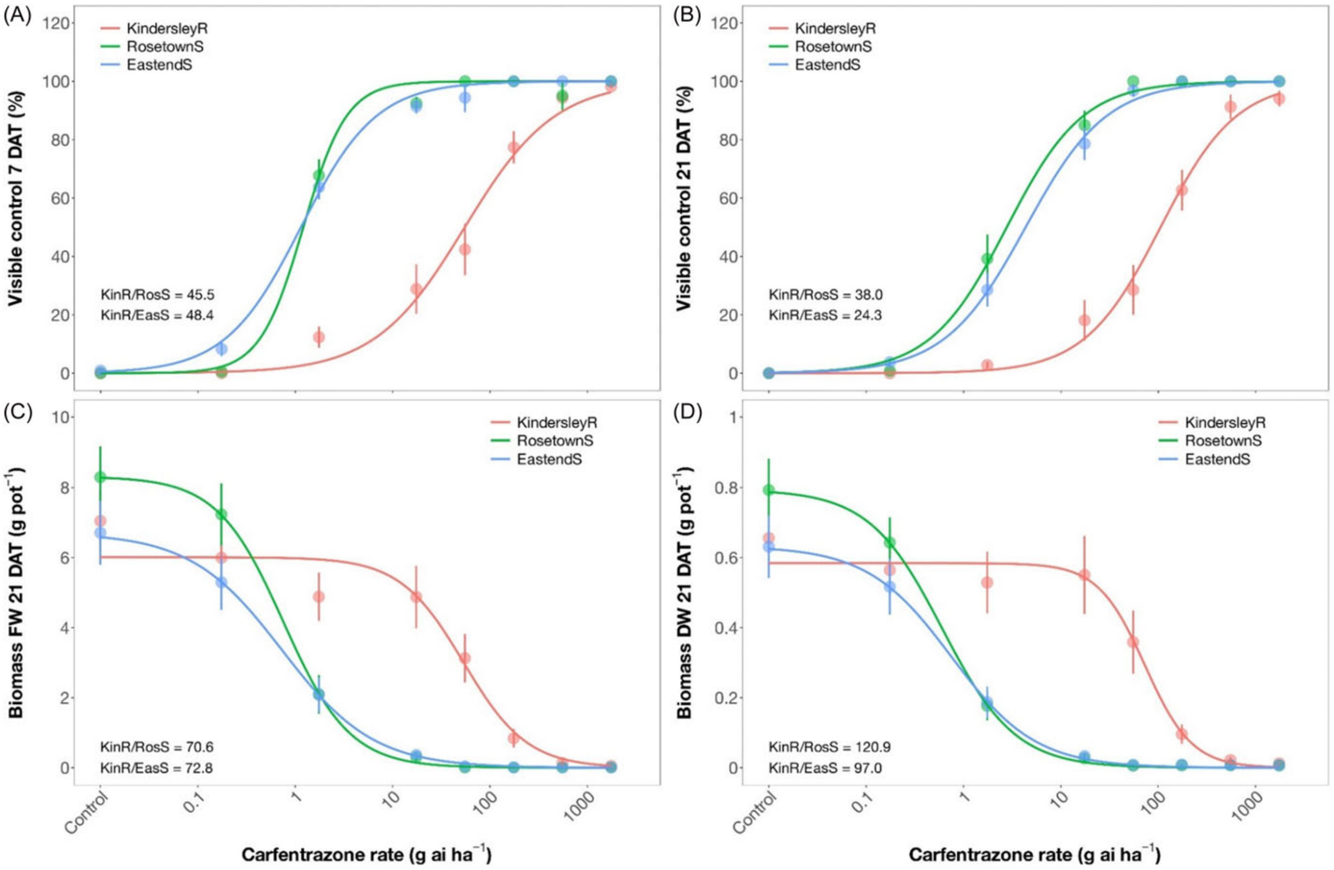

Figure 6. The response of one putative-resistant (KindersleyR) and two susceptible (RosetownS and EastendS) *Bassia scoparia* accessions from Saskatchewan, Canada, to a range of foliar-applied carfentrazone rates based on visible control at (A) 7 and (B) 21 d after treatment (DAT) and shoot biomass (C) fresh weight (FW) and (D) dry weight (DW) at 21 DAT. Dots indicate treatment means; bars represent standard errors. Embedded text indicates the resistance index (R/S ratio) for each putative-resistant accession relative to each susceptible accession.

study suggest that field-scouting efforts to identify PPO inhibitor–resistant *B. scoparia* should be effective when conducted between 1 and 3 wk after foliar treatment, but may be more obvious later given the initial necrosis injury observed after treatment of resistant plants.

The current study showed that the PPO inhibitor–resistant *B. scoparia* accessions exhibited cross-resistance to two chemical families of PPO-inhibiting herbicides (Table 3; Figures 2–7); saflufenacil belonging to the *N*-phenylimides and carfentrazone belonging to the N-phenyltriazolinones (HRAC 2024b). Cross-resistance in *B. scoparia* among the other families of PPO inhibitors warrants further research. Indeed, variable cross-resistance to PPO-inhibiting chemical families has been noted in other weed species and depends on herbicide application method and timing, the weed species, and the resistance mechanism (Barker et al. 2023). Further research aimed at understanding the mechanism conferring PPO-inhibitor resistance in *B. scoparia* may help further elucidate the associated pattern of cross-resistance.

Practical Implications

Spread of PPO inhibitor–resistant *B. scoparia* could limit options for herbicidal control, especially given the likely stacking of multiple resistance traits in this species resulting in resistance across a wide range of herbicide sites of action. Herbicide resistance traits can evolve and spread efficiently in *B. scoparia*, as demonstrated by the rapid increase in frequency of populations resistant to glyphosate and ALS-inhibiting herbicides in recent decades (Geddes et al. 2022c, 2023; Kumar et al. 2019; Sharpe et al. 2023; Westra et al. 2019). In *B. scoparia*, rapid evolution and spread of these traits is due to a combination of ample selection pressure due to heavy use of herbicides across a large area, high genetic diversity (Martin et al. 2020), and efficient seed- and pollen-mediated gene flow (Beckie et al. 2016). PPO-inhibitor resistance in *B. scoparia* will create a gap, particularly during the preplant/preemergence weed control window in several field crops grown in the Northern Great Plains (Tables 6 and 7). As multiple herbicide resistance traits continue to stack in this species, the available options for herbicidal control become limited, causing reliance on contact-type herbicides like glufosinate (HRAC Group 10) postemergence in crops engineered to resist this glutamine synthetase inhibitor or the PSII inhibitor bromoxynil (HRAC Group 6) alone or mixed with an inhibitor of 4-hydroxyphenyl-pyruvate dioxygenase (HRAC Group 27). One key difference between Canada and the United States, among others, is the commercial availability of the photosystem I–inhibiting herbicide paraquat (HRAC Group 22) in the United States (Ikley et al. 2024) but not Canada (Anonymous 2024a, 2024b), which further limits herbicidal control options north of the Canada/U.S. border.

Careful stewardship of herbicides that remain effective on multiple herbicide–resistant *B. scoparia* is warranted through further and more targeted implementation of integrated weed management programs. Past research has shown that *B. scoparia* responds to competitive crop scenarios by substantially reducing

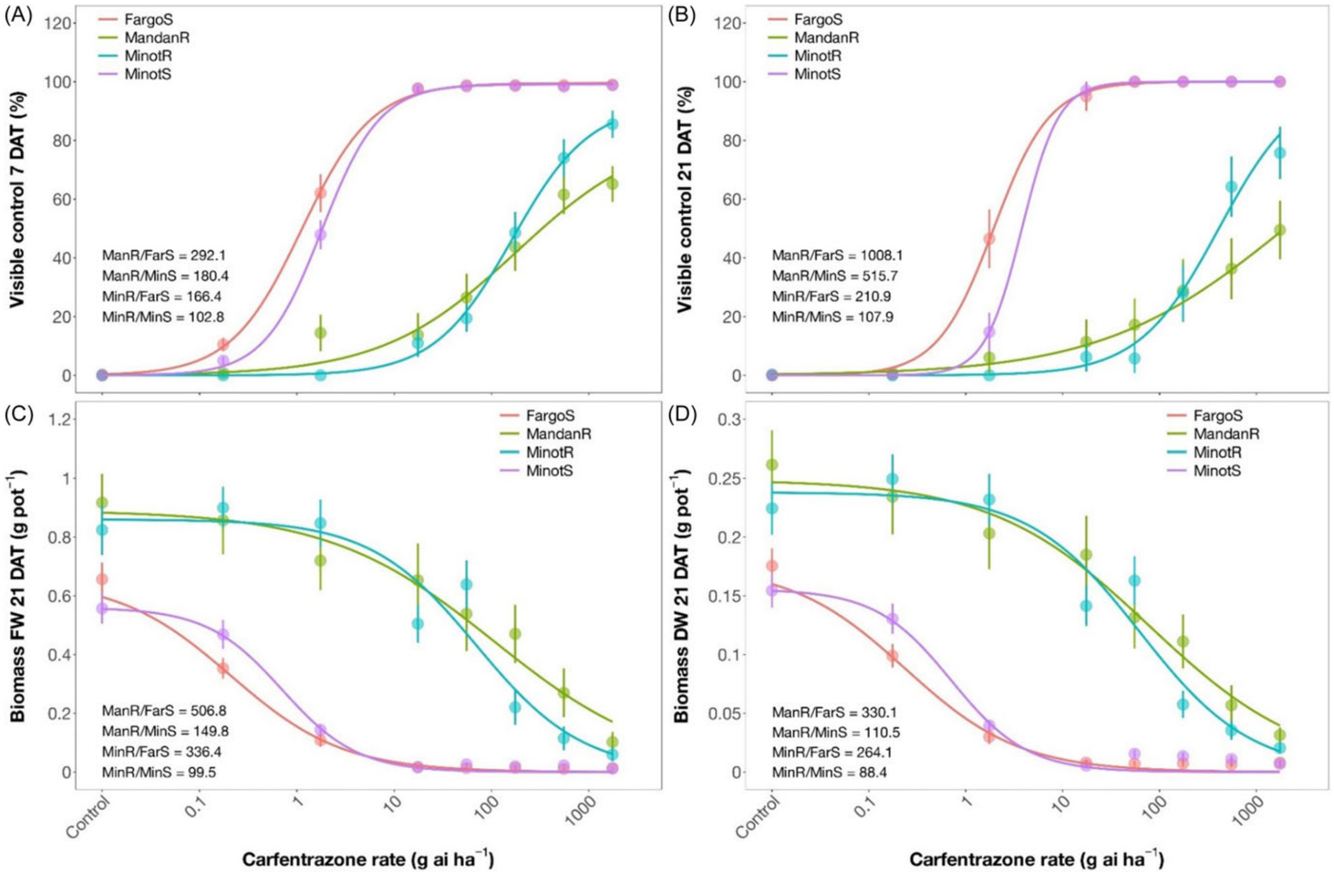

Figure 7. The response of two putative-resistant (MandanR and MinotR) and two susceptible (FargoS and MinotS) *Bassia scoparia* accessions from North Dakota, USA, to a range of foliar-applied carfentrazone rates based on visible control at (A) 7 and (B) 21 d after treatment (DAT) and shoot biomass (C) fresh weight (FW) and (D) dry weight (DW) at 21 DAT. Dots indicate treatment means; bars represent standard errors. Embedded text indicates the resistance index (R/S ratio) for the putative-resistant accession relative to each susceptible accession.

Table 6. Herbicide options registered for *Bassia scoparia* control or suppression in western Canada assuming blanket resistance to all active ingredients within Herbicide Resistance Action Committee (HRAC) Groups 2, 4, 9, and 14[a,b].

Use window and herbicide	HRAC Group	Wheat	Barley	Oat	Corn	Canola	Mustard	Flax	Soybean	Field pea	Lentil
Soil-applied preemergence											
Ethalfluralin[c]	3					C	C[d]		C	C	C
Triallate/trifluralin[c]	15/3	S	S			S	S	S			
Trifluralin + metribuzin[c]	3 + 5									C	
Pyroxasulfone	15	S			S				S	S	S
Foliar-applied preplant											
Bromoxynil[e]	6	C	C	C	C	C					
Bromoxynil + topramezone	6 + 27					C					
Bromoxynil + pyrasulfotole[e]	6 + 27	C	C								
Pyridate	6				C	C				C	
Foliar-applied postemergence											
Bromoxynil	6	C	C	C	C			C			
Bromoxynil + tolpyralate	6 + 27	C	C								
Bromoxynil + pyrasulfotole	6 + 27	C	C								
Glufosinate[f]	10					C					
Pyridate	6				C						
Pyroxasulfone	15				S				S		
Topramezone	27				C[g]						
Tembotrione	27				C						

[a] Adapted from Anonymous (2024a, 2024b); C indicates control (≥80% control), S indicates suppression (60–79% control).

[b] Wheat, *Triticum aestivum* L.; barley, *Hordeum vulgare* L.; oat, *Avena sativa* L.; corn, *Zea mays* L.; canola, *Brassica napus* L.; mustard, *Brassica juncea* (L.) Czern. or *Sinapis alba* L.; flax, *Linum usitatissimum* L.; soybean, *Glycine max* (L.) Merr.; field pea, *Pisum sativum* L.; lentil, *Lens culinaris* Medik.

[c] Preplant incorporated or late fall applied.

[d] Yellow mustard only.

[e] Mixed with glyphosate.

[f] Glufosinate-resistant varieties.

[g] Must be applied with tank-mix partner.

Table 7. Herbicide options registered for *Bassia scoparia* control or suppression in the United States assuming blanket resistance to all active ingredients within Herbicide Resistance Action Committee (HRAC) Groups 2, 4, 9, and 14[a,b].

Use window and herbicide	HRAC Group	Wheat	Barley	Oat	Corn	Canola	Mustard	Flax	Soybean	Field pea	Lentil
Soil-applied preemergence											
Atrazine	5				S/C						
Ethalfluralin[c]	3					S/C	S/C	S/C	S/C	S/C	S/C
Isoxaflutole	27				C						
Metribuzin	5								C	C	C
Pendimethalin	3				S				S	S	S
Pyroxasulfone	15	S			S				S	S	S
Trifluralin[c]	3	S	S		S	S	S	S	S	S	S
Foliar-applied preplant											
Glufosinate	10				C	C			C		
Paraquat	22	C	C	C	C	C	C	C	C	C	C
Pyridate	6				C						S
Foliar-applied postemergence											
Bentazon	6				S				S	S	
Bromoxynil	6	C	C	C	C			C			
Bromoxynil + bicyclopyrone	6 + 27	C	C								
Bromoxynil + pyrasulfotole	6 + 27	C	C								
Bromoxynil + tolpyralate	6 + 27	C	C								
Glufosinate[d]	10				C	C			C		
Mesotrione + atrazine	27 + 5				C						
Pyridate	6				C						S
Tembotrione + atrazine	27 + 5				C						
Tolpyralate + atrazine	27 + 5				C						
Topramezone + atrazine	27 + 5				C						

[a] Adapted from Ikley et al. (2024); C indicates control (≥80% control), S indicates suppression (60–79% control).
[b] Wheat, *Triticum aestivum* L.; barley, *Hordeum vulgare* L.; oat, *Avena sativa* L.; corn, *Zea mays* L.; canola, *Brassica napus* L.; mustard, *Brassica juncea* (L.) Czern. or *Sinapis alba* L.; flax, *Linum usitatissimum* L.; soybean, *Glycine max* (L) Merr.; dry pea, *Pisum sativum* L.; lentil, *Lens culinaris* Medik.
[c] Preplant incorporated or late fall applied.
[d] Glufosinate-resistant varieties.

seed production (Mosqueda et al. 2020). Management practices targeting *B. scoparia* seed production and return to the soil seedbank represent a key choking point in the life cycle of this weed (Geddes and Davis 2021) due to short seed longevity once it enters the soil seedbank (Beckie et al. 2018; Dille et al. 2017; Geddes 2021; Schwinghamer and Van Acker 2008). Integrating nonchemical practices, such as competitive crops (Mosqueda et al. 2020), alternative crop life cycles (Geddes and Davis 2021), higher crop seeding rates (Geddes and Kimmins 2021), strategic yet judicious tillage (Obour et al. 2021), or cutting *B. scoparia* for animal feed (Nair et al. 2021), may serve to reduce *B. scoparia* seedbank replenishment. In addition, cutting or mowing *B. scoparia* plants could help mitigate the globe-shaped growth structure characteristic of tumbleweeds by physically disrupting unfettered growth and development, thereby preventing *B. scoparia* movement beyond its source location. Physical barriers like fence lines or shelterbelts may also serve to mitigate seed-mediated gene flow by catching *B. scoparia* plants that move beyond field boundaries (Beckie et al. 2016; Geddes and Sharpe 2022). Mitigation efforts should employ the core foundational principles of integrated weed management wherein multiple cultural, physical, and biological tactics are implemented along with strategic herbicide use to limit *B. scoparia* proliferation. Of utmost importance will be continued investment in the design and understanding of sustainable integrated weed management strategies that target the unique biology of this troublesome and highly elastic species.

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