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Herbicidal control of deathcamas (*Zigadenus paniculatus*)

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Research Article

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Abstract

Foothill deathcamas is a bulbous, perennial, native forb found throughout the western United States. Deathcamas begins growth early in the spring. The lack of alternative forages at this time can result in livestock becoming poisoned from the consumption of deathcamas. Research on herbicides for deathcamas control is limited to work from the 1950s and 1960s that identified 2,4-D as a control agent. The objective of this study was to evaluate alternative herbicide options for deathcamas control that include 2,4-D, 2,4-D + triclopyr, quinclorac, aminopyralid, imazapic, and chlorsulfuron. We also investigated the impact of plant growth stage on deathcamas control by making herbicide applications at two growth stages. One set of plots was treated with herbicides when deathcamas was in the early vegetative stage and the second set was treated at flowering. There is some evidence that stress might affect alkaloid content; therefore, we monitored alkaloid content of treated and nontreated deathcamas. Plots were established at Mt. Sterling, UT, and Mt. Pleasant, UT. Deathcamas density was reduced in 2,4-D, 2,4-D + triclopyr, and imazapic treatments 1 and 2 yr after herbicide application ($P < 0.0001$). Compared with the pretreatment densities, deathcamas densities(± standard error of the mean) 2 yr after herbicide application were reduced 96% ± 1.4%, 100% ± 0%, and 98% ± 0.9% for 2,4-D, 2,4-D + triclopyr, and imazapic, respectively, at the Mt. Sterling site. At the Mt. Pleasant site, deathcamas density was reduced by 84% ± 2.8% with 2,4-D alone, whereas 2,4-D + triclopyr and imazapic provided similar density reductions as observed at the Mt. Sterling site. Steroidal alkaloid concentrations did not change in herbicide-treated deathcamas at either stage of plant growth. These data indicate that 2,4-D, 2,4-D + triclopyr, and imazapic can effectively control deathcamas in the vegetative and flowering growth stages.

Introduction

Deathcamas is indigenous to North and Central America, where at least 15 species are recognized (Burrows and Tyrl 2013). Taxa occur in a wide variety of habitats throughout North America, with species of toxic concern occurring primarily in grasslands, alpine meadows, pine woods, pinyon-juniper (*Pinus* spp.-*Juniperus* spp.) woodland, and shrublands (Burrows and Tyrl 2013). Deathcamas emerges and begins growth early in the spring at the start of the growing season.

The lack of alternative forages at this time can result in livestock consuming deathcamas. Similarly, placing hungry animals in deathcamas–infested areas later in the season can result in livestock poisonings at any stage of the growing season (Panter et al. 1987). Deathcamas is poisonous to cattle (*Bos taurus* L.) and sheep (*Ovis aries* L.) with most losses occurring in sheep (Kingsbury 1964). In the early 1900s, there were numerous reports of sheep poisoned by deathcamas (Marsh et al. 1915). In two separate poisoning cases, a total of 4,730 sheep were affected, resulting in 1,136 deaths. Poisonings continue to occur even with improved management. Several hundred sheep were lost in a poisoning in 1986 (Panter et al. 1987), and losses due to deathcamas poisonings continue today. In 2016, a producer in northern Utah lost approximately 300 sheep to deathcamas poisoning (Stonecipher, personal observation).

Research on herbicidal control of deathcamas is limited; most control studies were conducted decades ago (Bohmont 1952; Hyder and Sneva 1962). This research identified 2,4-D as an effective herbicide when applied early in the season during vegetative development and decreased rapidly after flower buds appeared (Hyder and Sneva 1962). Picloram, clopyralid, picloram + clopyralid, and metsulfuron methyl did not provide acceptable control of meadow deathcamas (*Zigadenus venenosus* S. Watson) (Carpenter 1986).

Herbicides can raise, lower, or have no effect on the concentration of toxic compounds in plants (Williams and James 1983). Ralphs et al. (1998) measured an increase in toxic alkaloid

Table 1. List of herbicides and application rates used to treat deathcamas.

Treatment	Trade name	Active ingredient	Application rate g ai ae ha^{-1}	Manufacturer
2,4-D amine	2,4-D amine	Dimethylamine salt of 2,4-dichloro-phenoxyacetic acid	2,130	Agristar, Allbaugh LLC, Ankeny, IA
2,4-D + triclopyr	Crossbow®	2,4-dichlorophenoxyacetic acid, butoxyethyl ester + 3,5, 6-trichloro-2-pyridinyloxyacetic acid, butoxyethyl ester	1,120 + 560	Dow AgroSciences LLC, Indianapolis, IN
Quinclorac	Facet® L	Dimethylamine salt of quinclorac: 3,7-dichloro-8-quinolinecarboxylic acid	420	BASF Corporation, Research Triangle Park, NC
Aminopyralid	Milestone®	Triisopropanolammonium salt of 2-pyridinecarboxylic acid, 4-amino-3,6-dichloropyridine	123	Dow AgroSciences LLC, Indianapolis, IN
Imazapic	Plateau®	Ammonium salt of imazapic (±)-2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1*H*-imidazol-2-yl]-5-methyl-3-pyridinecarboxylic acid	175	BASF Corporation, Research Triangle Park, NC
Chlorsulfuron	Telar®	2-chloro-*N*-[(4-methoxy-6-methyl-1,3,5-triazin-2-yl) aminocarbonyl]benzenesulfonamide	52.5	E. I. duPont de Nemours and Company, Wilmington, DE

concentrations in larkspur [*Delphinium barbeyi* (Huth) Huth] plants treated with metsulfuron, whereas glyphosate and picloram did not affect alkaloid concentrations.

Current herbicide options available for deathcamas control are limited. However, recently introduced herbicides have not been evaluated for their efficacy on this species and may provide an alternative to 2,4-D. Thus, the objectives of this study were to (1) evaluate 2,4-D, 2,4-D + triclopyr, quinclorac, aminopyralid, imazapic, and chlorsulfuron for their efficacy in controlling foothill deathcamas; (2) determine if plant growth stage at the time of application influences herbicide effectiveness; and (3) determine if herbicide treatment alters alkaloid content of deathcamas.

Materials and Methods

Study Site

Plots were established at two sites in northern (Mt. Sterling) and central (Mt. Pleasant) Utah. The Mt. Sterling site was located 8 km southeast of Wellsville, UT (41.56°N, 111.90°W), on a west-facing slope with an elevation of 1,662 m. The soil is a fine, montmorillonitic, frigid, Pachic Palexerolls (Mountain stony loam). The ecological site is classified as mountain big sagebrush [*Artemisia tridentata* Nutt. ssp. *vaseyana* (Rydb.) Beetle] (USDA NRCS 2018).

The Mt. Pleasant site was located 3.3 km northeast of Mt. Pleasant, UT (39.57°N, 111.42°W), on an east-facing slope with an elevation of 1,873 m. The soil is loamy-skeletal, carbonatix, frigid, Typic Calcixerolls (Upland stony loam). The ecological site is classified as Pinyon (*Pinus monophylla* Torr. & Frém.)-Utah juniper [*Juniperus osteosperma* (Torr.) Little].

Experimental Design

The study was a randomized complete block design in a factorial arrangement consisting of four blocks $site^{-1}$. Each block consisted of 13 plots (3 × 9 m) with six herbicide treatments applied at two timings, early (vegetative) and late (flowering) growth stages, and one nontreated control plot. Herbicides and application rates are listed in Table 1. Herbicide rates were chosen on the basis of manufacturer recommendations for closely related plant species. Treatment means are reported as original, nontransformed data ± standard error of the mean.

Early herbicide application occurred on April 12, 2017, at the Mt. Pleasant site. Deathcamas plants were in the vegetative growth stage; plant height was 20 ± 8 cm. Early application at the Mt. Sterling site occurred on April 13, 2017. Deathcamas plants were in the vegetative growth stage; plant height was 16.5 ± 6 cm. Late herbicide application occurred on May 3, 2017, at the Mt. Pleasant site. Deathcamas plants were in the flowering stage and were 28 ± 8 cm tall. Late herbicide application occurred on May 11, 2017, at the Mt. Sterling site. Deathcamas plants were in the flowering stage and were 30 ± 8 cm tall. A nonionic surfactant (0.25% vol/vol) was included with each herbicide treatment. Herbicides were applied using a CO_2-pressurized backpack sprayer at a rate of 168 L ha^{-1} at 207 kPa at 4.0 km h^{-1}. The spray boom consisted of six 8002 flat-fan nozzles (Spraying Systems Co., Wheaton, IL) spaced 51 cm apart.

Measurements

The total number of deathcamas plants in each treatment plot was counted at the Mt. Pleasant site before herbicide application, then again 1 yr after treatment (YAT) and 2 YAT. Because of the density of deathcamas plants at the Mt. Sterling site, whole-plot densities were estimated by counting the total number of plants within a 1-m belt transect down the center of each plot, following the same timeline as at the Mt. Pleasant site. Visual estimates of control were recorded for the early herbicide application at 21 d after treatment (DAT) and 28 DAT at the Mt. Pleasant and Mt. Sterling sites, respectively. Visual control estimates ranged from 0% to 100%, with 0% representing no injury and 100% representing apparent death (Nelson et al. 2014). Visual estimates were not made for deathcamas plants treated at the late herbicide application timing, because plants were naturally senescing before herbicide effects could be evaluated. The height of deathcamas plants was measured just before the application of herbicide treatments.

Plant Material

Six individual deathcamas plants were collected $plot^{-1}$ 7 or 8 DAT and stored at −18 C. Individual plants were dried in a FreeZone® 18-L console freeze-dry system (Labconco, Kansas City, MO) and then ground in a Wiley mill to pass through a 1-mm screen. Two of the six plants collected were randomly selected for alkaloid analysis.

Plant Sample Analytical Extraction

Aliquots (50 mg) of dry, ground plant material were placed into 1.5-mL, plastic, conical, snap-cap tubes (Eppendorf safe-lock tubes), and 1.0 mL of methanol (containing 100 ppm reserpine as an internal standard) was added and the samples extracted for 24 h by mechanical rotation on a tube rotator (Rugged Rotator; Glas Col, LLC, Terre Haute, IN). Samples were centrifuged in a microcentrifuge at 13,200 rpm for 10 min. An aliquot (50 µL) of the methanol extract

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Table 2. Deathcamas density before herbicide treatment and 1 and 2 y after treatment at the Mt. Sterling, UT, and Mt. Pleasant, UT, study sites.

Treatment	Plant density[a]					
	Pretreatment		1 yr posttreatment		2 yr posttreatment	
	no. m^{-2}					
Mt. Sterling[b]						
Nontreated	6.0 ± 1.40	a	3.3 ± 0.81	cde	4.0 ± 1.19	abcde
2,4-D	5.7 ± 1.22	ab	0.3 ± 0.07	f	0.2 ± 0.08	f
2,4-D + triclopyr	4.7 ± 0.44	abcde	0.1 ± 0	f	0.1 ± 0	f
Quinclorac	5.6 ± 0.74	a	3.1 ± 0.53	bcde	2.9 ± 0.68	e
Aminopyralid	4.7 ± 0.89	abcde	3.0 ± 0.58	cde	3.0 ± 0.69	cde
Imazapic	5.0 ± 0.95	abcd	0.0 ± 0.82	f	0.0 ± 0.07	f
Chlorsulfuron	5.6 ± 1.20	abc	3.3 ± 1.10	de	4.0 ± 0.87	abcde
Mt. Pleasant[c]						
Nontreated	0.9 ± 0.19	tuv	0.6 ± 0.19	uvw	0.7 ± 0.21	uvw
2,4-D	1.7 ± 0.35	st	0.4 ± 0.18	vwx	0.3 ± 0.09	wxy
2,4-D + triclopyr	2.0 ± 1.17	stu	0.0 ± 0.02	z	0.0 ± 0.03	yz
Quinclorac	1.5 ± 0.43	stu	1.6 ± 0.36	stu	1.4 ± 0.30	stu
Aminopyralid	2.4 ± 0.74	s	2.1 ± 0.47	st	2.2 ± 0.65	st
Imazapic	2.1 ± 0.41	st	0.5 ± 0.02	xyz	0.3 ± 0.02	xyz
Chlorsulfuron	2.2 ± 1.12	st	1.4 ± 0.75	tu	1.5 ± 0.72	stu

[a]There was no difference in the early or late herbicide application timing ($P \geq 0.13$), so data were combined over the two application timings.
[b]Means followed by the same letter are not significantly different between treatments at the Mt. Sterling site ($P < 0.05$).
[c]Means followed by the same letter are not significantly different between treatments at the Mt. Pleasant site ($P < 0.05$).

was added to 950 µL of a methanol and water solution (50:50) contained in a 1.5-mL glass autosample vial. Standards were prepared and analyzed along with plant samples, following procedures previously described (Stonecipher et al. 2020) using high-performance liquid chromatography-mass spectrometry.

Data Analyses

Deathcamas density, percent change in deathcamas density, and alkaloid concentration were assessed as a randomized block design in a factorial arrangement using a generalized linear mixed model (PROC GLIMMIX) method in a mixed-model ANOVA with repeated measures in SAS, version 9.4 (SAS Institute, Cary, NC). Visual estimates of deathcamas control were assessed using the same model without repeated measures.

Plant density and alkaloid concentration values were averaged over blocks and the means used for analysis. Plots were the experimental units and the four blocks were replicates. Herbicide treatment, application timing, and year were the fixed effects factors and block and repeated measures were incorporated as random effects factors. Alkaloid concentrations were log transformed and deathcamas density was square-root transformed to meet assumptions of normality and homogeneity of variance. As noted previously, treatment means are reported as original, nontransformed data ± standard error of the means. Treatment means were separated using the LSMEANS method, and main effects were adjusted for Type I error inflation using the Tukey method.

Deathcamas control was evaluated at two different growth stages: vegetative and flowering. The two sites were analyzed separately. Deathcamas plant counts were converted to the number of plants m^{-2}. Percent change in plant densities was calculated by the difference in deathcamas plants before herbicide treatment and at 1 and 2 YAT.

Results and Discussion

Deathcamas density before herbicide application was 5.3 ± 0.61 and 1.8 ± 0.32 plants m^{-2} at the Mt. Sterling and Mt. Pleasant sites, respectively. Deathcamas density was similar among treatment plots before herbicide application at both sites (Table 2); however, there was a natural decrease in deathcamas density at the Mt. Sterling site in the first YAT, which was evident in the decrease in deathcamas density in the nontreated control plots. There were no differences in control of deathcamas when comparing herbicide application at the different phenologic stages ($P > 0.13$); thus, data were combined for the two stages of phenology.

Deathcamas control was 96% ± 1.3% and 93% ± 1.4% for 2,4-D + triclopyr at the Mt. Sterling and Mt. Pleasant sites, respectively ($P < 0.0001$; Figure 1) compared with 88% ± 1.4% and 84% ± 4.7% for 2,4-D alone at these sites ($P < 0.0001$; Figure 1). Deathcamas control with aminopyralid, chlorsulfuron, quinclorac, and imazapic was low, ranging from 6% to 34% at both sites (Figure 1).

Deathcamas density was significantly reduced by 2,4-D + triclopyr and by imazapic applications 1 and 2 YAT at the Mt. Sterling and Mt. Pleasant sites, respectively ($P < 0.0001$; Table 2). Deathcamas density was reduced by 2,4-D at 1 and 2 YAT at the Mt. Sterling site; however, at the Mt. Pleasant site, the 2,4-D treatment reduced deathcamas density, but the reduction was not significantly different from the nontreated plants. The 2,4-D + triclopyr reduced deathcamas densities by 100% ± 0%, and 97% ± 1.1% at the Mt. Sterling and Mt. Pleasant sites, respectively, whereas imazapic reduced deathcamas densities by 98% ± 0.9% and 99% ± 0.4% at the same sites ($P < 0.0001$; Figure 2). The 2,4-D alone reduced deathcamas densities by 96% ± 1.4% and 84% ± 2.8% at the Mt. Sterling and Mt. Pleasant sites, respectively (Figure 2).

Deathcamas density at the Mt. Sterling site decreased in quinclorac-treated plots in the first YAT and remained at this level at the second YAT, which is similar to what was observed in the nontreated plots (Table 2). It is likely that the decrease in deathcamas density was due to natural fluctuations, because deathcamas density did not decrease after quinclorac treatment at the Mt. Pleasant site. The 2,4-D-, 2,4-D + triclopyr-, and imazapic-treated plots had the greatest reduction in deathcamas density both years after herbicide treatment at both sites (Table 2).

The herbicide recommended to control deathcamas has been 2,4-D and is reported to be effective when plants are in the

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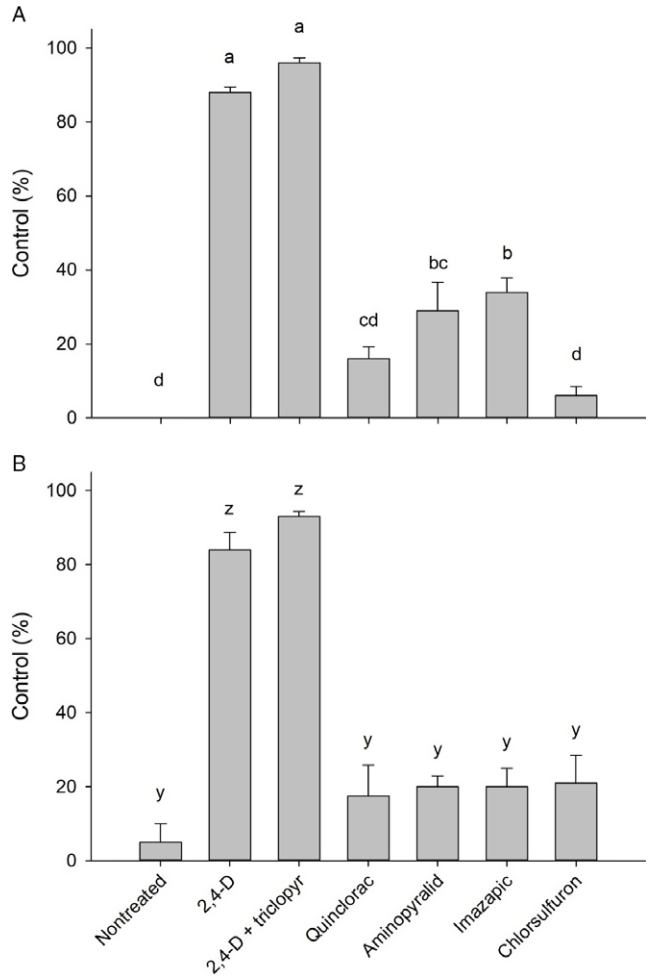

Figure 1. Visual control estimates of deathcamas plants treated with herbicides during the vegetative growth stage. Visual control was assessed (A) 28 d after treatment (DAT) at the Mt. Sterling, UT, site; and (B) 21 DAT at the Mt. Pleasant, UT, site. Bars with the same letter are not significantly different at $P < 0.05$.

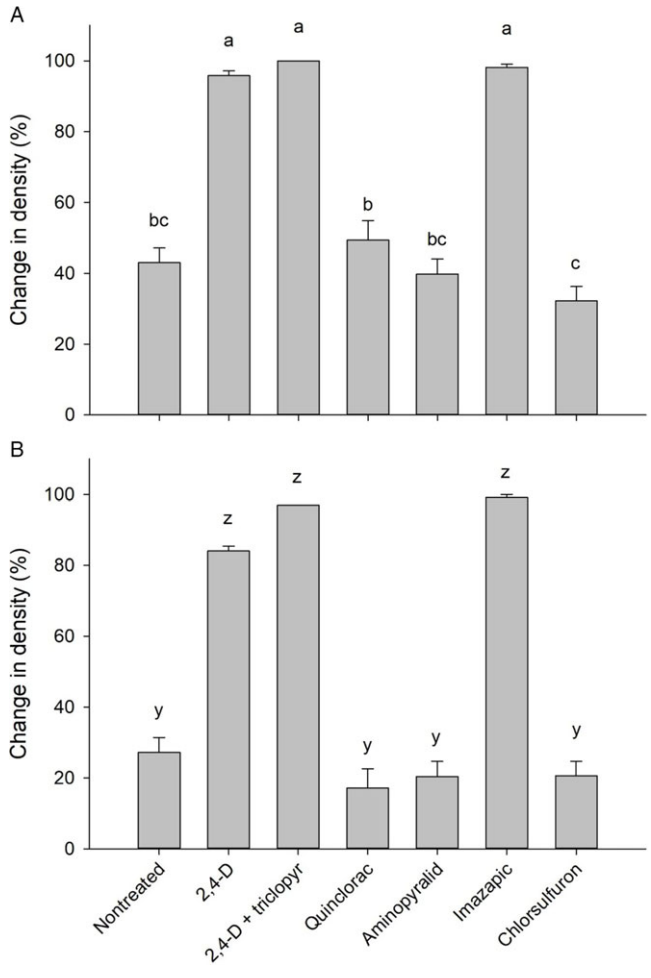

Figure 2. Percent decrease in deathcamas density the 2 yr after herbicide treatment combined at the (A) Mt. Sterling, UT, and (B) Mt. Pleasant, UT, sites. There was no difference in the early or late herbicide application timing ($P \geq 0.35$), so data were combined over the two application timings. Bars with the same letter are not significantly different at $P < 0.05$.

vegetative growth stage; however, efficacy decreases after flower buds appear (Hyder and Sneva 1962). These researchers used an ester formulation of 2,4-D; in our studies, the amine formulation of 2,4-D was used. The amine formulation of 2,4-D was effective in controlling deathcamas at both the early vegetative and flowering growth stages. Bohmont (1952) reported that the 2,4-D ester formulation was superior to the amine type in controlling deathcamas. The amine formulation of 2,4-D was effective at controlling deathcamas in this study; this result could be due to the addition of a nonionic surfactant, which may have aided in controlling deathcamas during the later growth stage.

Deathcamas contains steroidal alkaloids that are similar to those found in hellebore (*Veratrum* spp.) (Burrows and Tyrl 2013). Zygacine and zygadenine have both been found in deathcamas (Majak et al. 1992; Stonecipher et al. 2020), with zygacine reported as one of the primary toxic components (Welch et al. 2011); it was the major alkaloid detected in deathcamas in this study. Zygacine is reported to represent greater than 50% of the total steroidal alkaloids of deathcamas at the different phenological growth stages (Stonecipher et al. 2020). Zygacine concentrations were higher in plants collected at the early vegetative growth stage compared with plants collected at the flowering stage ($P \leq 0.016$) at 19.3 ± 1.84 mg g^{-1} and 11.8 ± 1.84 mg g^{-1} at the Mt. Sterling site and 11.7 ± 0.36 mg g^{-1} and 6.5 ± 0.36 mg g^{-1} at the Mt. Pleasant site, respectively. Majak et al. (1992) also found that zygacine concentrations were higher at the early vegetative growth stage compared with the flower growth stage in meadow deathcamas. Likewise, Stonecipher et al. (2020) reported higher concentrations of zygacine in the early vegetative growth stage compared with the flower growth stage in foothill deathcamas; the concentrations ranged from 16.6 to 56.6 mg g^{-1} in early vegetative growth to 2.7 to 17.3 mg g^{-1} in full flower for aboveground plant parts.

Plant secondary metabolites can vary depending on external factors such as light, temperature, soil water, soil fertility, and salinity (Yang et al. 2018). Makeiff et al. (1997) reported a difference in zygacine concentrations at two different sites and attributed the difference to a site with southern exposure, compared with a site with northern exposure. Herbicide treatment in the present study did not alter zygacine or zygadenine concentrations of deathcamas ($P \geq 0.12$) at either site. Zygadenine was detected in deathcamas plants at levels 9 to 29 times lower than zygacine.

Toxic risk is associated with the concentrations of the bioactive principle and the relative palatability of the plant. Sheep have shown signs of intoxication when dosed with 0.17 kg of dried plant

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material, and death has occurred when they were dosed at 0.34 kg of dried plant material (Panter et al. 1987). Welch et al. (2011) reported an LD_{50} of 2.0 mg kg^{-1} of zygacine in mice.

One issue land managers should be aware of is that plants may not display herbicide damage during the year of application. Herbicides can potentially increase palatability of treated plants and thus result in increased consumption of treated plants by livestock (Gatford et al. 1999; Leys et al. 1991). Deathcamas plants treated with imazapic did not display any signs of herbicide injury in the year after plants were treated. However, imazapic treatments were highly effective and deathcamas plant densities were reduced at 1 and 2 YAT, similar to 2,4-D and 2,4-D + triclopyr treatments. Imazapic is typically applied PRE or POST and works with a combination of foliar and soil activity. Deathcamas treated with imazapic did not show any signs of herbicide injury after treatment that would indicate foliar activity. Therefore, control of deathcamas with imazapic probably occurred due to soil activity.

Livestock producers may inadvertently turn livestock into an area with deathcamas that has been treated with imazapic and not know the plants were treated, because there may be no sign of herbicide injury. Even though imazapic treatment did not increase the alkaloid concentrations of deathcamas, imazapic treatment could potentially increase palatability of the plant, resulting in an increased poisoning risk. Deathcamas poisoning typically occurs early in the spring, because it is one of the first plants to begin growth and other forages are dormant or slow to emerge at this time. The higher alkaloid concentrations detected in deathcamas early in the spring can exacerbate poisonings, thus caution should be taken when grazing livestock on rangelands early in the spring that contain deathcamas.

The herbicides 2,4-D, 2,4-D + triclopyr, and imazapic were all effective in controlling deathcamas at the two locations in this study. Results from this study indicated 2,4-D, 2,4-D + triclopyr, and imazapic treatments were similarly effective at controlling deathcamas regardless of application timing. These treatments provided the same level of deathcamas control after application at the flowering and early vegetative growth stages. The option of using imazapic for deathcamas control also offers the advantage of providing invasive annual grass control and allows the use of a different mode of action in deathcamas management efforts.

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