Alternative herbicides for flupropanate resistant serrated tussock

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Summary Serrated tussock populations have become resistant to the group J herbicide flupropanate. Dose response trials show that the herbicides imazapyr, clethodim, haloxyfop-P as methyl and hexazinone were all effective in controlling serrated tussock resistant to flupropanate. Pinoxaden+cloquintocet-mexyl was not effective. The group J herbicide 2,2-DPA was only partially effective suggesting that flupropanate - resistant serrated tussock may also reduce effectiveness of other group J herbicides.

The findings presented in this paper provide an outline of the use of chemicals in a manner that is not specified on the product label. Further work needs to be undertaken in conjunction with the Australian Pesticide and Veterinary Medicines Association and product registrants.

Keywords Serrated tussock, flupropanate, herbicide resistance.

INTRODUCTION

Serrated tussock (ST) (*Nassella trichotoma* Trin. & Rupr Barkworth) is a Weed of National Significance in Australia (Thorp and Lynch 2000). ST grows in both arable and non-arable areas. In areas that are not arable or able to be cultivated, management options are often limited to manually chipping or spot/boom spraying with a selective chemical (Osmond *et al*. 2008). Often these non arable areas need to be aerially sprayed as vehicle access can be limited, or slopes are too steep for human access.

The main chemical used to selectively control ST in introduced pastures is flupropanate (Campbell and Vere 1995). Flupropanate is a soil residual chemical that is slowly taken up by the plant roots and foliage. The chemical can persist in the soil for up to three years, inhibiting the growth of germinating ST plants, whereas the seedbank of ST persists for longer than this period (Campbell and Vere 1995).

The repeated re-application of flupropanate to control re-establishing ST has led to the development of herbicide resistance to flupropanate and plants that are able to tolerate high doses of flupropanate (McLaren *et al.* 2006). Resistance to flupropanate is now becoming wide-spread with resistant populations now located in Diggers Rest and the Rowsley Valley in Victoria and at Armidale and Goulburn in NSW (McLaren 2010). The management options for these paddocks, if non-arable, are now restricted to manual means (chipping or spot spraying) or holding techniques such as spray topping to inhibit seed production.

This paper assesses a variety of herbicides with different modes of action for control of resistant ST in a pot trial.

MATERIALS AND METHODS

Growing plants Seed for the plants used was sourced from a site of known flupropanate resistance (Missen 37°41' 144°21') as well as seed that has been tested to be susceptible (St Albans 37°45' 144°47'). Seedlings were grown in jiffy media and transferred into 6 inch pots filled with loamy potting mix to be grown in the glasshouse maintained at 20–25°C ± 5°C receiving automatic overhead watering of three minutes duration every second day.

Herbicide selection and spraying Herbicides that showed levels of selectivity, and significant activity towards ST were chosen for the experiment. This was determined by past research (Melland and McLaren 1998, Pritchard 1998) as well as recent producer workshops (Grech 2011) and landholder consultations. Three rates of each herbicide were used based on label recommendations for the control of similar listed grassy weed species.

Plants were sprayed in a laboratory track sprayer travelling at 6.79 km h^{-1} fitted with Teejet AI 110015 nozzles operating at 2.7 bar, or Teejet AI 11002 nozzles operating at 3.2 bar, delivering equivalent to 100 or 150 litres of water per hectare respectively.

Statistical analysis Plant damage caused by the herbicide application (day 0) was measured on a scale of 0 (not affected) to 9 (dead) on a weekly basis up to day 63 and then fortnightly to day 84 and monthly assessment to day 119.

Scores from the final assessment (119 days) were used to determine plant susceptibility to herbicides. Two outcome measures of susceptibility were used:

the number of dead plants (damage score = 9) and the number of severely affected plants (damage score ≥7). Both outcome measures were estimated for each category of herbicide [flupropanate (Kenock®), 2,2 – DPA, imazapyr, clethodim, haloxyfop-P as methyl, hexazinone, pinoxaden+cloquintocet-mexyl and Flupropanate (Frenock®)], application rate (none, low, medium and high) and plant resistance to flupropanate (yes/no).

Comparisons of deaths and severe adverse effects between experimental groups were made using Fisher exact test. Logistic regression analysis was used to test for trends. Stata SE 12.1 was used for all statistical analyses.

RESULTS

A breakdown of plant responses to different herbicides and the rates of herbicide application is given in Table 2. As expected, in the presence of flupropanate, flupropanate-resistant plants had significantly fewer deaths than flupropanate-susceptible plants (p-value ≤0.003).

There was no significant difference in the effects of other herbicides on flupropanate-resistant and flupropanate-susceptible plants. Both flupropanate-susceptible and -resistant plants appeared to tolerate treatment 7 (pinoxaden+cloquintocet-mexyl; rate of death=8.3%; p-value). Both were also susceptible to the harmful effects of Treatments 3–6 (100% death rate at medium and high doses).

Table 1. Treatments applied.

Treatment number	Group	Trade name	Active constituent	Rate 1 l/ha	Rate 2 l/ha	Rate 3 l/ha	Wetter	Water l/ha
1	J	Kenock	Flupropanate (745g/l)	1	2	3	no	150
2	J	Propon	2,2 - DPA (740g/kg)	2kg	4kg	8kg	bs1000	150
3	BM	Arsenal express	Glyphosate (present as isopropylamine salt) (150g/L) & imazapyr (present as isopropylamine salt) (150g/L)	0.5	1	2.5	no	100
4	A	Select	Clethodim (240g/l)	250ml	500ml	1l	hasten	150
5	A	Verdict	Haloxyfop-P as methyl(520g/L)	400ml	800ml	1.2l	bs1000	100
6	C	Velpar	Hexazinone (750g/kg)	3.5kg	5kg	8kg	bs1000	100
7	A	Axial	Pinoxaden (100g/L)+cloquintocet-mexyl (25g/L)	150ml	300ml	450ml	adigor	100
8	J	Frenock	Flupropanate (745g/L)	1	2	3	no	150

Table 2. Breakdown of plant death (damage scores of 9) by type of treatment (active compounds), the rate of application and plant susceptibility to Flupropanate.

	Number of dead plants [dead plants /total (%)]		Susceptible vs. resistant
Independent Variables	susceptible	resistant	[p-value]
Control	0 / 4 (0%)	0 / 4 (0%)	---
Active compounds			
1. Flupropanate (Kenock)	9 / 12 (75.0%)	0 / 12 (0%)	<0.001
2. 2,2 - DPA	8 / 12 (66.7%)	5 / 12 (41.7%)	0.414
3. imazapyr	12 / 12 (100%)	12 /12 (100%)	---
4. clethodim	11 / 12 (91.7%)	11 / 12 (91.7%)	1.000
5. haloxyfop-P as methyl	12 / 12 (100%)	12 / 12 (100%)	---
6. hexazinone	12 / 12 (100%)	12 / 12 (100%)	---
7. pinoxaden+cloquintocet-mexyl	1 / 12 (8.3%)	1 / 12 (8.3%)	1.000
8. Flupropanate (Frenock)	9 / 12 (75.0)	1 / 12 (8.3%)	0.003
Rates			
Low	19 / 32 (59.4%)	15 / 32 (46.9%)	0.453
Medium	27 / 32 (84.4%)	19 / 32 (59.4%)	0.050
High	28 / 32 (87.5%)	20 / 32 (62.5%)	0.041

Fisher exact test cannot estimate the probability of death when it is a certainty.
(12 dead/12 tested or 4 alive / 4 tested).

Flupropanate-susceptible and -resistant plants had different death rates at medium (84.4% *vs.* 59.4%) and high (87.5% *vs.* 62.5%) rates of application. These differences were of borderline significance, with respective p-values of 0.05 and 0.041. There was no detectable difference in the deaths of flupropanate-susceptible and -resistant plants at the low rate of herbicide application. Similar results were obtained for severe adverse effects: 90.6% *vs.* 59.4% at medium application rate; 87.5% *vs.* 62.5% at high application rate (p-values: 0.008 and 0.041 respectively).

The differences between flupropanate-susceptible and -resistant plants at medium and high rates of application in Table 2 appear to reflect different responses to two herbicides: flupropanate and possibly 2,2-DPA, (Table 3). For all other herbicides, increasing the rate of application had similar effects on both susceptible and resistant plants. Overall trends were similar for both outcomes (Tables 2 and 3).

DISCUSSION

The commonly used group J chemicals in the trial (Kenock® and Frenock®) were effective at killing susceptible ST plants (generally greater than 75% death at rates of 2 l ha^{-1} or more) but were not effective in killing flupropanate resistant plants at any rate tested. Although Propon® is also a group J chemical, it did provide some activity against flupropanate resistant plants although it was only 50% and 75% effective at the 4 kg ha^{-1} and 8 kg ha^{-1} rates respectively.

Chemicals that were not group J herbicides were able to kill both the flupropanate- susceptible and -resistant ST plants. Generally, the plants were readily killed with the low rate application, although Axial® was the exception. This herbicide had very low activity against flupropanate-susceptible (8.3%) or -resistant plants (8.3%) at all rates tested. The selectivity of the tested chemicals to non target species was not tested in this trial and needs to be considered when planning applications. Herbicides such as Arsenal Express® and Velpar® are known to be non selective, where as Select® and Verdict® are grass selective and suited to broadleaf crops. The rates used in this experiment may also influence the degree of selectivity in certain crops – see the chemical label.

CONCLUSION

This trial has found that flupropanate-resistant ST can be killed by using chemicals from other herbicide resistance groups, with moderate levels of control provided by other active constituents from group J. The findings of this trial will be used to consult with the APVMA and investigate the opportunity to obtain an off-label permit, and it is hopeful that product registration may also be arranged so as these chemicals can be used to control ST in the future.

Table 3. Comparisons of plant death between rate categories. Treatment groups were combined for comparison as shown. Separate comparisons were made for Flupropanate-susceptible and Flupropanate-resistant plants. The total numbers of dead/near dead plants and the percentage of dead/near dead plants are shown; in each instance, a total of four plants was tested.

	Death in Flupropanate-Susceptible plants				*Death* in Flupropanate-Resistant plants			
Active compounds	Low rate	Medium rate	High rate	P-value*	Low rate	Medium rate	High rate	P-value*
Control	0 (0%)	N/A	N/A	N/A	0/4 (0%)	N/A	N/A	N/A
1. Flupropanate (Kenock)	2 (50%)	3 (75%)	4 (100%)	0.042*	0 (0%)	0 (0%)	0 (0%)	1.000*
8. Flupropanate (Frenock)	2 (50%)	3 (100%)	4 (100%)	0.108**	0 (0%)	1 (25%)	0 (0%)	1.000**
2. 2,2 – DPA	0 (0%)	4 (100%)	4 (100%)	---				0.068*
				0.006**	0 (0%)	2 (50%)	3 (75%)	0.091**
3. imazapyr	4 (100%)	4 (100%)	4 (100%)	0.693*	4 (100%)	4 (100%)	4 (100%)	
4. clethodim	3 (75%)	4 (100%)	4 (100%)	0.919**	3 (75%)	4 (100%)	4 (100%)	
5. haloxyfop-P as methyl	4 (100%)	4 (100%)	4 (100%)		4 (100%)	4 (100%)	4 (100%)	0.432*
6. hexazinon	4 (100%)	4 (100%)	4 (100%)		4 (100%)	4 (100%)	4 (100%)	0.919**
7. pinoxaden + cloquintocet-mexyl	0 (0%)	1 (25%)	0 (0%)		0 (0%)	0 (0%)	1 (0%)	

* Test for trend using logistic regression. ** Fisher's exact test, --- Fisher exact test cannot estimate the probability of death when it is a certainty (4 dead/4 tested or 0 dead/4 tested).

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